# Examining Emotion Regulation in Binge Eating Disorder

by © Therese E. Kenny

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Abstract

**Background:** Emotion regulation (ER) theory posits that individuals with binge eating disorder (BED) experience difficulties in ER and consequently engage in binge eating to modulate intense emotions. **Method:** Individual with BED (N = 62) and individuals with no history of an eating disorder (NED; N = 80) were recruited from the community and completed measures of eating disorder psychopathology, difficulties in ER, depression, insomnia, and impulsivity. **Results:** Individuals with BED reported increased difficulties in ER and in emotion-related facets of impulsivity compared to individuals with NED. Increased difficulties in ER were associated with eating disorder psychopathology and depression modulated the relationship between ER and binge frequency, such that this association was significant in individuals who reported higher depression. Insomnia symptom severity did not moderate the relationship between ER and eating disorder pathology. **Conclusions:** The findings support the ER model of BED. Clinical implications and future directions for research are discussed.

*Key words:* binge eating disorder, emotion regulation, impulsivity

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# List of Abbreviations

| | |
|---|---|
| Binge eating disorder | BED |
| American Psychological Association | APA |
| Bulimia nervosa | BN |
| Anorexia nervosa | AN |
| Newfoundland and Labrador | NL |
| Borderline Personality Disorder | BPD |
| Body Mass Index | BMI |
| Cold Pressor Test | CPT |
| Ecological Momentary Assessment | EMA |
| Dialectical Behaviour Therapy | DBT |
| Waitlist Control | WLC |
| Active Control Group Therapy | ACGT |
| Difficulties in emotion regulation scale | DERS |
| Five Facet Model of personality | FFM |
| Prefrontal cortex | PFC |
| Newfoundland Center for Applied Health Research | NLCAHR |
| Memorial University of Newfoundland | MUN |
| Guided self-help | GSH |
| Unguided self-help | USH |
| Self esteem | SE |
| Normal weight control group | NW |
| Overweight control group | OW |
| No history of an eating disorder | NED |
| Health Research Ethics Board | HREB |
| Eating Disorder Foundation of Newfoundland and Labrador | EDFNL |
| Diagnostics and Statistical Manual of Mental Disorders – Fifth Edition | DSM-5 |
| Eating Disorder Examination | EDE |
| Beck Depression Inventory | BDI-II |
| Eating Disorder Examination - Questionnaire | EDE-Q |
| Drug Abuse Screening Test - 10 | DAST-10 |
| Alcohol Use Disorders Identification Test | AUDIT |
| World Health Organization | WHO |
| Insomnia Severity Index | ISI |
| Brief Symptom Inventory | BSI |
| Analysis of Variance | ANOVA |
| Analysis of Covariance | ANCOVA |
| Eating Disorder Not Otherwise Specified | EDNOS |
| Objective binge episode | OBE |
| Subjective binge episode | SBE |
| Standard deviation | SD |

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# Examining Emotion Regulation in Binge Eating Disorder

Binge eating disorder (BED) is characterized by the presence of binge eating episodes at least once per week over the past three months in the absence of regular extreme compensatory behaviours (American Psychological Association [APA], 2013). Binge eating refers to the consumption of an abnormally large amount of food (i.e., more than would be consumed by most people under the same circumstances) in a discrete period of time (e.g., two hours), accompanied by a sense of loss of control (i.e., feeling that one cannot stop eating or control what or how much one is eating). Contrary to bulimia nervosa (BN) however, in BED episodes of binge eating are not followed by extreme compensatory behaviours such as purging or laxative misuse. In addition, individuals with BED experience most, if not all, of the following during episodes of binge eating: (1) eating much more rapidly than normal, (2) eating until feeling uncomfortably full, (3) eating large amounts of food when not physically hungry, (4) eating alone because of feeling embarrassed about how much one is eating, and (5) feeling disgusted with oneself, depressed, or very guilty (APA, 2013). Importantly, binge eating causes significant impairment and distress (APA, 2013), interfering with the individual's ability to function.

The prevalence of BED is 2-3 times greater than the prevalence of the more commonly acknowledged eating disorders anorexia nervosa (AN) and BN (Hudson, Hiripi, Pope & Kessler, 2007). Furthermore, BED has strong associations with obesity, with the prevalence of BED between 4-8% in obese community samples (Hudson et al., 2007) compared to 1-3% in the general population (Dingemans, Bruna & van Furth, 2002; Hudson et al., 2007; Kessler et al., 2013). Importantly though, individuals with

BED experience greater physical and psychological impairments compared to their obese non-BED counterparts (Rieger, Wilfley, Stein, Marino & Crow, 2005), suggesting that BED represents a distinct phenotype of obese individuals. For example, obese adolescents who binge eat report greater deficits on the health and self-esteem subscales of the Impact of Weight on Quality of Life scale compared to their obese non-binge eating counterparts (Ranzenhofer et al., 2012). Currently, Newfoundland and Labrador (NL) has the highest rate of obesity in Canada at 34% (Shields & Tjepkema, 2006) and – although this suggests a high prevalence of BED[1] – no publically funded services are currently available for BED. Thus, there is an urgent need for research examining factors that are associated with the development and maintenance of BED (especially in this province), to increase our understanding of the disorder thereby allowing for the development and dissemination of more effective treatments. The current project aims to provide a comprehensive examination of difficulties in emotion regulation in BED, informing not only the current conceptualization of BED, but also providing alternate therapeutic targets that could be advantageous in the treatment of BED. Before describing emotion regulation and its relation to binge eating in detail, I will start by giving an overview of current theories of BED which have contributed to our understanding of the disorder.

### 1.1 Current Theories of Binge Eating Disorder

A number of theories have been proposed to explain the development and maintenance of BED (for overview see Polivy & Herman, 1993), the majority of which

[1] Given the prevalence of BED in obese community samples (Hudson et al., 2007) and obesity rates in NL (Shields & Tjepkema, 2006), our lab estimates that between 7,000 and 14,000 individuals in NL suffer from BED.

were derived from models of BN, which is also characterized by the presence of binge eating episodes. Of these, two predominant models have emerged (Whiteside et al., 2007): dietary restraint (e.g., restraint theory; Herman & Polivy, 1984) and negative affect models (e.g., masking theory; Herman & Polivy, 1988, escape theory; Heatherton & Beaumeister, 1991), though the cognitive-behavioural theory (Fairburn, 2002; Fairburn, Cooper, & Shafran, 2003; Fairburn, Marcus, & Wilson, 1993) is typically considered to be the most influential theory of BED. This theory proposes that four core psychopathologies (low self esteem, clinical perfectionism, mood intolerance, and interpersonal difficulties) lead to behaviours such as extreme restriction, binge eating, and compensatory behaviours in individuals with eating disorders (Fairburn, Cooper, & Shafran, 2003). It is considered to be a transdiagnostic model of eating disorders (i.e., applies to AN, BN, OSFED, and BED) however it was initially developed to describe BN (Fairburn et al., 2003). Cognitive behavioural theory has been well described elsewhere (e.g., Fairburn et al., 2003) and as such will not be considered in great detail here except to note that the majority of support for this model in BED has come from treatment studies with variable results (for overview see Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Ricca, Mannucci, Zucchi, Rotella, & Faravelli, 2000). Such variability calls for the examination of other models to determine whether the nature of BED can be better characterized in order to develop more effective treatment options.

### 1.1.1 Dietary Restraint.

***1.1.1.i Restraint theory.*** There is significant evidence to suggest that binge eating is associated with dieting (Marcus, 1993), alternatively referred to as dietary restraint (Howard & Krug Porzelius, 1999). Restraint is defined as “the process of self-imposed

food deprivation used to achieve a weight below one's physiological set point" (Nisbitt, 1972 as cited in Howard & Krug Porzelius, 1999, pp. 29). That is, individuals who score high on measures of dietary restraint actively try to restrict their food intake in order to achieve weight loss or, at minimum, to avoid weight gain. Herman and Polivy (1984), in their counter-regulation theory of binge eating, proposed that high levels of dietary restraint lead to a "diet boundary", whereby once a food rule has been broken (e.g., by having a "forbidden" food) there is a loss of inhibition and the individual, rather than stopping when full, eats past the point of satiety. This ultimately leads to overeating or, in extreme cases, a binge. This model has been used to explain both BN and BED, arguing that individuals who suffer from these disorders are high in dietary restraint increasing their susceptibility to binge eating. There is some empirical evidence to support this model in BED. For instance, dietary restraint has been associated with binge eating tendencies (Leon, Fulkerson, Perry, & Cudeck, 1993; Ruderman, 1985) and, in one study, the severity of binge eating correlated with the severity of restraint (Hawkins & Clement, 1980). Another study by Elran-Barak and colleagues (2015) however, found that while dietary restraint was correlated with binge frequency in BN and AN binge-purge subtype, there was no association between restraint and binge frequency in BED. Ultimately, a review by Howard & Krug Porzelius (1999) concluded that in some, but not all, cases dietary restraint may increase susceptibility to binge eating. That the dietary restraint hypothesis can explain binge eating in some cases but not others suggests that dietary restraint cannot be the sole factor driving binge eating.

**1.1.2 Negative affect.** In addition to dietary restraint theory, researchers have suggested that negative affect may contribute to the development and maintenance of

BED. Negative affect theories posit that individuals with BED experience increased negative affect, which in turn leads to binge eating (Polivy & Herman, 1993). Two of the most prominent negative affect theories of BED are masking theory (Herman & Polivy, 1988) and escape theory (Heatherton & Beaumeister, 1991) which argue that binge eating emerges as a way to displace intense negative affect or as a consequence of cognitive narrowing, respectively.

***1.1.2.i Masking theory.*** Herman and Polivy (1988) subsequently proposed a second theory to explain why dieters are more susceptible to binge eating. Masking theory argues that dieters use overeating to mask distress experienced in other areas (Herman & Polivy, 1988). Rather than directly facing the emotional target, the individual is able to displace the distress, attributing it to the binge eating episode (Blackburn, Johnston, Blampied, Popp, & Kallen, 2006). Masking theory was experimentally examined in a sample of self-reported dieters by assigning individuals to a neutral or failure condition (Herman & Polivy, 1999). In the failure condition, university level students ostensibly completed an IQ test (anagrams) on which they scored at a grade six level. In reality, the test was developed so that no participant could complete it. In the neutral condition, individuals were led to believe that the anagrams were not part of the current study and that the majority of people have trouble completing them. After completing the anagrams, participants were asked to complete a taste test and were given either (1) three spoonfuls of ice cream or (2) three bowls of ice cream and told that they could eat as much as they wanted (ad lib). Individuals then attributed their distress levels to eating the ice cream or to completing the anagrams. As expected, individuals who scored high on levels of dietary restraint (i.e., try to abide by strict dietary rules) were

more likely to attribute distress to the ice cream (Polivy & Herman, 1999). Moreover, individuals who were unable to overeat in response to failure (i.e., those who were only given three spoonfuls of ice cream) were less likely to attribute distress to ice cream. While these findings provide partial support for the masking theory of BED, the authors note that displacement was more complicated than simply re-attributing distress to ice cream (Polivy & Herman, 1999) suggesting the need for a more comprehensive theory of BED. Consistent with this assertion, since Herman and Polivy (1999), researchers appear to have abandoned the masking theory of BED, though components of it can still be identified within other, more comprehensive theories.

***1.1.2.ii Escape theory.*** Possibly the most influential theory of BED – aside from the cognitive behavioural theory (Fairburn, 2002; Fairburn, Cooper, & Shafran, 2003; Fairburn, Marcus, & Wilson, 1993) – is escape theory, originally proposed by Heatherton and Beaumeister (1991). This theory proposes that individuals with BED have high standards for themselves (often related to shape and weight), a desire to be perceived favourably by others, and high levels of self-awareness. High self-awareness can be uncomfortable, especially when individuals have perfectionistic tendencies and become aware of their (perceived) failures (i.e., negative self-evaluation; Duval & Wicklund, 1972), ultimately leading to negative affect. In response individuals are motivated to reduce negative affect, thereby avoiding emotional discomfort. Heatherton and Beaumeister (1991) suggest that it is possible to alleviate negative affect through cognitive narrowing. Cognitive narrowing focuses attention on concrete aspects of the immediate environment. Unlike mindfulness however, cognitive narrowing allows the individual to avoid acknowledging and accepting feelings, thoughts, and body sensations,

by solely focusing on the task at hand. In the context of BED, directing energy towards cognitive narrowing releases inhibition of self-destructive behaviours such as overeating. Binge eating in turn facilitates cognitive narrowing through attention to the sensations of food and eating (Heatherton & Beaumeister, 1991). Thus, there is a bidirectional relationship between cognitive narrowing and binge eating; however, it is important to note that in this model binge eating is a consequence – and not a cause – of cognitive narrowing (Blackburn et al., 2006).

Studies of the escape model of binge eating have yielded inconsistent results with different statistical techniques suggesting different conclusions. Structural equation modeling demonstrated that escape theory is a good fit for binge eating (Blackburn et al., 2006) whereas hierarchical regression analysis did not provide support for this model (Paxton & Diggens, 1997). This discrepancy could be attributed to different variables of interest. Paxton and Diggens (1997) focused on avoidance coping which, although a hypothesized consequence of the escape model, is not an aspect of the model itself. Alternatively, Blackburn and colleagues (2006) focused exclusively on measures of the key aspects of escape theory (see Figure 1), demonstrating adequate support for the escape theory of BED. Evidence from the ice cream study (Polivy & Herman, 1999) however suggests that escape theory may be moderated by other variables, such as dietary restraint. In the ad lib condition, individuals who scored high on levels of dietary restraint and experienced the failure condition consumed the most ice cream, compared to individuals with low levels of dietary restraint in the failure condition and individuals with high levels of restraint in the neutral condition (Polivy & Herman, 1999). Therefore,

escape theory may describe individuals with high – but not low – levels of dietary restraint.

Although there was much interest in these models in the 1980's and 90's, very little attention has been paid to them in the past twenty years. Rather, it has been argued that there is a need for the amalgamation of negative affect and dietary restraint theories of binge eating (McManus & Waller, 1995; Meyer, Waller, & Waters, 1998). According to Whiteside and colleagues (2007) subsequent models of binge eating (e.g., Stice, 2001; Stice & Agras, 1999; Stice et al., 2001; Waters, Hill, & Waller, 2001) including cognitive behavioural theory (e.g., Fairburn, 2002) "have been integrated to include both dietary restraint and affect or emotion regulation-based models" (pp. 163), though it should be noted that these models were developed to explain binge eating in BN and not BED. As will be discussed later, research has demonstrated that binge eating serves a different function in BN and BED (for review see Haedt-Matt & Keel, 2011) suggesting the need for different conceptualizations. Moreover, Whiteside et al. (2007), as have a large number of researchers, refer to negative affect and emotion regulation models synonymously. While the two are related, negative affect and emotion regulation represent distinct constructs. Gross (1998) defines emotion regulation as "how individuals influence which emotions they have, when they have them, and how they experience and express them" (pp. 271), while negative affect refers to the experience of an emotion.

Typically Heatherton and Beaumeister (1991) are credited with the emotion regulation theory of BED (e.g., Telch, Agras, & Linehan, 2001) however, as alluded to earlier, there are significant differences between the escape and emotion regulation theories of BED. The emotion regulation theory proposes that binge eating emerges in

response to poor emotion regulation skills as a maladaptive coping mechanism. Thus, the key aspect in the emotion regulation theory is not that individuals experience more negative emotions (as in negative affect models) or greater self-awareness (as in escape theory), but rather that individuals do not have the skills to cope with these experiences. Consequently, they engage in harmful behaviours such as binge eating to relieve the emotional discomfort. According to escape theory, binge eating emerges as a consequence of cognitive narrowing (Blackburn et al., 2006; Heatherton & Beaumeister, 1991) while emotion regulation theory suggests that binge eating itself serves to decrease intense emotions such as negative moods and self-awareness. Thus, the emotion regulation theory of binge eating warrants a discussion beyond negative affect models, such as escape and masking theory.

## 1.2 Emotion Regulation Theory of BED

**1.2.1 Defining emotion regulation.** Before describing emotion dysregulation as a theory of BED, it is important to clearly define the construct of emotion regulation. Emotion regulation has its roots in the study of psychological defense mechanisms (Freud, 1926; 1959), stress and coping (Lazarus, 1966), attachment theory (Bowlby, 1969), and emotion theory (Frijda, 1986), but is conceptualized as a distinct construct[2] (Gross and Thompson, 2007). Gross and Thompson (2007) define emotion regulation as the "heterogenous set of processes by which emotions are themselves regulated" (pp. 7) differentiating emotion regulation from the broader construct of affect regulation. Affect regulation encompasses coping, emotion regulation, mood regulation, and psychological

[2] The authors note that while there is significant overlap between different aspects of affect and affect regulation, including the distinction between emotion and emotion regulation, for research purposes it is necessary to distinguish between these concepts (Gross & Thompson, 2007).

defenses which, although related, each represent a distinct construct. Coping is distinguished from emotion regulation in its focus on decreasing negative affect over longer periods of time, whereas psychological defenses focus on regulating emotions associated with aggressive and/or sexual impulses (Gross and Thompson, 2007). In contrast, mood regulation (or repair) emphasizes altering emotional experience rather than emotional behaviour. Thus, although many researchers use the terms affect regulation, mood regulation, and emotion regulation interchangeably (Gross and Thompson, 2007), these constructs are not synonymous and as such the remainder of this discussion will focus exclusively on emotion regulation.

Emotion regulation involves changes in "emotion dynamics" (Thompson, 1990) including changes in latency, rise time, magnitude, duration, and offset of responses in behaviour, experiential, and physiological domains (Gross and Thompson, 2007). This can result in decreasing, increasing, or maintaining the intensity of an emotion. Emotion regulation can be automatic or controlled, conscious or unconscious, and may have effects at more than one point during the emotion generation process (to be discussed more fully later in this section). Gross and Thompson (2007) have noted three core features of emotion regulation: (1) people can regulate negative or positive emotions by either increasing or decreasing the emotional experience – though individuals are most likely to down-regulate negative emotions (e.g., Gross, Richards, & John, 2006); (2) emotion regulation is initially deliberate but later occurs without conscious awareness; (3) emotion regulation processes are not inherently good or bad (Thompson & Calkins, 1996) – how adaptive an emotion regulation strategy is depends on the context in which it is used.

To understand emotion regulation, emotion generation has been conceptualized using the "modal model" of emotion. This model proposes that a situation (internal or external) directs an individual's attention leading to cognitive appraisal. It is this appraisal that generates the emotional response (see figure 2a). Emotion regulation strategies can thus be antecedent-focused (i.e., occurring before the emotion has been generated) or response focused (i.e., in response to the emotion). Gross and Thompson (2007) describe four basic antecedent focused emotion regulation strategies: (1) situation selection (placing one's self in situations that are less likely to produce an emotional response); (2) situation modification (directly modifying the situation so that it is less emotionally salient); (3) attentional deployment (directing attention within a situation in order to influence emotions); (4) cognitive change (reappraising the situation to alter the emotional impact – recall that emotions are generated from cognitive appraisals). Response modulation on the other hand is a response-focused strategy, referring to the influence of physiological, experiential, or behavioural correlates of emotion. This includes the ability to label an emotion and identify appropriate emotion regulation strategies. To see how these strategies map onto the modal model of emotions see figure 2b.

Having described different emotion regulation strategies it is important to note that these strategies do not occur in isolation. That is, an individual may use many different emotion regulation strategies at once to minimize the impact of the emotion. For instance, in preparing for a stressful exam you might arrive right before the exam to avoid hearing others worry about the exam (situation selection), bring your favourite writing utensils and sit in your usual seat (situation modification), distract yourself before the

exam by listening to music (attentional deployment), run to class so that you can tell yourself that your racing heart is due to running and not nerves (cognitive change), and make plans to meet a friend after the exam (response modulation). Moreover, each of these actions will influence the situation at hand. In figure 2a, emotion generation has been described as a linear process with each emotional event as distinct. This, however, is not how emotion generation occurs in real life. Instead, every time an aspect of the situation (internal or external) is changed, attention, appraisal, and consequently the emotional response are changed. Changes in the emotional response can in turn change the situation (Gross and Thompson, 2007). Thus, the system is always reciprocally interacting, influencing the situation, attention, appraisals, and emotions.

**1.2.2 Emotion regulation in BED.** Having defined emotion regulation, we turn now to how this construct could contribute to the development and maintenance of binge eating. Emotion regulation theory was initially proposed to describe borderline personality disorder (BPD; Linehan, 1993). To account for the development of emotion dysregulation – and subsequent BPD traits – Linehan (1993) described a biosocial model of BPD. In this model, individuals have an innate (e.g., genetic) disposition to experience emotions more intensely (biological component) and are raised in what is known as an "invalidating environment" (social component). That is, the individual's emotional needs were not met as a child (e.g., the child did not receive empathy or validation of their emotions). Consequently these individuals fail to develop effective emotion regulation skills, ultimately resulting in borderline personality traits. Currently, the gold standard for BPD treatment is dialectical behavioural therapy (DBT), which teaches emotion regulation skills, as well as related concepts of distress tolerance and mindfulness.

DBT has recently been adapted for BED and BN (Masson, von Ranson, Wallace, & Safer, 2013; Palmer et al., 2003; Safer, Robinson, & Jo, 2010; Safer, Telch, & Agras, 2001a; Safer, Telch, & Agras, 2001b; Salbach, Klinkowski, Pfeiffer, Lehmkuhl, & Korte, 2007; Telch, 1997; Telch, Agras, & Linehan, 2000; Telch et al., 2001) with relatively successful outcomes, suggesting that emotion dysregulation may also contribute to the development and maintenance of binge eating, a model that was initially proposed by Wiser and Telch (1999). As in BPD, individuals with BED are hypothesized to have an innate vulnerability to more intense emotions including sensitivity, intensity, and duration (Safer, Adler, and Masson, in press), as well as more intense neurobiological responses to the rewarding properties of food (Schienle, Schafer, Hermann, & Vaitl, 2009). These individuals are also more likely to experience invalidating environments as children and subsequently fail to develop effective emotion regulation skills, turning to food as a mechanism to regulate their emotions. While Gross and Thompson (2007) note that food can be an effective emotion regulation strategy, among individuals with BED food becomes the dominant form of emotion regulation, leaving the individual unable to effectively engage other (more adaptive) strategies. This leads to significant impairment and distress as noted in the diagnostic criteria for BED (APA, 2013).

**1.2.3 Empirical evidence for the emotion regulation model of BED.** Few empirical studies have tested different aspects of and predictions made by the emotion regulation model to date. Only a handful of studies have directly measured difficulties in emotion regulation in individuals with BED (Danner, Sternheim, & Evans, 2014; Harrison, Mitchison, Rieger, Rodgers, & Mond, 2016; Robinson, Safer, Austin, & Etkin, 2015; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012; Whiteside et al., 2007).

Rather support for the emotion regulation hypothesis has emerged indirectly from studies of emotional eating and stress/coping, ecological momentary assessments of mood before and after binge eating, and mood induction studies.

***1.2.3.i Emotional eating.*** As the emotion regulation model of BED proposes that individuals with BED overeat in response to intense (and seemingly unmanageable) emotions, emotional eating has been (incorrectly) considered a proxy for emotion regulation (e.g., Elderedge & Agras, 1996; Masheb & Grilo, 2006; Ricca et al., 2009; Vanderlinden, Dalle Grave, Vandereycken, & Noorduin, 2001). An exploratory study found that university age females were most likely to endorse emotional and physiological (e.g., hunger) triggers for binge eating (Vanderlinden et al., 2001). Moreover, Stein and colleagues (2007) reported that individuals with BED were more likely to attribute binge eating to mood compared to hunger and abstinence violation. In addition, individuals with BED are more likely to eat in response to negative moods compared to their obese counterparts (Elderidge & Agras, 1996), providing further support for emotional eating in BED that cannot be attributed to increased body mass index[3] (BMI). Similarly, Masheb and Grilo (2006) reported that emotional eating was associated with binge frequency and eating disorder pathology but not BMI, consistent with other research that has demonstrated an association between binge eating and emotional eating (e.g., Ricca et al., 2009). Despite the association between binge eating and emotional eating however, these findings do not provide sufficient evidence to support an emotion regulation model of BED. Namely, these studies did not actually measure emotion regulation. Additional research is needed to confirm the hypothesis that

[3] Body mass index (BMI) is a measure of body fat based on height and weight ($kg/m^2$).

individuals with BED also have poor emotion regulation skills and that these skills (or lack thereof) mediate the relationship between emotion and eating.

***1.2.3.ii Stress and coping.*** Negative affect and stress are the most commonly reported triggers for binge eating (Gross and Thompson, 2007). For example, Wolff and colleagues (2000) found that negative mood and stress independently predicted binge eating episodes, concluding that stress and negative mood are common antecedents of binge eating. Moreover, irrespective of depressed affect, increased stress was associated with same day binge eating (Yacono Freeman, & Gil, 2004), a finding which has been corroborated by evidence that individuals who binge eat consume more on high stress days (Crowther, Sanftner, Bonifazi, & Shepherd, 2001). Individuals with BED also reported greater hunger and desire to binge after submerging their hand in ice water for two minutes (cold pressor test [CPT]) compared to a weight matched control group (Gluck, Geliebter, Hung, & Yahav, 2004), providing further evidence that stress can precipitate binge eating. In addition, individuals who engage in binge eating rated daily hassles as significantly more stressful, suggesting that these individuals also perceive stress differently from their non-binge eating counterparts (Crowther et al., 2001).

In response to – or rather to deal with – stress, individuals engage in coping, the "process of adaptation to perceived threat" (Roger, Jarvis, & Narjarian, 1993; pp. 619). Roger and colleagues (1993) have empirically demonstrated that there are four primary coping mechanisms: (1) rational coping (e.g., planning to solve a problem); (2) emotion focused coping (e.g., rumination); (3) avoidant coping (e.g., pretending everything is alright) and; (4) detached coping (e.g., viewing a problem as separate from the self). While rational and detached coping are adaptive, emotion focused and avoidant coping

can have detrimental effects (Roger et al., 1993), as evidenced in a non-clinical sample whereby emotion focused and avoidant coping were associated with stress and binge eating (Sulkowski, J. Dempsey, & A. Dempsey, 2011). Notably, emotion focused coping partially mediated the relationship between stress and binge eating strengthening the association between maladaptive coping and dysregulated eating behaviour.

Coping can also be considered at a biological level. For instance, glucocorticoids, such as cortisol (a human stress hormone), act via primary effects, such as by increasing blood glucose (e.g., Lecocq, Mebane, & Madison, 1964) and inhibiting lysozyme activity (e.g., Lappin & Whaley, 1991), as well as via mediators, such as hormones (i.e., insulin; e.g., Kahlan & Adam, 1975), lymphocytes (i.e., interferon; e.g., Guyre, Bodwell, & Munck, 1981), and prostaglandins (i.e., arachadonic acid metabolites; e.g., Araki, Peck, Lefer, & Smith, 1981) to protect the body from over-engaging defense mechanisms in response to stress. It has been noted that individuals with BED have higher baseline cortisol levels compared to non-BED controls, though cortisol did not differ following a dexamethasone suppression test[4] (Gluck et al., 2004). Cortisol was also higher in individuals with BED following the cold pressor test (i.e., immersing the hand in ice water) and this difference approached significance ($p = 0.057$). Gluck and colleagues (2004) therefore suggest that individuals with BED may have altered glucocorticoid

[4] Dexamethasone is an exogenous glucocorticoid that can be administered orally. Like endogenous cortisol release, dexamethasone increases cortisol secretion (i.e., acts as a stressor). Therefore the dexamethasone suppression test can be used to examine neuroendocrine responses to biological stressors. Previously, increased cortisol suppression (i.e., less cortisol is released) has been reported in stress related disorders such as post-traumatic stress disorder (e.g., Grossman et al., 2003; Yehuda et al., 1993; Yehuda, Boiseneau, Lowy, & Giller, 1995; Yehuda, Golier, Halligan, Meaney, & Bierer, 2004; Yehuda, Halligan, Golier, Grossman, & Bierer, 2004; Yehuda, Halligan, Grossman, Golier, & Wong, 2002).

functioning (otherwise known as the stress response) which could contribute to alterations in eating patterns.

As with studies of emotional eating, an assumption has been made that because stress precedes binge eating, this can be considered evidence for an emotion regulation model. Again I counter that these studies have failed to include measures of emotion regulation. While coping and emotion regulation have been referred to interchangeably, recall that there are significant differences between the two (Gross and Thomson, 2007). Namely, coping focuses exclusively on reducing negative affect over the long-term. In contrast, emotion regulation can be applied to negative or positive emotions, and occurs in the immediate moment. Thus, using coping as evidence for the emotion regulation theory of BED is misdirected.

***1.2.3.iii Ecological momentary assessment.*** Ecological momentary assessment (EMA) involves momentary and repeated ratings over a discrete period of time (e.g., a participant may be randomly paged – via a handheld computer or pager – to complete an assessment x times over the course of a day). EMA is especially effective for assessing antecedents and consequences of binge eating because individuals can be probed about mood and related factors throughout the day (Haedt-Matt & Keel, 2011). It has been proposed that if the emotion regulation model holds for binge eating there should be an increase in negative affect and decrease in positive affect prior to the binge eating episode, and a reversal of these states subsequent to the binge. EMA examination of binge eating in BN demonstrated support for this hypothesis reporting increases in negative emotion prior to both bingeing and purging, and subsequent relief following the binge-purge episode (Smyth et al., 2007). Hilbert and Tuschen-Caffier (2007) reported similar

findings in a sample of women with BN. In BED however, findings from EMA studies have been less conclusive (e.g., Hilbert & Tuschen-Caffier, 2007; Stein et al., 2007). Namely, studies of binge eating in BED suggest that there is an increase in negative affect prior to the binge eating episode followed by an even greater increase in negative affect after the binge (Hilbert & Tuschen-Caffier, 2007; Stein et al., 2007; Wegner et al., 2002). A meta-analysis of 36 EMA studies of binge eating concluded that there is in fact an increase in negative affect following binge eating, and that this increase is later relieved by purging behaviour in BN (Haedt-Matt & Keel, 2011). While these results seem to contradict the emotion regulation theory of BED, they do not preclude the possibility that binge eating serves a regulatory (or numbing) function *during* the binge eating episode which is immediately followed by intense feelings of guilt or shame (Safer et al., in press; see Figure 3). Given the secretive nature of binge eating, even EMA is unlikely to obtain reports of emotions and feelings during the binge eating episode. Thus, while evidence from EMA studies may not fully support the emotion regulation model of BED, it does not discount it either.

***1.2.3.iv Mood induction studies.*** Researchers have argued that some of the most compelling evidence for the emotion regulation model of binge eating comes from mood induction studies. During mood induction, participants are made to experience a particular emotion, usually by asking them to recall a personal event or watching a sad film. In studies of binge eating, participants are typically randomized into neutral and negative affect conditions before being exposed to a sham taste test. During the taste test participants are given access to a buffet and told that they can eat as much as they want to be able to rate the tastiness of the food available. The amount of food consumed is used as

an indication of eating in response to emotions, and thus considered as evidence for the emotion regulation model of binge eating.

The findings from mood induction studies in BED have been inconsistent and difficult to interpret. Telch and Agras (1996) found that mood condition did not influence the amount of food consumed in obese individuals with BED and weight matched controls, though in *both* the neutral and the negative mood induction conditions individuals with BED ate nearly twice as much as control participants. Interestingly, individuals who labeled the eating episode as a "binge" had higher depression scores after the mood induction procedure. The authors also noted an association between negative mood and loss of control (Telch & Agras, 1996), a finding which was replicated by Agras and Telch (1998). While negative mood did not affect caloric consumption, it did appear to affect the perception of binge eating. Similarly, Munsch and colleagues (2008) reported that there was no main effect of mood on food intake following negative mood induction. Alternatively, within a sample of individuals with BED, Chua, Touyz, and Hill (2004) found that individuals in the negative mood condition ate significantly more food. The authors noted that individuals who scored high on measures of restraint ate the most, though the mood by restraint interaction was not significant ($p = 0.06$; Chua, Touyz, & Hill, 2004). Moreover, there is evidence that trait anxiety – but not trait anger – predicts emotional eating after mood induction (Schneider, Appelhans, Whited, Oleski, & Pagoto, 2010). Despite these inconsistencies, a systematic review of 18 experimental studies (Leehr et al., 2015) concluded that there is sufficient evidence to support the emotion regulation model of BED, noting an appreciable increase in negative affect prior to binge eating and short-term improvement of mood through binge eating. While this evidence is

compelling, these studies again lack measures of emotion regulation thereby neglecting a central prediction of the emotion regulation model – that individuals with BED experience emotion regulation deficits.

Taken together, the findings from emotional eating, stress/coping, EMA, and mood induction studies indicate that binge eating is associated with emotion. In particular, studies have concluded that negative moods and stress precede binge eating episodes (e.g., Haedt-Matt & Keel, 2011; Yacono Freeman & Gil, 2004) and that binge eating days are characterized by greater stress and negative mood (Crowther et al., 2001). Furthermore, following negative mood induction individuals with BED consume more than their non-eating disorder counterparts (Chua et al., 2004), providing compelling evidence for negative affect and stress as antecedents to binge eating. EMA studies have also examined mood after binge eating noting that in BED binge eating is followed by an even greater increase in negative affect (for review see Haedt-Matt & Keel, 2011). Some authors have argued that this is evidence against the emotion regulation model of binge eating; however, no studies have examined the possibility that negative affect is temporarily relieved *during* the binge eating episode but increases afterwards[5]. Furthermore, none of the above studies included a measure of emotion regulation, a central component of emotion regulation theory. While some studies have examined coping as a form of emotion regulation (e.g., Sulkowski et al., 2011), Gross and Thompson (2007) argue that these two terms are not synonymous. Thus the aforementioned studies of coping are insufficient to provide evidence for the emotion

[5] This proposition is consistent with the Dialectical Behaviour Theory of binge eating proposed by Safer and colleagues (in press; see Figure 3).

regulation model of BED. In the following section, evidence for the emotion regulation model from studies that have measured emotion regulation or examined dialectical behaviour therapy will be reviewed.

***1.2.3.v Emotion regulation studies.*** To date a handful of studies have examined measures of difficulties in emotion regulation in individuals with BED (e.g., Danner et al., 2014; Harrison et al., 2016; Robinson et al., , 2015; Svaldi et al., 2012; Whiteside et al., 2007). These studies suggest that individuals with BED demonstrate greater difficulties with emotion regulation compared to normal weight controls (Danner et al., 2014; Harrison et al., 2016; Robinson et al., 2015; Svaldi et al., 2012). Moreover, Whiteside and colleagues (2007) reported that emotion regulation difficulties – in particular limited access to emotion regulation strategies and lack of emotional clarity – uniquely accounted for an additional 6.2% of variance in binge eating episodes above and beyond the contribution of sex, restraint, and overvaluation of weight and shape (for discussion of overvaluation see section "Associated Factors"). Thus, there is preliminary evidence suggesting that individuals with BED, relative to non-BED controls, do in fact experience greater difficulties in emotion regulation. These studies are limited however in that they were either conducted in a non-clinical sample (Whiteside et al., 2007), did not include a control group (Harrison et al., 2016), or if a control group was included, they were not matched on BMI (Danner et al., 2014; Robinson et al., 2015; Svaldi et al., 2012). This begs the question as to whether differences in emotion regulation were associated with binge eating or were related to differences in BMI.

***1.2.3.vi Dialectical behaviour therapy for BED.*** Some of the most compelling evidence for the emotion regulation model of BED has emerged from trials of dialectical

behaviour therapy (DBT) for BED. As mentioned earlier, DBT is considered to be the gold standard for BPD treatment (Linehan, 1998). DBT has four overarching treatment goals (Wiser & Telch, 1999): (1) mindfulness skills (increasing non-judgment of the self and others, as well as awareness and acceptance of emotional states); (2) distress tolerance (increasing the individual's ability to sit with uncomfortable emotions); (3) emotion regulation (reducing vulnerability to intense emotions and increasing the ability to alter emotional states in an adaptive way) and; (4) interpersonal effectiveness (being able to identify what one wants/needs from a relationship and increasing assertiveness). Wiser and Telch (1999) argue that DBT provides the individual with a "toolbox" of emotion regulation skills, that could be effective in reducing binge eating and eating disorder pathology in BED.

A case study of DBT in a 36-year-old woman with severe BED (Telch, 1997) provided the first evidence for DBT treatment of BED. After 23 individual 50-minute sessions over ten months, the individual no longer met the diagnostic criteria for BED (Telch, 1997). Subsequently, an uncontrolled trial of group DBT in 11 women with BED reported that by the end of treatment 82% were abstinent (i.e., no longer binge eating; Telch et al., 2000). Moreover, abstinence rates were maintained at 80% and 70% at 3- and 6-month follow-up respectively (Telch et al., 2000), suggesting that the positive benefits of DBT persist beyond the end of treatment. Two randomized controlled studies of group DBT for BED have been conducted comparing DBT to a waitlist control (Telch et al., 2001) and active control group therapy (ACGT; Safer et al., 2010). Compared to the waitlist control (WLC), abstinence rates were significantly higher following DBT (DBT: 89% vs. WLC: 12.5%), though these dropped to 56% at 6-month follow-up (Telch et al.,

2001). Similarly, abstinence rates were higher following group DBT compared to ACGT (DBT: 64% vs. ACGT: 34%), although by 6-month follow-up rates in the ACGT group were no longer significantly lower (DBT: 64% vs ACGT: 56%; Safer et al., 2010). It is worth noting however that abstinence rates were maintained at follow-up in the DBT group and significantly fewer participants dropped out of the DBT condition. Thus, DBT may still be more effective than ACGT given that abstinence is achieved earlier and maintained for at least six months, and treatment adherence is significantly better. A third, randomized controlled trial was conducted examining guided self-help DBT treatment for BED compared to a waitlist control (Masson et al., 2013). Unlike group DBT, self-help DBT is administered individually via a self-help manual[6]. In guided self-help, participants in this study were invited to participate in six sessions over the telephone with a guided self-help therapist whose role was to support the participant in the completion of the program. At the end of treatment, participants in the DBT guided self-help condition reported fewer past month binge eating episodes compared to the control condition (DBT: 6.0 vs WLC: 14.4; Masson et al., 2013), although it is important to note according to the DSM-V these individuals would still meet the frequency criteria for BED. Individuals who received guided self-help DBT also reported greater rates of abstinence at the end of treatment (DBT: 40%[7] vs. WLC: 3.3%). At 6-month follow-up, DBT participants reported improved quality of life and decreased eating disorder psychopathology (e.g., overconcern about weight), and the majority of binge eating improvements were

[6] An advantage of self-help DBT is that it can be more easily disseminated.
[7] It is noted that abstinence rates in this study were lower than in previous randomized controlled trials of DBT for BED. This study however, examined self-help which requires significantly more motivation and self-direction by participants. Thus, it makes sense that abstinence rates would be lower.

maintained (DBT abstinence: 30%; Masson et al., 2013), suggesting that a significant subgroup of individuals with BED may benefit from a DBT guided self-help program.

The efficacy of DBT in reducing binge eating episodes indirectly suggests that emotion regulation difficulties contribute to binge eating in BED. Counter-intuitively however changes in binge eating have not always been accompanied by changes in scores on measures of emotion regulation. While Telch, Agras, & Linehan (2000) reported that there were decreases in difficulties with emotion regulation following DBT treatment for BED, subsequent studies have failed to replicate this finding (Masson et al., 2013; Safer et al., 2010; Telch et al., 2001). One study, however, reported that the extent of change in emotion regulation from pre- to post-treatment was associated with the likelihood of binge abstinence (Wallace, Masson, Safer, & von Ranson, 2014) which could account for the discrepancies in previous studies. Thus, there is a need for additional research examining changes in emotion regulation during DBT for BED.

**1.2.4 Components of emotion regulation.** To this point, I have discussed emotion regulation as it is defined by Gross and Thompson (2007) in *The Handbook of Emotion Regulation*. Other researchers however, have provided complimentary definitions that can enhance the understanding of emotion regulation in BED. Namely, Gratz and Roemer (2004) conceptualized emotion regulation as including four aspects: (1) awareness and understanding of emotions; (2) acceptance of emotions; (3) the ability to inhibit inappropriate and/or impulsive behaviours and behave in a goal directed manner in the face of intense emotions, and (4) the ability to use situationally appropriate emotion

regulation strategies to *modulate*[8] emotional responses. It is argued that deficits in any or all of these areas indicate difficulties with emotion regulation (Gratz & Roemer, 2004). A self-report measure – the difficulties in emotion regulation scale (DERS; Gratz & Roemer, 2004) – was developed to measure the four components of emotion regulation. Factor analysis revealed that these components are actually captured by six subscales. The first two subscales (AWARE and CLARITY) measure awareness and understanding of emotions, while the third (NON-ACCEPT) assesses acceptance of emotions. IMPULSE and GOALS address the ability to inhibit impulsive responding and remain on task during intense emotions and finally, the STRATEGIES subscale is a measure of access to situationally appropriate emotion regulation strategies (Gratz & Roemer, 2004). Thus, emotion regulation can be conceptualized as an overarching construct comprising six facets (awareness, clarity, non-acceptance, impulsivity, goal directed behaviour, and access to strategies).

From this, it follows that different aspects of emotion regulation may be differentially related to the development and maintenance of BED. Harrison and colleagues (2016) reported that the Goals, Impulse, Aware, and Clarity subscales differentiated between healthy controls and individuals with probable BED. In contrast, the Non-accept and Strategies subscales did not reliably differentiate between individuals with probable BED and healthy controls. Rather these subscales appeared to be related to a subpopulation of individuals with BED who reported clinically significant overvaluation of weight and shape (Harrison et al., 2016). In another study, the Goals and

[8] In contrast to theories which suggest that emotion regulation strategies act to "eliminate" emotions, Gratz and Roemer (2004) – like Gross and Thompson (2007) – argue that the function of emotion regulation strategies is to modulate emotions by decreasing their duration or intensity.

Strategies subscales explained unique variance in emotional eating whereas the Non-accept and Goals subscales predicted eating disorder psychopathology (Gianini, White, & Masheb, 2013), while Whiteside and colleagues (2007) found the Strategies and Clarity subscales to explain unique variance in binge frequency.

***1.2.4.i Impulsivity.*** Though markedly absent from the above findings (Gianini et al., 2013; Whiteside et al., 2007), impulsivity has also received considerable attention in BED using measures other than the DERS. Impulsivity is typically defined as a personality trait characterized by enhanced reward sensitivity and rash spontaneous behaviour (Dawe, Gullo, & Loxton, 2004). Other researchers (i.e., Cyders et al., 2007; Whiteside & Lynam, 2001) however argue that impulsivity is multi-faceted and can be better understood using the five-facet model (FFM) of personality (McCrae & Costa, 1990). Using a factor analytic approach Whiteside and Lynam (2001) identified a four-factor model of impulsivity derived from the FFM. According to this model impulsivity includes Urgency (the tendency to act impulsively when experiencing negative emotions), lack of Premeditation (not thinking ahead), lack of Perseverance (not carrying through), and Sensation Seeking (Whiteside & Lynam, 2001). Subsequently, Cyders and colleagues (2007) provided evidence for a fifth facet of impulsivity, Positive Urgency. Like urgency, positive urgency refers to the tendency to act impulsively when experiencing emotions. In this case, however, the emotions are positive as opposed to negative. From this conceptualization, it is clear that impulsivity in the context of emotion regulation refers to the urgency facets (both positive and negative). Thus, while researchers have suggested that individuals with BED report an overall increase in impulsivity (Annagur, Orhan, Ozer, Yalcin, & Tamam, 2015; Galanti, Gluck, & Geliebter, 2007; Hege et al., 2015;

Manasse et al., 2014; Manwaring, Green, Myerson, Strube, & Wilfley, 2011; Meule & Platte, 2015; Mobbs, Iglesias, Golay, & Van der Linden, 2011; Schag et al., 2013), the following discussion of impulsivity in BED will be limited to negative and positive urgency.

***1.2.4.ii Negative and positive urgency in BED.*** Research examining the role of negative urgency in BED has demonstrated a consistent positive relationship between negative urgency scores and binge eating (e.g., Farstad et al., 2015; Kelly, Cotter, & Mazzeo, 2014). For instance, Manasse and colleagues (2016) found that only the emotion related facet (i.e., negative urgency) of impulsivity was associated with improvement following treatment for BED. Moreover, negative urgency contributes to both aspects of binge eating episodes (i.e., objective overeating and loss of control; Racine et al., 2015). Racine and colleagues (2015) suggest that negative urgency increases sensitivity to the rewarding properties of food when individuals experience intense negative emotions leading to overeating, whereas negative urgency leads to behavioural impairments, which facilitate loss of control. Thus, negative urgency appears to play a role in both the reward sensitivity and rash spontaneous behaviour aspects of impulsivity in BED.

Recently, research has turned to the role of possible disorder specific moderators, noting that negative urgency is a risk factor for many mental disorders aside from eating disorders, including depression and problematic alcohol use (Racine & Martin, 2016). Consistent with the conceptualization of negative urgency as a global risk factor which interacts with disorder specific factors, the interaction between negative urgency and

eating disorder specific factors[9] explained unique variance in binge eating, but not in non-eating related symptoms such as alcohol misuse (Racine & Martin, 2016). Other studies have shown that negative urgency interacts with distress tolerance (Anestis, Selby, Fink, & Joiner, 2007), dietary restraint (Emery, King, & Levine, 2014), positive emotions (Emery et al., 2014), and emotional awareness (i.e., understanding and clarity; Manjrekar, Barenbaum, & Bhayani, 2015) to predict binge eating. Specifically, individuals who report high negative urgency tend to report more binge eating episodes if they have lower distress tolerance, higher dietary restraint, lower positive emotions, or decreased understanding/clarity of emotions, indicating the complex – yet undeniable – role of negative urgency in BED.

In contrast, research examining the role of positive urgency in BED has been sparse. To my knowledge, as of yet no studies have examined positive urgency in individuals with BED. One study by Cyders and colleagues (2007), that examined impulsivity and binge eating behaviour in individuals with eating disorders (but not specifically BED), concluded that negative – but not positive – urgency contributed to binge frequency (Cyders & Smith, 2008). Similarly, in heavy drinkers, positive urgency predicted eating concerns, while negative urgency – but not positive urgency – predicted binge eating (Stojek, Fischer, Murphy, & MacKillop, 2014). In individuals displaying self-harm, positive urgency was a significant predictor of disordered eating, while negative urgency was a global predictor of disordered eating, problematic alcohol use, and self-harm (Dir, Karyadi, & Cyders, 2013). Thus, while the relationship between

[9] The authors selected factors that have previously been shown to influence the development of BN via the dual pathway model.

positive urgency and binge eating remains unclear, positive urgency does appear to play a role in eating behaviour. In a personal communication, Dr. Jacqueline Carter, PhD, R. Psych, revealed that in her clinical experience, individuals with BED often report binge eating episodes following intense positive emotions such as happiness (J. Carter, personal communication, 2016), providing clinical (albeit anecdotal) evidence that positive urgency may in fact contribute to the development and maintenance of binge eating in BED. Discounting the role of positive urgency in BED based on these three studies is premature and additional research is required before we can comment on the role of positive urgency in BED.

### 1.3 Associated Factors

From the evidence provided above it is clear that emotion regulation and urgency play a role in binge eating. As with all mental disorders though, it is unlikely – incomprehensible even – that these factors act in isolation. In considering theoretical models of BED, it is important to note that many different factors interact to affect the individual's experience and behaviour. Any comprehensive model must therefore consider factors that are likely to interact with the factor of interest (in this case emotion regulation). As such, the next section provides a discussion of two factors that could have important implications in the role of emotion regulation in BED: (1) sleep and (2) overvaluation of weight and shape.

**1.3.1 Sleep disturbance and emotion regulation in BED.** Recently it has been proposed that there is an interplay between sleep and emotion regulation (Gruber & Cassoff, 2014). In particular, nighttime sleep patterns affect emotion reactivity and the ability to regulate emotions. In turn, emotional experiences during the day can affect

sleeping patterns at night. Recall that one of the emotion regulation strategies proposed by Gross and Thompson (2007) was cognitive control. Cognitive control includes a number of cognitive abilities that can be succinctly characterized as executive functions (Davidson, Amso, Anderson, & Diamond, 2006). Findings from recent neuroscience studies suggest that the same neural pathways that underlie executive functions, also underlie emotion regulation (Gruber & Cassoff, 2014). Given this finding, it has been hypothesized that sleep deprivation alters emotion regulation similar to the way it alters executive functions, namely by altering prefrontal cortex (PFC) – amygdala[10] connectivity. This hypothesis has been corroborated by subsequent brain-imaging studies (Sotres-Bayon, Bush, & LeDoux, 2004; Yoo, Gujar, Hu, Jolesz, & Walker, 2007), with associations between alterations in connectivity and difficulties regulating strong emotions following sleep deprivation (Yoo et al., 2007).

Given the possible associations between sleep deprivation and emotion dysregulation (for review see Gruber & Cassoff, 2014) and emotion regulation and psychopathology (for review see Sheepes, Suri, & Gross, 2015) researchers have begun to examine the role of sleep and emotion regulation in clinical disorders. Relevant to BED, sleep deprivation has been shown to exacerbate the relationship between emotion dysregulation and BPD symptoms (Selby, 2013). Further, studies have suggested that sleep difficulties may maintain emotional difficulties in bipolar disorder during periods of remission (Boudebesse & Henry, 2012). It is thus possible that sleep deprivation may have similar exacerbating effects on binge eating and eating disorder psychopathology in

[10] The prefrontal cortex is responsible for higher-order cognitive processes and is involved in executive functions. The amygdala is the part of the brain involved in emotion processing.

BED. In line with this assertion, unpublished data from our lab suggests that insomnia symptom severity is associated with binge frequency in individuals with BED (Van Wijk, 2017).

**1.3.2 Overvaluation of shape and weight.** Overvaluation of weight and shape – placing excessive importance on the way one's body looks[11] - is currently a diagnostic criterion for AN and BN, but not BED (APA, 2013) though recently there have been arguments for the inclusion of this criterion in BED as well (Goldschmidt et al., 2010; Hrabosky, Masheb, White, & Grilo, 2008; Masheb & Grilo, 2003). Specifically, Grilo (2013) argues that the overvaluation of weight and shape should be included as a diagnostic specifier of BED as it has been reliably associated with increased severity of the disorder. Previous studies have indeed found that individuals with BED reported higher levels of weight and shape concern compared to non-eating disorder controls (Eldredge & Agras, 1996) and those who reported clinical levels of overvaluation of weight and shape experienced the most extreme eating disorder pathology (e.g., Mond, Hay, Rodgers, & Owen, 2006).

It has been proposed that activation of negative body schema can contribute to the development of negative affect thereby influencing an individual's desire to binge eat (e.g., Williamson, White, York-Crowe, & Stewart, 2004). EMA has demonstrated that women were most likely to attribute negative mood to shape and weight issues prior to an episode of binge eating (Stein et al., 2007). Experimental studies have supported these findings, demonstrating that exposure to a film clip designed to trigger negative body

[11] This is not simply dissatisfaction with how one looks. Individuals who report overvaluation of weight and shape are unable to disentangle their self-worth from their weight and/or shape.

schemas (Svaldi et al., 2009) or mirror-exposure (Naumann, Trentowska, & Svaldi, 2013) increased desire to binge in individuals with BED but not healthy controls. It was further demonstrated that individuals with BED actually showed an increase in saliva following mirror-exposure (Naumann et al., 2013), a response which is not under conscious control. Thus, there is substantial evidence to suggest that body image disturbance does contribute to negative affect thereby triggering binge eating; however Whiteside and colleagues (2007) have demonstrated that difficulties in emotion regulation contribute to binge eating above and beyond overvaluation of weight and shape in a non-clinical sample. Thus, body image disturbance, though it should be considered in studies of BED, cannot fully explain the association between emotion regulation and binge eating.

### 1.4 Current Study

After reviewing the literature, it is clear that there is strong preliminary evidence for an emotion regulation model of BED. Studies used to support this model however have been limited in the lack of (1) emotion regulation measures and (2) weight matched control groups, as well as the use of (3) non-clinical samples. Furthermore, while previous studies have accounted for overvaluation of shape and weight, to my knowledge, no studies have examined the role of sleep in the relationship between emotion regulation and binge eating. The current study will address these limitations by examining difficulties in emotion regulation and impulsivity (i.e., negative and positive urgency) in a community sample of individuals with BED compared to non-BED normal-weight and overweight control groups. Furthermore, the associations between sleep, emotion regulation, and eating disorder pathology will be examined after controlling for BMI, age, and depression. Finally, all analyses will be re-run adding overvaluation of weight and

shape to the model in order to confirm that the findings cannot be attributed to overvaluation of weight and shape, given its significance in BED (Grilo, 2013). It is hypothesized that: (1) individuals with BED will report greater difficulties in emotion regulation and higher urgency scores than individuals in the normal and overweight control groups; (2) after controlling for BMI, age, and depression, greater emotion regulation difficulties will be associated with greater eating disorder psychopathology in BED; (3) level of sleep difficulties will moderate the association between emotion regulation and eating disorder psychopathology in BED such that the relationship will be exacerbated in individuals with greater insomnia symptoms; (4) adding overvaluation of weight and shape to the model will not change the pattern of results.

## 2.0 Method

This study was part of a larger NLCAHR funded randomized controlled trial conducted by Dr. J. Carter at Memorial University of Newfoundland (MUN) examining the effectiveness of dialectical behavioural therapy (DBT) self-help for BED. Individuals who met the inclusion criteria were randomized to one of three conditions: (1) guided self-help (GSH) DBT; (2) unguided self-help (USH) DBT or; (3) USH self-esteem (SE), as an active control condition. A DBT self-help manual (Safer et al., in press) or self-esteem manual (McKay & Fanning, 2015) was mailed to participants in the DBT and SE conditions respectively. Treatment lasted 12 weeks in all conditions. Individuals randomized to the GSH treatment were invited to participate in six 30-minute video sessions with a student therapist, while those in the USH condition were instructed to work through the manual independently. The current project focused on a subset of data from the randomized controlled trial. Namely, a cross sectional analysis of individuals

with BED was performed at a single time point (baseline) and compared to normal weight (NW) and overweight/obese (OW) control groups with no history of an eating disorder (NED).

## 2.1 Ethical Approval

Procedures for this study were in accordance with provincial safety and ethical standards, and were approved by the Health Research Ethics Board (HREB) of Newfoundland and Labrador. Full ethics approval was obtained from the HREB (reference numbers: 2016.202 and 2016.208).

## 2.2 Participants

**2.2.1 BED sample.** Individuals with BED were recruited throughout the province of Newfoundland and Labrador (NL) via the Eating Disorder Foundation of NL (EDFNL), family doctor practices, local radio stations and newspapers, weekly church bulletins, and posters/brochures with information about the study posted in hospitals, colleges, medical clinics, and universities. The HOPE program (day-treatment program for eating disorders in St. John's, NL), which is not equipped to treat BED, was also sent information about the study and asked to refer any clients that they felt may meet the criteria for BED. Interested participants were directed to a website to complete a screening questionnaire (via Qualtrics), and those who met the initial screening criteria were contacted by one of the investigators to complete a telephone interview to confirm eligibility (the full telephone interview is included in Appendix A).

***2.2.1.i Inclusion Criteria.*** Individuals were eligible for the randomized controlled trial if they met the DSM-5 criteria for BED according to the Eating Disorder Examination (EDE) Version-17 presented in Appendix B (Fairburn, Cooper & O'Connor,

2014). Briefly, the EDE specifies that in order to be diagnosed with BED, individuals must report at least one binge episode per week on average over the past three months, endorse 3/5 binge characteristics, and experience distress or impairment related to binge eating. Moreover, binge eating cannot be associated with extreme compensatory behaviours and cannot occur during a period of anorexia nervosa (AN). To ensure that we were not mistakenly including individuals with bulimia nervosa (BN), we developed a more conservative operational definition of compensatory behaviour than what has been proposed by Fairburn and colleagues (2014; see Appendix B). In the current study, compensatory behaviour was defined as purging (self-induced vomiting), laxative misuse (taking at least twice the recommended dosage), diuretic misuse (taking at least twice the recommended dosage), excessive exercise (exercise that is excessive in terms of duration, intensity, and frequency, and interferes with daily functioning), and/or fasting (not eating anything for a period of eight or more waking hours) at least once per month over the past six months. In cases where individuals reported frequent binge eating (e.g., more than three episodes per week over the past three months) and compensatory behaviours between six and twelve times over the past six months[12], the primary investigators who have extensive clinical experience with BED (JC, OH, and DS) were consulted. Eligibility was determined based on whether the individual was judged to be eligible for treatment of BED in a clinical setting. Additional inclusion criteria for the current study included: (1) at least 19 years of age; (2) body mass index (BMI) greater than or equal to

[12] In this case, the ratio of binge eating to compensatory behaviours would be low (maximum 6-12%) suggesting that the dominant tendency is binge eating rather than the binge-purge cycle.

18.5; (3) ability to read English; (4) high school graduate or equivalent; (5) access to a computer, tablet or cell phone (with microphone and camera) and high speed internet[13].

***2.2.1.ii Exclusion Criteria.*** Individuals were deemed ineligible if they: (1) were receiving specialized psychological treatment with a registered psychologist for BED at the time of screening; (2) met the criteria for AN or BN; (3) reported a major medical illness that could interfere with treatment (e.g. Type II diabetes, hypothyroidism); (4) reported current acute suicidal risk as determined by the BDI-II (Beck, Ward, Medelson, Mock, & Erbaugh, 1961) or; (5) reported a possible substance-related and addictive disorder determined by the DAST-10 and AUDIT. Individuals who had been on a stable dose of an antidepressant for at least three months were eligible to participate in the study.

**2.2.2 Non-BED control group.** Individuals with NED were recruited via social media and posters/brochures in doctor's offices, other medical practices, colleges, universities, and local cafes/restaurants. Individuals were instructed to visit a screening website and asked to complete a short questionnaire (administered by Qualtrics). The screening questionnaire asked participants to provide a brief medical history and to complete the SCOFF screening questionnaire for eating disorders (Morgan, Reid, & Lacey, 1999). Participants in the NED control group were excluded if they responded "yes" to three or more questions on the SCOFF questionnaire indicating suspicion of an eating disorder, or if they reported previously/currently being diagnosed with an eating disorder. All other inclusion and exclusion criteria were the same as for the BED group to ensure that the samples were matched on as many variables as possible. Individuals who

[13] Guided self-help sessions were completed via video conferencing requiring a video camera and microphone, as well as access to high speed internet in a private location.

reported a current diagnosis of BED[14] were provided with information about the self-help study and given the opportunity to participate in a telephone interview if they wished. Alternatively, if an individual in the self-help study did not meet the diagnostic criteria for BED they were provided with information about the current study and asked if they would be interested in participating. After data was collected for the non-BED control group, individuals were categorized based on their BMI as either normal (BMI between 18.5-25) or overweight (BMI >25). This formed the basis for the normal (NW) and overweight (OW) control groups.

**2.3 Procedure**

**2.3.1 BED Group.** Except for the initial EDE interview to confirm BED diagnosis, which occurred over telephone, all data were collected on-line through Qualtrics. Once eligibility was confirmed during the telephone interview, participants were then sent a web-link to access the baseline measures online. Following completion of the baseline measures, participants were randomized to either the GSH, USH, or SE condition as part of the randomized controlled trial. The larger study administered measures at baseline, mid-treatment (6 weeks), and post-treatment (12 weeks), but only the baseline measures were used for the current analysis and will be reported here.

**2.3.2 Non-BED control group.** After completing the screening questionnaire, eligible participants were sent a link to the study questionnaires and data was collected via Qualtrics. Participants who completed the study questionnaire were entered into a draw for a chance to win a $100 Visa gift card.

[14] Individuals who were suspected to suffer from an eating disorder other than BED according to the SCOFF were provided with a list of the appropriate services.

## 2.4 Assessment Measures

### 2.4.1 Screening measures.

***2.4.1.i Eating Disorder Examination Interview (Version 17).*** (Fairburn et al., 2014) The Eating Disorder Examination (EDE-17) is an investigator-based interview designed to make eating disorder diagnoses based on DSM-5 criteria. The EDE-17 examines the frequency and severity of symptoms over the past month, though there is an option to extend the time frame for diagnostic purposes (see Appendix B for criteria used in the current study). The validity and reliability of the EDE has been established (Berg, Peterson, Frazier & Crow, 2012). The EDE demonstrates good test-retest (Grilo, Masheb, Lozano-Blanco, & Barry, 2004; Rizvi, Peterson, Crow, & Agras, 2000) and interrater (Cooper & Fiarburn, 1987; Grilo et al., 2004; Rizvi et al., 2000; Rosen, Vara, Wendt, & Lettenberg, 1990) reliability, as well as adequate internal consistency (Beaumont, Kopec-Schrader, Talbot, & Touyz, 1993; Byrne, Allen, Lampard, Dove, & Fursland, 2010; Cooper, Cooper, & Fairburn, 1989; Grilo et al., 2010). Moreover, the criterion (Cooper et al., 1989; Rosen et al., 1990; Wilfley, Schwartz, Spurrell, & Fairburn, 2000; Wilson & Smith, 1989) and convergent (Barnes, Masheb, White, & Grilo, 2011; Farcus Stein & Corte, 2003; Loeb, Pike, Walsh, & Wilson, 1994; Rosen et al., 1990) validity have been well established. For the purpose of this study, a brief version of the EDE-17 was administered including only the key diagnostic items for BED (see Appendix A).

***2.4.1.ii SCOFF questionnaire.*** (Morgan et al., 1999) The SCOFF questionnaire is a five-item questionnaire designed to identify individuals at risk for an eating disorder (see Appendix C for items). All questions require a “yes” or “no” answer and can be administered on paper or verbally (as with other questionnaires in the current study the

SCOFF was administered on-line via Qualtrics). Previous research has demonstrated good convergence between the oral and written versions (Perry et al., 2002). The SCOFF criteria specifies that individuals who respond "yes" to two or more questions are suspected to have an eating disorder (Morgan et al., 1999); however subsequent research has demonstrated that using a cutoff of three "yes" responses or more increases the specificity – ability to correctly classify individuals as disease-free – from 87.5% to 95.8%, with only a 0.9% decrease in sensitivity – ability to correctly identify positive cases of the disease – from 100% to 99.1% (Hill, Reid, Morgan & Hubert Lacey, 2010). As such, we used a cutoff of three "yes" responses, given that it maximizes the specificity-sensitivity trade-off. Any individuals who reported suspicion of an eating disorder were provided with information regarding eating disorder services in NL. The SCOFF has demonstrated good reliability and validity (Morgan et al., 1999; Perry et al., 2002). Studies have consistently supported the specificity and sensitivity of the SCOFF in clinical (Hill et al., 2010; Luck et al., 2002; Mond et al., 2008; Morgan et al., 1999) and university (Rueda Jaimes et al., 2005) samples, though it performs slightly worse than the EDE-Q (Mond et al., 2008)

***2.4.1.iii Drug abuse screening test - 10 (DAST-10).*** (Skinner, 1972). The 10-item short form of the Drug Abuse Screening Test (DAST; Skinner, 1972), referred to as the DAST-10, is a brief screening tool that provides a quantitative index of drug misuse – other than alcohol – over the past 12 months. The questions ask about drug use (e.g., Do you abuse more than one drug at once?), loss of control during drug use (e.g., Are unable to stop abusing drugs when you want to?), physical (e.g., Have you had medical problems as a result of your drug use?) and psychological (e.g., Do you ever feel bad or guilty

about your drug use?) consequences of drug use, and drug-related problems (e.g., Have you ever neglected your family because of your use of drugs?). All questions are presented as "yes" or "no" responses and summing the number of yeses yields a total score from 0-10. A score of 1 or 2 indicates a low level of drug related problems, while scores from 3-5 indicate a moderate degree of problems related to drugs. It is advised that individuals who report scores between 6 and 10 receive intensive assessment. This is broken down into individuals reporting substantial levels of problems related to drugs (6-8) and severe levels of problems related to drugs (9-10). When determining the optimal cut-off point there appears to be a sensitivity-specificity trade off with greater sensitivity and lower specificity using lower cut-offs and lower sensitivity but greater specificity using higher cut-offs (Cocco & Carey, 1998). Cocco and Carey (1998) argued that 3 was the optimal cutoff point (sensitivity = 74%; specificity = 86%; hit rate = 77%); however, in the current study we felt that specificity was more important than sensitivity. Namely, we did not want to inaccurately exclude someone because of a false positive score. As such, we opted for a cut-off of 5 which had a specificity of 90% (Cocco & Carey, 1998) and corresponds to the point where intensive examination is encouraged. Despite the low sensitivity of this cut point, the hit rate was still 79% (Cocco & Carey, 1998). The reliability and validity of the DAST-10 has been well established (for comprehensive review see Yudko, Lozhkina, & Fouts, 2007).

***2.4.1.iv Alcohol use disorders identification test (AUDIT).*** (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). The alcohol use disorders identification test (AUDIT) is a screening tool used to detect hazardous alcohol use. The AUDIT was

developed as part of a six country[15] collaborative project initiated by the World Health Organization (WHO). Ten questions were selected to reflect four aspects[16] of hazardous drinking over the past 12 months: (1) alcohol consumption (e.g., How often do you drink more than six drinks on one occasion?), (2) drinking behaviour (e.g., How often … have you found that you were not able to stop drinking once you had started?), (3) adverse reactions to alcohol (e.g., How often… have you had a feeling of remorse or guilt after drinking?), and (4) alcohol related problems (e.g., Have you or someone else been injured as a result of your drinking). The first eight items are scored on a five-point scale ranging from 0 (never) to 4 (daily). For the "alcohol related problems" questions (the final two questions) individuals are asked to select one of three responses: no (0), yes, but not in the last year (2), or yes, during the last year (4). A total score is computed by determining the sum of the ten questions. Thus, scores range from 0 to 40. The validity and reliability of the AUDIT have been well established. The AUDIT has demonstrated good convergent validity, correlating highly with other measures of problem alcohol use (Bohn, Babor, & Kranzler, 1995; Hays, Merz, & Nicholas, 1995) as well as biochemical markers of prolonged alcohol consumption (for discussion see Allen, Litten, Fertig, & Babon, 1997). In addition the AUDIT has demonstrated discriminant (Hays et al., 1995) and predictive (Claussen & Aasland, 1993; Congrave, Sunders, & Reznik, 1995) validity. Studies reporting Cronbach's alpha suggest good to excellent internal consistency (Allen, Litten,

[15] The six countries involved in the development of the AUDIT were Australia, Bulgaria, Kenya, Mexico, Norway, and the USA.

[16] In the current AUDIT manual (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001) the questions have been regrouped into three domains. Hazardous alcohol use includes items on the alcohol consumption factor (questions 1-3). Dependence symptoms include items on the drinking behaviour factor (questions 4-6). Harmful alcohol use includes items on both the adverse reactions and alcohol related factors (questions 7-10).

Fertig, & Babor, 1997; Saunders et al., 1993) and one study has established test-retest reliability (Sinclair, McRee, & Babon, 1992).

In the development of the AUDIT, receiver operating characteristic (ROC) analysis was used to determine cut-off value(s) that would maximize sensitivity and specificity. The analysis yielded two cut-offs, 8 and 10 (Sanders et al., 1993). The authors reported that of the individuals reporting hazardous alcohol use, 92% had an AUDIT score greater than or equal to 8. On the other hand, 94% of individuals who did not report hazardous alcohol use had an AUDIT score less than or equal to 8. Overall specificity results were similar when a cutoff of 10 was used; however, sensitivity scores decreased (Saunders et al., 1993), suggesting that 8 may be a better cut-off. Further, the AUDIT manual provides intervention guidelines for health professionals based on total AUDIT scores: (1) Zone I (0-7): alcohol education; (2) Zone II (8-15): simple advice; (3) Zone III (16-19): simple advice plus brief counseling and continued monitoring; (4) Zone IV (20-40): referral to specialist for diagnostic evaluation or treatment. While a cut-off of 8 provides the best sensitivity and specificity, we noted that it is possible to exceed this value simply by providing high responses to the first three questions (alcohol consumption questions) but not endorsing any of the remaining seven questions. Since distress and/or impairment (as would be measured by the last seven questions) is necessary for the diagnosis of any mental illness (APA, 2013), we felt that a cut-off of 8 was too conservative. In fact where alcohol consumption is not a diagnostic criterion for alcohol use disorder (APA, 2013), it is possible that using a cut-off of 8 an individual could have been mistakenly excluded from the study based on frequent alcohol consumption. This concern was exacerbated by the importance of drinking in

Newfoundland culture. In 2014, NL reported the greatest provincial alcohol sales per capita at $948.30, $252 above the national average (Statistics Canada, 2014), highlighting the importance of alcohol in NL culture relative to other provinces. Thus, we selected a more liberal cut-off of 16 for two reasons: (1) in order to achieve a score this high an individual would need to endorse at least one of the indicators of distress or impairment so individuals only reporting high frequency would not be excluded and (2) this is the point at which the WHO recommends counseling (Babor et al., 2001). Individuals who scored above this cut-off were not eligible to participate in the current study due to possible hazardous drinking which could interfere with treatment outcome.

**2.4.2 Baseline measures.** Given the number of questionnaires administered as part of this study it was not possible to counterbalance the measures. Having said this, there was no reason to suspect that any of the measures would influence responding on subsequent measures. As such all individuals completed the demographics questionnaire followed by the remaining measures in alphabetical order (BSI, DERS, EDE-Q, ISI, UPPS-P).

***2.4.2.i Demographics.*** A demographics questionnaire was administered at baseline assessing sex at birth, gender, age, body weight/ BMI, weight history, dieting history, treatment history (in the BED group), marital status, education level, ethnicity, onset of binge eating (in the BED group), and health history.

***2.4.2.ii Difficulties in Emotion Regulation Scale.*** (Gratz & Roemer, 2004) The Difficulties in Emotion Regulation Scale (DERS) is a 36-item self-report questionnaire that examines aspects of emotion dysregulation. Each question is scored on a five-point scale ranging from “Almost Never” to “Almost Always”. Items are recoded so that higher

scores always reflect greater difficulties in emotion regulation. As discussed earlier, the DERS provides a total score, as well as six subscale scores: (1) CLARITY: lack of emotional clarity (e.g., "I have difficulty making sense of my feelings"); (2) AWARE: lack of awareness of emotional states and responses (e.g., Reverse scored item: "I am attentive to my feelings"); (3) IMPULSE: difficulties controlling impulsive behaviour when experiencing negative or distressing emotions (e.g., "When I'm upset I lose control over my behaviour"); (4) GOALS: difficulties engaging in goal directed behaviour under negative or distressing emotions (e.g., When I'm upset I have difficulties concentrating"); (5) NON-ACCEPT: inability to accept emotional states (e.g., "When I am upset I feel ashamed for feeling that way"); (6) STRATEGIES: inability to access emotion regulation strategies (e.g., "When I'm upset I believe I will feel that way for a long time"). Total and subscale scores are calculated by summing the scores reported for each item on the corresponding scale. The reliability and validity of the DERS have been well established. Research has found that the DERS demonstrates good test-retest reliability (Gratz & Roemer, 2004), construct and predictive validity (Fox, Hong, & Sinha, 2008; Gratz, Paulson, Jukupcak, & Tull, 2009; Gratz & Roemer, 2004; Gratz & Roemer, 2008), convergent and discriminant validity (Baer, Smith, Hopkins, Krietemeyer, & Toney, 2006; Roemer, Lee, Salters-Pedneault, Erisman, & Mennin, 2009), and high internal consistency (Neumann, van Lier, Gratz, & Koot, 2010; Gratz & Roemer, 2004). Moreover, the psychometric properties of the DERS have been established across demographic groups including sex and ethnicity (Ritschel, Tone, Schoemann, & Lim, 2015). In the current study, the total DERS score demonstrated excellent internal

consistency (*Cronbach's alpha* = .956). Internal consistency for subscale scores was also good, ranging from .830 (Clarity) to .934 (Strategies).

***2.4.2.iii UPPS-P Impulsivity Scale.*** (Lynam, Smith, Whiteside, Fischer, & Cyders, 2007) The UPPS-P impulsivity scale is a 59-item scale, which measures five aspects of impulsivity: (1) negative urgency, (2) positive urgency, (3) (lack of) premeditation, (4) (lack of) perseverance and (5) sensation seeking. Negative urgency refers to the tendency to act rashly in response to distress and includes items such as "When I feel bad, I will often do things I later regret to make myself feel better now". Relatedly, positive urgency is the tendency to behave rashly in response to unusually positive emotions. For example, one item included in this subscale is "When I am very happy I can't stop myself from doing things that can have bad consequences". Premeditation refers to the tendency to think and reflect on the consequences of an action before acting. An example of premeditation would be "I have a reserved and cautious attitude towards life", however as mentioned above, impulsive behaviour is defined by a lack of premeditation. Similarly, perseverance is defined positively – the ability to remain focused on a task even if it is difficult or boring (e.g., "I generally like to see things through to the end) – though individuals with increased impulsivity demonstrate a lack of perseverance. Finally, sensation seeking encompasses two components: (1) the tendency to enjoy and seek out activities that are exciting and (2) openness to trying new experiences that may or may not be dangerous. An example of an item on this subscale would be "I generally seek new and exciting experiences and sensations". Each item is scored from 1 (agree strongly) to 4 (disagree strongly), and as most items are reverse scored, higher scores reflect greater impulsivity. Subscale scores are reported as means by

summing all of the items on the subscale and dividing by the number of items. The validity and reliability of the UPPS-P have been established in both males and females (Cyders, 2013). Cyders and colleagues (2007) found that the UPPS-P demonstrated good-excellent internal consistency, as well as criterion and convergent validity. The UPPS-P has also demonstrated good test-retest reliability (Weafer, Baggot, & deWit, 2013). In the current study, the UPPS-P scale demonstrated excellent internal consistency (*Cronbach's alpha* = .944), with subscale alphas ranging from .840 (Premeditation) to .953 (Positive urgency).

***2.4.2.iv Eating Disorder Examination – Questionnaire.*** (Fairburn, 2008) The Eating Disorder Examination – Questionnaire (EDE-Q) is a 28-item self-report questionnaire derived from the EDE interview, which has demonstrated good test-retest reliability and internal consistency (Berg et al., 2012). The EDE-Q has shown good test-retest reliability (Luce & Crowther, 1999; Reas, Grilo, & Masheb, 2006), temporal stability (Bardone-Cone & Agras, 2007; Mond, Hay, Rodgers, Own, & Beaumont, 2004a), and internal consistency (Bardone-Cone & Agras, 2007; Luce & Crowther, 1999; Mond et al., 2004a; Peterson et al., 2006) with Cronbach's alpha ranging from .70 to .93 (Berg et al., 2012). Moreover, the EDE-Q differentiates between individuals with and without eating disorders (i.e., criterion validity; Elder et al., 2007; Engelsen & Laberg, 2001; Mond, Hay, Rodgers, Owen, & Beaumont, 2004b; Wilson, Nonas, & Rosenblum, 1993) and converges with related measures (Bardone-Cone & Agras, 2007) and food diary reports (Grilo, Masheb, & Wilson, 2001). Factor analysis of the EDE-Q however, has been more variable (Berg et al., 2012; Grilo et al., 2010). To date no studies have provided evidence for the four-factor structure initially proposed by Fairburn (2008).

For the current study, a modified seven-item, three-factor version of the EDE-Q was used as it has been validated in both men and women (Grilo, Reas, Hopwood, & Crosby, 2015) and in obese bariatric surgery candidates (Grilo, Henderson, Bell, & Crosby, 2013). Moreover, the same seven-item three-factor structure has emerged for the EDE interview when examining patients with BED (Grilo et al., 2010), suggesting that it is an appropriate measure for our study. Grilo and colleagues (2015) have shown that this version of the EDE-Q consists of three subscales: (1) dietary restraint (e.g., Have you been consciously trying to restrict the amount of food you eat to influence shape or weight regardless of whether or not you have been successful?); (2) body dissatisfaction (e.g., How dissatisfied have you felt about your weight/shape?); (3) overvaluation of shape and weight (Has your weight/shape influenced how you think about (judge) yourself as a person?). All questions are scored on a seven-point scale; however, the Dietary Restraint scale examines frequency of behaviours over the past four weeks (0 = 0 days to 6 = everyday), while the Body Dissatisfaction and Overvaluation scales examine the severity of experiences ranging from 0 (not at all) to 6 (extremely). Scores are determined by taking the average of all the items on each subscale. A Global score can then be computed by taking the average of the subscale scores. Higher scores reflect greater pathology. Reliability analyses for the 7-item short form of the EDE-Q demonstrated good internal consistency for the total score (*Cronbach's alpha* = .837), as well as the dietary restraint (*Cronbach's alpha* = .877) and body dissatisfaction (*Cronbach's alpha* = .884) subscales. The remaining subscale, overvaluation of weight and shape, demonstrated excellent internal consistency (*Cronbach's alpha* = .965) in the current study.

***2.4.2.v Insomnia Severity Index.*** (Bastien, Vallieres, & Morin, 2001). The Insomnia Severity Index (ISI) is a seven-item self-report questionnaire measure of perceived sleep difficulties. Each item is scored on a five-point likert scale with higher scores reflecting more severe insomnia symptoms. Difficulties falling asleep, staying asleep, and waking up too early are measured from 0 (none) to 4 (very severe). The remaining four questions ask about dissatisfaction, noticeability, worry/distress, and interference of sleep problems. These four items are rated from 0 (not at all) to 4 (very much). An individual's score on the ISI is determined by taking the sum of the seven items yielding a total score from 0-28. The reliability and validity of the ISI have been well established. This scale demonstrates good internal consistency (Chahoud, Chachine, Salemeh, & Sauleau, 2017; Morin, Belleville, Belanger, & Ivers, 2011), test-retest reliability (Chahoud et al., 2017), convergent and discriminant validity (Bastien et al., 2001; Morin et al., 2011), and is sensitive to changes in perceived sleep difficulties following treatment (Bastien et al., 2001). Moreover, scores on the ISI converge with reports by clinicians and significant others (Bastien et al., 2011). In the current study, internal consistency for the ISI was excellent with *Cronbach's alpha* at .920.

***2.4.2.vi Brief Symptom Inventory.*** (Derogatis & Melisaratos, 1983) The Brief Symptom Inventory (BSI) is a 53-item self-report symptom inventory adapted from its longer parent version, the SCL-90-R, that measures how often an individual has been bothered by and the intensity of various symptoms in the past week. Although other measures target the symptoms of depression more clearly, the BSI was selected as an indicator of distress in the larger randomized controlled trial. In order to minimize participant burden, we opted to use the depression scale provide by this measure rather

than including another questionnaire. In the current study this time frame was changed to the past four weeks, to correspond with the EDE-Q. Items are scored on a five-point scale ranging from 0 ("not at all") to 4 ("extremely"). The BSI assesses nine symptom groups: (1) somatization: psychological distress arising from the perception of bodily dysfunction (e.g. cardiovascular complaints); (2) obsessive-compulsive: intrusive, unremitting thoughts accompanied by compulsive responses (e.g., checking/rechecking); (3) interpersonal sensitivity: feelings of inadequacy and inferiority (e.g., self-deprication during social interactions); (4) depression: a broad range or symptoms indicative of clinical depression (e.g., dysphoria); (5) anxiety: symptoms associated with clinical anxiety (e.g., restlessness); (6) hostility: includes hostile thoughts, feelings and actions (e.g., urges to break things); (7) phobic anxiety: symptoms that occur in phobic states or agoraphobia (e.g., phobias towards open spaces); (8) paranoid ideation: views paranoid phenomena as a *mode of thinking* (e.g., projection); (9) psychoticism: reflects a continuum ranging from "mildly alien life…to floridly psychotic status" (Derogatis & Melisaratos, 1983, p. 597; e.g., the idea that something is wrong with your mind). The current study used only the BSI depression subscale score as an indicator of the severity of depressive symptoms, which is determined by taking the average of the items included on the depression subscale (Items #9, 16, 17, 18, 35, and 50). The BSI has demonstrated very good test-retest reliability and internal consistency, as well as convergent and construct validity (Derogatis & Melisaratos, 1983). In the current study, internal consistency for the depression subscale of the BSI was good (*Cronbach's alpha* = .886). At the end of the BSI we also included "Item 9: Suicidal Thoughts or Wishes" from the Beck Depression Inventory II (BDI-II; Beck et al., 1961) to identify any participants at

risk of suicide. Participants who responded ‘2’ (I would like to kill myself) or ‘3’ (I would kill myself if I had the chance) to this question were to be contacted by one of the primary investigators on the project who are both registered psychologists. Since no participants indicated risk of suicide no participants were contacted. Studies have demonstrated that the BDI-II exhibits good reliability and validity across both sexes (Dozois, Dobson & Ahnberg, 1998).

**2.5 Data Analysis**

All data were analyzed using IBM SPSS software and significance was determined at $\alpha = .05$. To determine whether difficulties in emotion regulation and impulsivity differed between individuals with and without BED, DERS and UPPS-P scores were compared across the three groups (BED, NW, OW) using one-way analyses of variance (ANOVAs). Post-hoc tukey tests were conducted to determine where (if any) differences occurred. Effect sizes are reported as partial eta squared for the overall analysis (as reported by SPSS) and Cohen’s d for pairwise differences. To determine Cohen’s d, equality of variance was examined using Levene’s test to determine which variance term to use. If Levene’s test was not violated, a pooled standard deviation term was used to compute the effect size. If Levene’s test was violated – indicating unequal variances – the greater of the two standard deviations was used to compute Cohen’s d rendering the most conservative estimate. Table 19 indicates which effect size analyses used the pooled variance term.

Subsequently, regression analyses were conducted on data from the BED group with emotion regulation (DERS total score) as the predictor variable and binge frequency or EDE-Q Global score as the outcome variable. Since the bivariate regressions were

significant, hierarchical regression analyses were conducted controlling for sex, BMI, and BSI depression score. In this analysis, binge frequency or EDE-Q total score was entered as the criterion while DERS total score, age, BMI, and BSI depression score were entered as the predictors. Age and BMI were entered in the first block, BSI depression score in the second block, and DERS total score in the final block.

A series of moderation analyses were then conducted examining the interaction between depression and DERS, as well as insomnia symptom severity and DERS. Two separate analyses were conducted for each interaction (i.e., BSI x DERS and ISI x DERS), one with binge frequency as the outcome variable and one with EDE-Q Global score as the outcome variable. In all cases variables were centered before running the regression. In block one, the two centered predictors (either DERS and ISI score *OR* DERS and BSI score) were entered. In the second block the corresponding interaction term was entered (DERS x ISI or DERS x BSI). Post hoc analyses were conducted for significant interactions by re-centering one of the variables such that the zero point indicated one standard deviation above or below the mean and examining the association between the other variable and the criterion. Finally, in order to confirm that the effects reported here were not the consequence of differences in overvaluation of weight and shape, all of the above analyses were rerun including overvaluation of weight and shape as a covariate (mean difference analyses) or predictor (regression analyses). While overvaluation of weight and shape could have been included in the first series of analyses, re-running the analyses including overvaluation allowed us to identify the precise role of overvaluation of weight and shape given the importance of this factor in BED (Grilo, 2013).

# 3.0 Results

## 3.1 Recruitment

**3.1.1 NED group.** Two hundred and forty-nine individuals completed the screening questionnaire for the control group. Of these, 111 (44.6%) reported at least one exclusion criterion and were not eligible to participate in the study. The most common reasons for ineligibility were self-reported binge eating episodes (49.5%) and current/past eating disorder diagnosis (13.5%). A breakdown of the reasons for ineligibility is presented in Table 1. The remaining 138 individuals met all inclusion criteria and were sent an email informing them of their eligibility. Eighty-three individuals (60.1%) completed the questionnaires and 53 (38.4%) did not. Two individuals were eligible to participate in the study but did not provide a valid email address (1.5%). One participant who did not meet criteria for the larger study but was eligible for the control group also completed the questionnaires for a total of 84 respondents. After examining the completed responses, an additional four individuals were removed because current BMI was less than 18.5 leaving a final sample of 80 participants in the NED group. Approximately half of individuals (n = 38; 47.5%) in this group reported a BMI between 18.5 and 25 and were categorized as normal weight (NW). The remaining 42 participants (52.5%) reported a BMI of 25 or greater and were categorized as overweight (OW). A consort diagram outlining recruitment of the control groups is presented in Figure 4a.

**3.1.2 BED group.** Five hundred and thirty-seven individuals completed the screening questionnaire for the intervention study. At the time of data analysis for the current study however, only 529 individuals (98.5%) had received an email detailing their eligibility. As such, only these individuals are discussed here. Two hundred and forty

three individuals (45.9%) were ineligible to participate in the study; the reasons for ineligibility following screening are displayed in Table 2. The most common reason for ineligibility prior to the telephone interview was not reporting objective binge episodes (n = 64; 26.3%). The remaining 286 individuals (54.1%) met the initial inclusion criteria for the study and were sent an email offering a telephone interview to confirm eligibility. One hundred and thirty-three individuals (46.5%) completed the telephone interview in addition to three individuals recruited from NED screening. Thus, a total of 136 individuals participated in the telephone interview. Sixty-nine individuals (51.9%) who completed the telephone interview were not eligible to partake in the current study. The majority, n = 64 (92.8%), were ineligible because they did not meet the DSM 5 criteria for a diagnosis of BED according to the EDE interview. A breakdown of the reasons for ineligibility following the telephone interview is provided in Table 3. The remaining 64 individuals (47.1%) met full inclusion criteria and were entered into the larger study. Given the deadline for my Master's thesis, only questionnaires completed before April 25, 2017 were analyzed for the current project. At that time, 62 of the 64 participants (96.9%) had completed the baseline questionnaires, yielding a total sample of 62 individuals in the BED group. A consort diagram outlining recruitment of the BED group is presented in Figure 4b.

**3.2 Descriptive Characteristics of the Sample**

The majority of the NED and BED samples was female (NW = 78.9%; OW = 88.1%; BED = 93.5%), Caucasian (NW = 89.4%; OW = 97.6%; BED = 96.8%), and single (NW = 52.6%) or married (OW = 57.1%; BED = 58.1%). A breakdown of the gender, ethnicity, and marital status of participants is provided in Table 4. Chi-squared

tests of independence revealed that there were no differences in biological sex, $\chi^2(2, n = 142) = 4.766$, $p = .092$, ethnicity, $\chi^2(8, n = 142) = 10.055$, $p = .261$, or marital status, $\chi^2(6, n = 142) = 5.513$, $p = .480$ between groups, and as such these variables were not considered as covariates in subsequent analyses. Individuals in the NW and OW groups reported a mean age of 31.34 years (*SD* = 12.796) and 37.55 years (*SD* = 14.681) respectively, while individuals in the BED group reported a mean age of 40.29 years (*SD* = 11.576). A one-way ANOVA revealed that age was significantly different across the three groups, $F(2, 139) = 5.722$, $p = .004$, $\eta^2_p = .076$. Individuals in the BED group were significantly older than individuals in the NW group, $p = .003$, *Cohen's d* = 0.750, though the OW group did not differ from the NW or BED groups, $p > .537$. Mean age for the three groups is presented in Table 5.

As expected, a one-way ANOVA revealed a significant difference in BMI across groups, $F(2, 139) = 61.156$, $p < .0005$, $\eta^2_p = .468$. Individuals in the BED group (*M* = 37.600, *SD* = 9.550) reported significantly higher BMIs than individuals in both the OW (*M* = 31.023; *SD* = 4.575), $p < .0005$, *Cohen's d* = 0.689, and NW (*M* = 21.984, *SD* =1.815), $p < .0005$, *Cohen's d* = 1.635, groups. Moreover, individuals in the OW group reported a significantly higher mean BMI than the NW group, $p < .0005$, *Cohen's d* = 1.976. The groups also differed in terms of highest, $F(2,135) = 58.880$, $p < .0005$, $\eta^2_p = .466$, and lowest, $F(2, 135) = 18.079$, $p < .0005$, $\eta^2_p = .209$, adult BMI. Current, lowest adult, and highest adult BMI for the three groups are presented in Table 5.

Consistent with the difference in BMI across groups, there was a significant difference in the proportion of individuals who responded 'yes' when asked whether they currently consider themselves to be overweight, $\chi^2(2, n = 142) = 99.192$, $p < .0005$,

*Cramer's V* = .836. Post hoc analyses revealed that individuals in the BED and OW groups (92.3%) were significantly more likely to self-report being overweight than individuals in the NW groups (5.26%), $\chi^2(1, n = 142) = 98.607$, $p < .0005$, *Cramer's V* = .833. Moreover, there was a difference in the proportion of individuals who had previously gone on a weight loss diet across groups, $\chi^2(2, n = 142) = 31.466$, $p < .0005$, *Cramer's V* = .471. Again, individuals in the OW and BED groups (91.3%) were more likely to report having gone on a diet in the past than individuals in the NW group (50.0%), $\chi^2(1, n = 142) = 30.055$, $p < .0005$, *Cramer's V* = .460. These data are presented in Table 6.

**3.3 Group Differences in Eating Disorder Psychopathology**

Individuals in the BED group reported an average of 53.226 (*SD* = 56.500) binge eating episodes over the past three months. Since individuals in the NED group did not complete the EDE interview or report binge eating episodes, the groups were compared on EDE-Q Global and subscale scores (as a measure of eating disorder psychopathology) using one-way ANOVAs to gain evidence that the BED group was in fact a clinical sample. There was a significant difference in Global EDE-Q score, $F(2, 139) = 48.126$, $p < .0005$, $\eta^2_p = .409$, with individuals in the BED group ($M = 4.482$, $SD = 0.918$) reporting significantly higher EDE-Q Global scores – indicating greater eating disorder psychopathology – than individuals in both the NW ($M = 2.003$, $SD = 1.383$), $p < .0005$, *Cohen's d* = 1.792, and the OW ($M = 3.303$, $SD = 1.476$), $p < .0005$, *Cohen's d* = 0.799, groups. Individuals in the OW group also reported higher EDE-Q total scores than individuals in the NW group, $p < .0005$, *Cohen's d* = 0.881, suggesting that EDE-Q scores increase with BMI. After controlling for BMI in an analysis of covariance

(ANCOVA) however, EDE-Q total scores still differed significantly across groups, $F(2, 138) = 17.292$, $p < .0005$, $\eta^2_p = .200$ (Figure5a). Carter, Stewart, and Fairburn (2001) reported that EDE-Q Global and subscale scores greater than or equal to 4 indicate clinically significant eating disorder psychopathology (as cited in Panelo, Negrete, Portell, & Raich, 2013). Based on mean EDE-Q Global score, only the BED group reported clinically significant eating disorder psychopathology according to Carter et al. (2001). Consistently, after individuals were classified as either reporting clinically significant eating disorder psychopathology (EDE-Q Global score $\geq 4$) or not (EDE-Q Global score $< 4$), a chi-squared test of independence revealed that there was a significant difference in the proportion of individuals reporting a Global score greater than or equal to 4, $\chi^2(2, n = 142) = 38{,}754$, $p < .0005$, *Cramer's V* = .522. Follow-up analyses revealed that individuals with BED (72.6%) were more likely to report clinically significant EDE-Q Global scores than individuals in the NED group (23.75%), $\chi^2(1, n = 142) = 33.642$, $p < .0005$, *Cramer's V* = .487 (see Table 7). Thus, we can conclude with adequate certainty that the BED sample in the current study was in fact a clinical sample.

There was also a significant difference in scores on the EDE-Q Body Dissatisfaction subscale, $F(2,139) = 61.602$, $p < .0005$, $\eta^2_p = .470$, that remained after controlling for BMI, $F(2, 138) = 18.725$, $p < .0005$, $\eta^2_p = .213$. Similar to the total score, individuals in the BED group ($M = 5.484$, $SD = .815$) reported higher scores than individuals in the OW group ($M = 4.060$, $SD = 1.839$), *Cohen's d* = 0.774, who in turn reported greater scores than individuals in the NW group ($M = 2.237$, $SD = 1.663$), *Cohen's d* = 0.991 (Figure 5b). Scores on the Dietary Restraint subscale were also significantly different across groups, $F(2,139) = 6.804$, $p = .002$, $\eta^2_p = .089$, and post hoc

Tukey's HSD tests revealed that individuals in the NW group ($M$ = 1.746, $SD$ = 1.889) reported lower scores than individuals in the OW ($M$ = 2.873, $SD$ = 2.077), $p$ = .037, *Cohen's d* = 0.574, and the BED ($M$ = 3.269, $SD$ = 2.063), $p$ = .765, *Cohen's d* = 0.385, groups, and this difference persisted after controlling for BMI, $F(2, 138) = 3.640$, $p$ = .029, $\eta^2_p$ = .050 (Figure 5c). Although dietary restraint is an aspect of eating disorder psychopathology depending on the nature and degree, none of the groups reported clinically significant dietary restraint scores as defined by Carter and colleagues (2001). In the current study dietary restraint appears to differentiate NW individuals from those who are OW rather than indicating eating disorder pathology.

The only subscale that significantly differentiated individuals with BED from those in the NED control groups was EDE-Q Overvaluation of Weight and Shape (Figure 5d). A one-way ANOVA revealed a significant difference in scores on this subscale across groups, $F$ (2, 139) = 27.771, $p < .0005$, $\eta^2_p$ = .286, which persisted after controlling for BMI, $F(2, 138) = 10.722$, $p < .0005$, $\eta^2_p$ = .134. Namely, individuals in the BED group ($M$ = 4.694, $SD$ = 1.535) reported greater (and clinically significant) overvaluation of weight and shape compared to individuals in the NW ($M$ = 2.062, $SD$ = 1.896), $p <$ .0005, *Cohen's d* = 1.407, and OW ($M$ = 2.976, $SD$ = 2.092), $p < .0005$, *Cohen's d* = 0.821, groups, while the NW and OW groups did not differ from each other, $p$ = .053. This is consistent with overvaluation of weight and shape as a diagnostic criterion for other eating disorders (i.e., AN and BN; APA, 2013) and provides evidence for Grilo and colleagues' (2008) argument that overvaluation should be – at minimum – a diagnostic specifier of BED if not a diagnostic criterion. BSI Depression scores also differentiated between individuals in the BED group and NED groups, $F(2, 139) = 8.0681$, $p < .0005$,

$\eta^2_p$ = .104. As expected, individuals in the BED group (*M* = 1.393, *SD* = .811) reported higher BSI Depression scores than individuals in both the NW (*M* = .781, *SD* = .721), *p* = .001, *Cohen's d* =0.794, and the OW (*M* = .889, *SD* = .930), *p* = .008, *Cohen's d* =0.591, groups. Clinical characteristics of the sample are presented in Table 8.

**3.4 Individuals with BED Report Greater Difficulties in Emotion Regulation**

Given evidence for increased difficulties in emotion regulation in BED (Danner et al., 2014; Harrison et al., 2016; Robinson et al., 2015; Svaldi et al., 2012; Whiteside et al., 2007) and studies of DBT for BED (Masson et al., 2013; Safer et al., 2010; Telch, 1997; Telch et al., 2000; Telch et al., 2001), the first goal of this project was to examine whether individuals with BED would report greater difficulties in emotion regulation (as measured by the DERS) compared to NW and OW individuals with no history of an eating disorder (NED). There was a significant difference between the three groups in total DERS scores, $F(2, 139) = 19.259$, $p < .005$, $\eta^2_p = 0.217$, with individuals in the BED group (*M* = 99.332, *SD* = 23.815) reporting higher scores (indicating greater difficulties in emotion regulation) than individuals in the NW (*M* = 75. 308, *SD* = 21.679), *p* < .0005, *Cohen's d* = 1.054, and OW (*M* = 74.805, *SD* = 23.269), *p* < .0005, *Cohen's d* = 1.048, groups, indicating large effect sizes. The NW and OW control groups did not differ significantly from each other, *p* = .995. This difference was still significant after controlling for age, BMI, and BSI depression scores, $F(2,136) = 7.606$, *p* = .001, $\eta^2_p = 0.101$, suggesting that individuals with BED do in fact report greater difficulties in emotion regulation and that this is not better accounted for by differences in BMI, age, or depression scores.

Furthermore, with the exception of the Clarity subscale (being clear about what one is feeling), $F(2, 139) = 2.425$, *p* = .092, there were significant differences across

groups on all of the DERS subscales (Figure 6). The BED, NW, and OW groups reported significantly different scores on the Non-acceptance, $F(2,139) = 16.260$, $p < .0005$, $\eta^2_p = .190$, Impulsivity, $F(2, 139) = 14.707$, $p < .0005$, $\eta^2_p = 0.175$, Awareness, $F(2, 139) = 7.544$, $p = .001$, $\eta^2_p = .098$, and Strategies subscales, $F(2, 139) = 17.323$, $p < .0005$, $\eta^2_p = .200$. Based on Tukey's HSD tests, individuals in the BED group reported higher scores than those in both the NW (all $p < .002$; all *Cohen's d* > 0.712) and OW (all $p < .010$; all *Cohen's d* > 0.591) control groups indicating moderate to large effect sizes. There was also a significant difference in scores on the Goals subscale, $F(2, 139) = 5.334$, $p = .006$, $\eta^2_p = .071$, though individuals in the BED group only reported higher scores compared to the OW group, $p = .004$, *Cohen's d* = 0.660. On this subscale, there were no differences between the NW and OW groups, $p = .149$, or the BED and NW groups, $p = .502$. Mean scores and effect sizes for the DERS total and subscale scores are presented in Tables 9 and 10. After controlling for age, BMI, and BSI depression scores, significant differences remained on the Non-acceptance, $F(2, 136) = 5.430$, $p = .005$, $\eta^2_p = 0.074$, Goals, $F(2, 136) = 3.357$, $p = .038$, $\eta^2_p = .047$, Impulsivity, $F(2, 136) = 3.570$, $p = .031$, $\eta^2_p = .050$, and Strategies, $F(2, 136) = 6.304$, $p = .002$, $\eta^2_p = .085$, subscales. Differences on the Aware subscale were no longer significant after controlling for covariates, $F(2, 136) = 2.931$, $p = .057$. These results are presented in Table 9. Overall, the results indicated that individuals with BED experience greater difficulties in emotion regulation, specifically non-acceptance of emotions, difficulty maintaining goal directed behaviour while experiencing difficult emotions, and difficulty accessing appropriate emotion regulation strategies.

**3.5 Individuals with BED Report Greater Negative and Positive Urgency**

Given the role of negative/positive urgency in emotion regulation (Cyders & Smith, 2009; Gratz & Roemer, 2004), we also sought to examine differences in impulsivity in individuals with BED and NED controls. We examined all five subscales of the UPPS-P and total score to determine whether BED-related deficits in impulsivity were global or limited to the urgency subscales (Figure 7). Prior to controlling for covariates, there were significant differences in scores across groups on all five subscales: Negative Urgency, Premeditation, Perseverance, Sensation Seeking, and Positive Urgency (all $F(2, 139) > 4.568$, all $p < .012$). After controlling for age, BMI, and BSI depression score however, only the differences on the Negative Urgency, $F(2, 136) = 9.912$, $p < .0005$, $\eta^2_p = .127$, and Positive Urgency, $F(2, 136) = 7.017$, $p = .001$, $\eta^2_p = .094$, subscales remained significant, as would be predicted by the emotion regulation model. Specifically, individuals in the BED group reported significantly higher negative ($M = 2.836$, $SD = 0.528$) and positive ($M = 2.046$, $SD = 0.676$) urgency scores relative to both the NW (negative urgency: $M = 2.026$, $SD = 0.569$; positive urgency: $M = 1.462$, $SD = 0.488$) and OW (negative urgency: $M = 2.206$, $SD = 0.670$; positive urgency: $M = 1.582$, $SD = 0.564$) control groups. Effect sizes for these comparisons were all large (*Cohen's d* ranged from 0.645 to 1.497) and are presented in Table 11. Total UPPS-P scores also differed significantly across groups, $F(2, 139) = 9.975$, $p < .0005$, $\eta^2_p = .126$, with individuals in the BED group ($M = 2.254$, $SD = 0.433$) reporting higher scores (indicative of greater impulsivity) than individuals in the NW ($M = 1.943$, $SD = 0.317$), $p = .001$, *Cohen's d* = 0.794, and OW ($M = 1.959$, $SD = 0.418$), $p = .001$, *Cohen's d* = 0.609, groups with moderate effect sizes. This difference remained significant after controlling for age, BMI, and BSI depression score, $F(2, 136) = 4.848$, $p = .009$, $\eta^2_p =$

.067. Since the total score on the UPPS-P includes all items from the subscales, it is reasonable to assume that the difference in total UPPS-P score was driven by differences on the urgency subscales. Mean total and subscale scores for the three groups are presented in Table 12. Thus, while individuals with BED do report greater impulsivity scores than their NED counterparts, these differences appear to be the result of increased negative and positive urgency in individuals with BED compared to both NW and OW controls.

### 3.6 Depression and Difficulties in Emotion Regulation Predict Binge Frequency and Eating Disorder Psychopathology Respectively in BED

Having demonstrated that individuals with BED reported greater difficulties in emotion regulation and impulsivity, we next sought to examine the relationship between DERS total score and severity of eating disorder symptoms (i.e., binge episode frequency) and psychopathology. This is important because while demonstrating that there are differences in emotion regulation is interesting, this does not provide insight as to how emotion regulation may be related to the etiology of the disorder. To better inform BED treatment, it is necessary to examine (and ultimately determine) how difficulties in emotion regulation may be related to binge eating and eating disorder psychopathology. Thus, a bivariate regression analysis was run with binge frequency as the criterion and DERS total score as the predictor. This regression analysis was significant, $R^2 = .157$ (*adjusted* $R^2 = .143$), $F(1, 60) = 11.161$, $p = .001$, with DERS total score positively predicting binge frequency, $b = .940$, $t(60) = 3.341$, $p = .001$. After controlling for age, BMI, and BSI depression score however, DERS total score no longer explained unique variance in binge frequency, $b = 0.514$, $t(60) = 1.482$, $p = .114$, though BSI depression

score remained a significant predictor, $b$ = 27.611, $t$(60) =2.762, $p$ = .008. Similarly, when EDE-Q Global score (a measure of eating disorder psychopathology composed of dietary restraint, overvaluation of weight and shape, and body dissatisfaction[17]) was entered as the criterion, DERS total score was a positive predictor, $R^2$ = .199 (*Adjusted* $R^2$ = .185), $F$(1, 60) = 14.889, $b$ =.017, $t$(60) = 3.859, $p$ < .0005. In contrast to binge frequency however, DERS total score remained a significant predictor of EDE-Q Global score even after controlling for age, BMI, and BSI depression score, $b$ = .014, $t$(60) = 2.360, $p$ = .022. Coefficients and t-values for these analyses are presented in Tables 13 (binge frequency as criterion) and 14 (EDE-Q Global score as criterion). In both tables, model 1 refers to the model including only DERS total score while model 2 includes BMI, age, and BSI depression score as control variables. Taken together the findings presented above suggest that depression is the best predictor of binge frequency while difficulties in emotion regulation is the best predictor of eating disorder psychopathology in BED.

### 3.7 The Relationship Between Difficulties in Emotion Regulation and Binge Frequency is Moderated by Depression Severity

In the above analyses, DERS and BSI depression scores were considered as independent variables. It is important, however, to consider that depression and emotion regulation strategies are likely to interact. For instance, an individual is more likely to require the use of emotion regulation strategies in the face of intense emotions such as depression. Therefore, a follow-up moderation analysis was conducted including the

[17] While there is a question on the EDE-Q that asks about binge frequency this is not captured in the EDE-Q Global score. This score only includes measures of dietary restraint, overvaluation of weight and shape, and body dissatisfaction, making it distinct from the measure of binge frequency.

DERS by BSI interaction as a moderator of the relationship between DERS and eating disorder symptoms (i.e., binge frequency and EDE-Q Global score). This analysis revealed a significant interaction between BSI depression score and DERS total score when binge frequency was entered as the criterion. BSI score and DERS score were entered in the first block of the model explaining 24.6% of variance in binge frequency, $R^2 = .246$, $F(2,59) = 9.603$, $p < .0005$. Adding the interaction term explained an additional 7.2% of variance and this change was significant, $\Delta R^2 = .072$, $F(1, 58) = 6.145$, $p = .016$. Examination of the interaction revealed that for individuals who reported higher BSI depression scores, DERS total score was a positive predictor of binge frequency, $b = 0.956$, $t(58) = 2.374$, $p = .016$. In individuals who reported lower BSI depression scores, this relationship was negative and non-significant, $b = -0.295$, $t(58) = -0.693$, $p = .491$. This relationship is presented in Figure 8. Thus, increased difficulties in emotion regulation are associated with greater binge frequency in individuals who report high (but not low) depression scores on the BSI. In contrast, when EDE-Q Global score (a measure of eating disorder psychopathology including dietary restrain, overvaluation of weight and shape, and body dissatisfaction) was entered as the criterion, the interaction was not significant, $b = -0.004$, $t(58) = -0.680$, $p = .499$, suggesting a more direct relationship between difficulties in emotion regulation and eating disorder psychopathology compared to binge frequency. Namely, difficulties in emotion regulation predict eating disorder psychopathology at all levels of depressive symptoms, but are only related to binge frequency at high levels of depressive symptoms. These findings are presented in Tables 15 and 16.

### 3.8 The Severity of Insomnia Symptoms Does Not Moderate the Relationship Between Difficulties in Emotion Regulation and Eating Disorder Psychopathology

To address the third research question, two moderation analyses were conducted to determine whether insomnia symptoms moderated the effect of sleep difficulties on eating disorder pathology, as has previously been documented in BPD (Selby, 2013). First a hierarchical regression was conducted entering DERS total score and ISI total score in the first block and the interaction term in the second block. When binge frequency was entered as the criterion, only DERS total score emerged as a significant predictor of binge frequency in both the first, $b = .847$, $t(59) = 2.994$, $p = .004$, and the second model, $b = .793$, $t(58) = 2.710$, $p = .009$. Neither ISI total score, $b = 1.754$, $t(58) = 1.686$, $p = .097$, nor the interaction, $b = .034$, $t(58) = .754$, $p = .454$, explained unique variance in the final model. Namely, in the final model DERS total score was a significant predictor of binge frequency, $b = 0.013$, $t(58) = 2.522$, $p = .014$, while ISI total score, $b = .024$, $t(58) = 1.302$, $p = .198$, and the interaction term, $b = .001$, $t(58) = 1.217$, $p = .228$, were not. A similar pattern of results emerged when EDE-Q Global score was entered as the criterion. These findings are presented in Tables 17 and 18. Thus, it appears that the severity of insomnia symptoms does not interact with difficulties in emotion regulation to predict binge frequency or eating disorder psychopathology in individuals with BED.

### 3.9 Controlling for Overvaluation of Weight and Shape Does Not Change the Pattern of Results

Grilo (2013) has argued that overvaluation of weight and shape should be included, at minimum, as a diagnostic specifier in BED in the DSM given that individuals

with BED who present with higher overvaluation of weight and shape also experience more severe psychopathology, highlighting an important role for overvaluation of weight and shape in BED. Thus, we added overvaluation of weight and shape as a predictor in the regression models to determine whether depression and difficulties in emotion regulation would explain unique variance above and beyond overvaluation. Moreover, since the BED, NW, and OW groups differed significantly in overvaluation of weight and shape we included this as a covariate in the mean difference analyses to confirm that differences in difficulties in emotion regulation and urgency could not be attributed to differences in overvaluation.

After controlling for overvaluation, individuals with BED still reported greater total DERS scores compared to NW and OW controls, $F(2, 135) = 5.594$, $p = .005$, $\eta^2_p = .007$. Similarly, significant differences on the Non-Accept, $F(2, 135) = 3.090$, $p = .049$, $\eta^2_p = .044$, Goals, $F(2,135) = 3.458$, $p = .034$, $\eta^2_p = .049$, and Strategies, $F(2, 135) = 3.640$, $p = .029$, $\eta^2_p = .051$, subscales persisted after controlling for overvaluation. Interestingly, while differences on the Impulse subscale were significant after controlling for BMI, age, and depression, they were no longer significant after controlling for overvaluation of weight and shape, $F(2, 135) = 2.607$, $p = .077$. On the other hand, the differences on the Aware subscale, which were not significant after controlling for BMI, age, and depression, became significant when overvaluation was added to the model, $F(2, 135) = 3.376$, $p = .037$, $\eta^2_p = .048$. As in the initial analysis, there was no difference in scores on the Clarity subscale between groups, $F(2, 135) = 0.512$, $p = .600$. Thus, after controlling for overvaluation a similar pattern of results emerges for the DERS total and subscale scores. On the UPPS-P subscale scores, the results after controlling for

overvaluation of weight and shape did not change. While there were no significant differences on the Premeditation, $F(2, 135) = 1.586$, $p = .209$, Perseverance, $F(2, 135) = 1.957$, $p = .145$, and Sensation Seeking, $F(2, 135) = 0.436$, $p = .648$, subscales, individuals with BED reported higher scores than their NED counterparts (regardless of BMI) on the Negative, $F(2,135) = 5.796$, $p = .004$, $\eta^2_p = .079$, and Positive, $F(2, 135) = 3.979$, $p = .021$, $\eta^2_p = .056$, urgency subscales. However, after controlling for overvaluation of weight and shape, differences in total UPPS-P score were no longer significant, $F(2, 135) = 2.942$, $p = .056$.

Because items on the Overvaluation subscale are also included in the EDE-Q Global score (suggesting colinearity), only the regression analyses with binge frequency as the outcome variable were reassessed after controlling for overvaluation of weight and shape. For the first analysis, BMI and age were entered into the first block, Overvaluation score in the second, BSI Depression score in the third, and DERS total score was entered into the final block. As in the initial analysis only BSI depression score was a significant predictor of binge frequency, $b = 27.296$, $t(56) = 2.652$, $p = .010$ (see Model 3 in Table 13). Next, we conducted the moderation analysis between BSI depression and DERS total score, with Overvaluation entered in the first block as a control variable. The interaction term explained an additional 7.4% of variance in binge frequency above the contributions of overvaluation of shape and weight, depression, and difficulties in emotion regulation, $F(1, 57) = 6.219$, $p = .016$. As in our initial analysis, it was found that in individuals who reported higher – but not lower, $p = .429$ – depression scores, DERS total score was a significant predictor of binge frequency, $b = .914$, $t(57) = 2.200$, $p = .032$. Finally, when overvaluation was added to the insomnia moderation analysis the interaction between

DERS total score and ISI total score was not significant, $t(57) = .815$, $p = .418$, and there were no significant predictors of binge frequency. The results after controlling for overvaluation of weight and shape, which are nearly identical to the results prior to controlling for this factor, suggest that the findings reported here cannot be accounted for differences in overvaluation of weight and shape, even though overvaluation of weight and shape may be associated with more severe psychopathology (Grilo, 2013; Mond et al., 2006).

## 4.0 Discussion

BED is the most prevalent eating disorder with rates between 1-3% in the general population (Dingemans et al., 2002; Hudson et al., 2007; Kessler et al., 2013) and 4-8% in obese community samples (Hudson et al., 2007). Despite the association between BED and obesity, individuals with BED experience greater physical and psychological impairments compared to their obese non-BED counterparts (Rieger et al., 2005). Our lab estimates that between 7,000 and 14,000 individuals in the province of NL currently have BED and yet there are no publicly funded services for BED. Given the clear need for treatment programs in the province, research must start by examining the etiology of this disorder to better understand its development and maintenance, thereby facilitating the dissemination of effective treatment approaches.

Even before the introduction of BED in the DSM, a number of theories had been proposed to explain binge eating in bulimia nervosa (BN), highlighting the importance of dietary restraint (Herman & Polivy, 1984) and negative affect (Herman & Polivy, 1988; Heatherton & Beaumeister, 1991). Since then, emotion regulation theory has emerged as a possible model of BED. Emotion regulation theory, initially proposed by Linehan

(1993) to explain borderline personality disorder (BPD), argues that individuals with BED experience deficits in emotion regulation skills. When these individuals experience intense emotions instead of implementing adaptive emotion regulation strategies (e.g., cognitive reappraisal; Campbell-Sills & Barlow, 2007) they turn to food to diminish or numb the emotion. While this strategy is effective in the short term, feelings of shame, guilt, and disgust typically surface after the episode perpetuating the cycle (Safer et al., in press; see Figure 3). While there has been an abundance of indirect support for this argument, primarily demonstrating that individuals with BED experience greater negative affect prior to binge eating (for review see Haedt-Matt & Keel, 2011), little research has empirically examined the hypotheses that individuals with BED experience greater difficulties in emotion regulation and that these deficits are associated with increased eating disorder pathology. Thus, the goal of the current study was to address these limitations by examining (1) emotion regulation deficits in a sample of individuals with BED compared to individuals with no history of an eating disorder (NED) and (2) how these deficits correspond to eating disorder pathology, namely binge frequency and EDE-Q Global score. A secondary goal was to examine the moderating effect of insomnia symptoms on difficulties in emotion regulation, given that this association has emerged in other disorders characterized by emotion regulation deficits (i.e., BPD; Selby, 2013).

Overall, our findings support an emotion regulation model of BED demonstrating that individuals with BED report greater difficulties in emotion regulation, as well as negative and positive urgency, compared to individuals with NED. Moreover, DERS total score predicted EDE-Q Global score in individuals with BED, suggesting that greater difficulties in emotion regulation are associated with increased eating disorder

psychopathology. Although difficulties in emotion regulation did not predict binge frequency as expected, we found that depression moderated the relationship between DERS total score and binge frequency. This is consistent with the emotion regulation model of BED. Namely, difficulties in emotion regulation were associated with binge frequency in individuals with higher (but not lower) depression scores, suggesting that the relationship between DERS total score and binge frequency is only evidenced in individuals who experience greater distress/emotionality. Although we did not find evidence that insomnia symptom severity was a moderator of the association between DERS total score and eating disorder pathology (neither binge frequency, nor EDE-Q Global score), this does not preclude the influence of sleep (and sleep disturbance) in BED. A detailed discussion of each of these findings is provided below. When the analyses were rerun including overvaluation of weight and shape as a covariate, the pattern of results was the same, supporting our hypotheses and suggesting that the results of the current study cannot be attributed to differences in overvaluation of weight and shape.

### 4.1 Individuals with BED Reported Greater Difficulties in Emotion Regulation

Emotional eating (e.g., Masheb & Grilo, 2006), stress and coping (e.g., Gluck et al., 2004), ecological momentary assessment (EMA; for review see Haedt-Matt & Keel, 2011), and mood induction (for overview see Leehr et al., 2015) studies have suggested the applicability of an emotion regulation model in BED. This argument is strengthened by findings from studies examining difficulties in emotion regulation (Danner et al., 2014; Harrison et al., 2016; Svaldi et al., 2012; Whiteside et al., 2007) and dialectical behaviour therapy (DBT) for BED (Masson et al., 2013; Safer et al., 2010; Telch, 1997;

Telch et al., 2000; Telch et al., 2001) which have concluded that individuals with BED report greater difficulties in emotion regulation and respond relatively well to treatment programs with a focus on emotion regulation. Given these findings, it was hypothesized that individuals with BED in the current study would report greater difficulties in emotion regulation compared to NW and OW controls.

Consistent with previous research (Brockmeyer et al., 2014; Eichen, Chen, Boutelle, & McCloskey, 2017), individuals with BED reported significantly higher DERS total scores, indicating greater difficulties in emotion regulation, compared to individuals with NED. Moreover, as in Brockmeyer et al. (2014), DERS total score was significantly elevated in the BED group relative to both the NW and OW control groups, suggesting that the difference in difficulties in emotion regulation was not the result of differences in BMI. After including BMI, age, and depression as covariates the difference in total DERS score persisted, providing further evidence of the robustness of this finding. Thus, as predicted by the emotion regulation model, individuals with BED reported greater difficulties in emotion regulation compared to NW and OW individuals with NED that could not be attributed to differences in BMI, age, or depression.

Individuals with BED also reported greater non-acceptance of emotions, difficulties inhibiting impulsive behaviour and maintaining a goal when experiencing emotions, and difficulties accessing emotion regulation strategies. Although, there were significant differences between groups in emotional awareness before controlling for BMI, age, and depression, these were no longer significant after entering the covariates ($p$ = .057). Interestingly, when overvaluation of weight and shape was added to the model, differences in emotional awareness reemerged ($p$ = .037), whereas the ability to inhibit

impulsive behaviour was no longer significantly different across groups ($p$ = .077). In contrast, previous studies have found that individuals with BED reported elevated scores on all six DERS subscales (Brockmeyer et al., 2014; Svaldi et al., 2012). These analyses however did not control for BMI, age, depression, and overvaluation. While sample size in the current study was comparable to previous analyses (Brockmeyer et al., 2014; Svaldi et al., 2012), it may have been insufficient to detect differences on these subscales after controlling for covariates. This hypothesis is supported by moderate to large effect sizes (see Table 10) and near significant p-values for the Aware and Impulse subscales. With greater power, differences on these subscales likely would have been detected, suggesting that BED may in fact be associated with greater difficulties inhibiting impulsive responses to emotions and being aware of current emotions.

A logical extension of this argument is that, since individuals with BED exhibited greater non-acceptance of emotions, difficulties maintaining goal-directed behaviour, and decreased access to emotion regulation strategies at this level of power despite the presence of covariates, these areas may be more integral to BED. Consistent with this postulation, previous research has provided evidence for the importance of non-acceptance of emotions, goal-directed behaviour, and access to emotion regulation strategies in predicting binge eating and emotional eating (Gianini et al., 2014; Whiteside et al., 2007). In a non-clinical sample, access to strategies was a significant predictor of binge frequency (Whiteside et al., 2007), and in a clinical sample this was related to emotional overeating (Gianini et al., 2014). In the same study, Gianini and colleagues (2014) found that the non-acceptance of emotions was a significant predictor of eating disorder pathology, while difficulties in goal-directed behaviour was associated with both

eating disorder pathology and emotional overeating. The current findings present interesting parallels suggesting the importance of the non-acceptance of emotions, goal-directed behaviour, and access to emotion regulation strategies in BED, and provide compelling evidence that individuals with BED experience increased difficulties accepting difficult emotions, as well as maintaining a goal and accessing emotion regulation strategies when experiencing difficult emotions.

In contrast to previous findings (Brockmeyer et al., 2014; Svaldi et al., 2012), there were no significant differences between BED and NED groups in emotional clarity before or after controlling for covariates in the current study. Since there were no differences in clarity before controlling for BMI, age, depression, and overvaluation of weight and shape, the discrepancy between this and previous findings (Brockmeyer et al., 2014; Svaldi et al., 2012) cannot be attributed to the inclusion of covariates. Consistently, individuals with BED in the current study reported lower mean Clarity scores compared to previous samples (Brockmeyer et al., 2014; Svaldi et al., 2012; Whiteside et al., 2007), suggesting that the difference between this and previous studies is not likely the result of different analytic techniques. One possible explanation is that individuals with BED in the current study simply experienced fewer difficulties being clear about their emotions compared to previous samples. It is also worth noting however, that the Clarity subscale (like all the DERS subscales) measures an individual's *perception* of emotional clarity. For instance, one item on this subscale is, "I know exactly how I am feeling". Because we live in a world where emotions tend to be simplified (e.g., I am happy; I am sad), an individual may feel that they do understand what they are feeling. With increased understanding of emotions however, it can become apparent that emotions are more

nuanced than what was initially believed. For example, anger towards one's boss may also include feelings of sadness, hurt, and shame. Thus, while the individual may report feeling clear about an emotion (anger) prior to receiving psychoeducation, increased awareness and understanding about the nature of emotions may cause a shift in their perception of emotional clarity.

An interesting question that emerges from this explanation is whether there would be changes in Clarity scores as individuals progress through the DBT program. A key aspect of DBT is dialectical thinking: the understanding that one can hold two contradictory beliefs or feelings at once (Safer et al., in press). Dialectical thinking is also paramount to moving from level 3 (single emotions) to level 4 (blends of emotions) in Lane and Schwartz's (1978) cognitive developmental model of emotions. In order to understand and communicate blends of emotions, one must understand that two seemingly contradictory emotions can exist simultaneously. For example, at a funeral, it is possible to be sad for the loss of a friend and also to be happy for the time that was shared together. This concept can be difficult to grasp, especially when individuals have spent their whole lives living in a world of "either/or" emotions. It is reasonable then to hypothesize that after being introduced to the idea that emotions are not unidimensional, individuals may experience a period of increased uncertainty about their emotions. We would thus expect Clarity scores to produce an inverted U-shape in the DBT condition throughout the trial, with individuals reporting greater clarity at baseline (when they are experiencing single emotions) and at the end of the trial (after having mastered some emotion regulation skills such as dialectical thinking). At mid-treatment however, we would predict an increase in difficulties being clear about one's emotions as individuals

adjust to the understanding that emotions are not "either/or". In this way it may be possible to test this *very* tentative explanation for the discrepancy in clarity scores in the current study. Given the discrepancy between this and previous studies (Brockmeyer et al., 2014; Svaldi et al., 2012) further research is necessary to better understand the role of emotional clarity in BED.

**4.2 Individuals with BED Reported Greater Negative and Positive Urgency**

In addition to increased difficulties in emotion regulation, individuals with BED also reported greater negative and positive urgency compared to individuals with NED after controlling for age, BMI, depression, and overvaluation of shape and weight, suggesting that individuals with BED are likely to experience difficulties inhibiting impulsive behaviour (e.g., binge eating) when experiencing negative and positive emotions. Since emotion regulation encompasses emotion-related facets of impulsivity (i.e., urgency; Gratz & Roemer, 2004) but not other aspects that are unrelated to emotion, it was hypothesized that of the UPPS-P subscales only negative and positive urgency would be elevated in BED, as has been demonstrated in previous research (Manasse et al., 2016). Consistent with this hypothesis, individuals with BED did not differ from individuals with NED on measures of premeditation, perseverance, and sensation seeking after controlling for BMI, age, and depression. Moreover, these results did not change after adding overvaluation of weight and shape to the model. It is not surprising that individuals with BED reported elevated negative urgency scores compared to both NW and OW controls, given previous research which has consistently reported an association between negative urgency and binge eating (Anestis et al., 2007; Emery et al., 2013; Farstad et al., 2015; Kelly et al., 2014; Manasse et al., 2016; Manjrekar et al., 2015;

Racine et al., 2015; Racine & Martin, 2016). On the other hand, research examining positive urgency in binge eating has been sparse and the current study is the first to demonstrate that individuals with BED do in fact experience greater positive (as well as negative) urgency.

To my knowledge, only one previous study examined positive urgency in eating disorders and this study found that negative, but not positive, urgency was associated with binge eating (Cyders et al., 2007). In that study, the authors compared individuals with alcoholism to individuals diagnosed with an eating disorder according to the EDE, and individuals with no diagnosed eating or substance use disorder. The eating disorder group included 28 women diagnosed with Eating Disorder Not Otherwise Specified (EDNOS), two women with each BN and BED, and four individuals who reported restricting or subthreshold AN. Thus, the sample was heterogeneous with the majority of individuals meeting the diagnostic criteria for EDNOS. Individuals with EDNOS present with eating disorder symptoms (e.g., extreme restriction, purging) but do not meet the full diagnostic criteria for a specific feeding or eating disorder described in the DSM. Although at the time of this study BED was categorized as a subtype of EDNOS, Rockert, Kaplan, and Olmsted (2007) reported that individuals diagnosed with EDNOS in a tertiary care center were least likely to present with symptoms of BED. More common presentations included subthreshold BN or extreme restriction with a BMI above 18.5 (Rockert, Kaplan, & Olmstead, 2007). Therefore it is unlikely that all individuals in Cyder and colleagues' (2007) eating disorder group reported binge eating; in fact four individuals reported restricting or subthreshold AN which would be characterized by subjective rather than objective binge eating episodes if such episodes were present (for discussion of subjective

and objective binge episodes see section 4.3). Therefore the conclusions of Cyders and colleagues (2007) about the relationship between binge eating and positive urgency are questionable.

When looking at a homogeneous sample of individuals diagnosed with BED according to the EDE interview in the current study, we found that positive urgency does in fact differentiate between individuals with BED and those with NED. Namely, individuals in the BED group reported greater positive urgency than those in NW and OW groups even after controlling for covariates such as BMI, age, depression, and overvaluation of weight and shape. This suggests that people with BED may binge eat in the face of positive, as well as negative, emotions and is consistent with clinician reported experiences (J. Carter, 2016, personal communication). The discrepancy between the current results and those of Cyders and colleagues (2007) suggests that positive urgency may be differentially associated with different eating disorder (sub)types. This assertion would not be unreasonable given that certain theories of eating disorders are more applicable to one diagnosis over another (see Dietary Restrain section for full discussion) and that certain eating disorder subtypes (i.e., BN and BED) are better characterized by impulsive behaviour (Rosval et al., 2007).

Overall, the findings from the first set of analyses comparing individuals with and without BED support an emotion regulation model of BED. With the exception of the emotional clarity individuals with BED reported greater difficulties in emotion regulation compared with the NED group. Including BMI as a covariate confirmed that these differences in difficulties in emotion regulation were not the result of differences in BMI across the three groups. Additionally, individuals with BED reported significantly higher

negative and positive urgency compared to individuals in the NW and OW control groups indicating that they are more likely to act impulsively in response to negative and positive emotions. Such differences in impulsivity were not observed on the other (non-emotion related) subscales of the UPPS-P (i.e., premeditation, perseverance, and sensation seeking), suggesting that BED is characterized by deficits in impulsivity specifically related to an inability to tolerate intense emotions. Having confirmed the first hypothesis, we next sought to examine the relationship between DERS total score and eating disorder pathology, to better understand the role of emotion regulation in the etiology of BED.

**4.3 Difficulties in Emotion Regulation do not Predict Binge Frequency**

The emotion regulation model of BED posits that individuals with poorer emotion regulation skills will be more likely to turn to binge eating as a means of controlling intense emotions (Wiser & Telch, 1999). It was therefore hypothesized that difficulties in emotion regulation would be positively related to binge frequency. Although this hypothesis was supported before controlling for covariates, the relationship between DERS total score and binge frequency was no longer significant after BMI, age, and depression had been added to the model. This is in contrast to previous research which found that DERS total score explained unique variance in binge frequency (Whiteside et al., 2007). Two differences between the current study and Whiteside and colleagues' (2007) could account for this discrepancy. First, Whiteside and colleagues (2007) examined a non-clinical sample recruited from a university setting, whereas the current study examined a community sample of individuals meeting the diagnostic criteria for BED. It is possible that the relationship between emotion regulation and binge frequency is different between clinical and non-clinical populations as a result of the range

restriction problem. The range restriction problem suggests that the relationship between two variables may be masked when only a part of the data set is present. In this case, data was only analyzed for individuals with BED, including the upper (but not the lower) end of the binge frequency distribution. It is possible that if we had included binge frequency for individuals without BED as well – thereby capturing the full distribution – a relationship between difficulties in emotion regulation and binge frequency would have emerged.

Alternatively, the discrepancy could be related to differences in how binge frequency was measured. Before describing the two measures it is important to differentiate between objective binge episodes (OBEs) and subjective binge episodes (SBEs). According to the EDE, overeating episodes can fall into one of four categories based on the presence or absence of (1) loss of control and (2) the consumption of an objectively large amount of food (Fairburn & Cooper, 1993). If both criteria are present, the episode is considered an OBE. If the amount of food was not objectively large but the individual perceived it to be excessive and loss of control was present then the episode is classified as a SBE. Any other iteration (i.e., reporting overeating without loss of control or not reporting either criterion) is not considered an episode of binge eating (Fairburn & Cooper, 1993). In the current study only data regarding the frequency of OBEs was obtained since the presence of OBEs is a diagnostic criterion for BED (APA, 2013). In contrast, Whiteside and colleagues (2007) used a two item self-report measure of binge frequency that did not require an example of a typical binge episode; it is therefore likely that individuals reported both OBEs and SBEs.

Research suggests that individuals with BN who report SBEs do not differ significantly from those who report OBEs in terms of eating disorder psychopathology (Brownstone et al., 2013; Fitzsimmons-Craft et al., 2014). In fact, one study found that individuals reporting SBEs actually experienced greater depression than those reporting OBEs, leading the authors to suggest that it is the loss of control, rather than the amount of food consumed, that characterizes binge eating (Fitzsimmons-Craft et al., 2014). Similarly, Brownstone and colleagues (2013) argued that the presence of SBEs may indicate broader psychopathology in the context of BN. These findings have been replicated in a sample of individuals with BED (Palavras, Morgan, Borges, Claudino, & Hay, 2013), suggesting that there are no clinically significant differences between individuals who report SBEs and those who report OBEs. Consequently, the measure employed by Whiteside and colleagues (2007) may have captured a broader range of binge eating behaviours characterized by loss of control rather than the amount of food consumed. In the context of the current study, these findings suggest that difficulties in emotion regulation may be associated with loss of control eating but not necessarily the consumption of an objectively large amount of food. Consistent with this speculation, we found that DERS total score predicted eating disorder psychopathology (as measured by the EDE-Q; see section 4.6), but not OBE frequency, in the current sample.

**4.4 Depression Severity Predicts Binge Frequency**

Although DERS total score was not a significant predictor of OBEs, BSI depression score explained unique variance in binge frequency, even after controlling for overvaluation of weight and shape, suggesting that increased depression is associated with binge episodes that are objectively large. While not anticipated, this finding is

consistent with previous research examining negative affect, depression, and BED. As discussed earlier, negative affect models (in particular escape theory) have emerged as prominent theories of binge eating. Studies have demonstrated that individuals who binge eat are more likely to endorse emotional factors as triggers of binge eating (Vanderlinden et al., 2001), to attribute binge eating episodes to mood (Stein et al., 2007), and to eat in response to negative emotions (Elderidge & Agras, 1996) compared to their non-binge eating counterparts. Given the positive relationship between binge eating and emotional eating (Masheb & Grilo, 2006; Ricca et al., 2009), it has been suggested that emotions such as depression/sadness may precipitate binge eating. This hypothesis has been corroborated by evidence that individuals who binge eat experience increased negative affect prior and subsequent to binge eating episodes (Haedt-Matt & Keel, 2011; Hilbert & Tuschen-Caffier, 2007; Stein et al., 2007; Wegner et al., 2002). Moreover, individuals with BED report higher depression scores compared to their non-BED counterparts (Borges, Jorge, Morgan, de Silveira, & Custodio, 2002; Fontenelle et al., 2003; Smith, Marcus, Lewis, Fitzgibbon, & Schreiner, 1998; Telch & Stice, 1998).

The current finding that, of the variables included in the model, depression was the only significant predictor of binge frequency demonstrates that increased depression predicted increased binge frequency in a community sample of individuals with BED. This is consistent with previous studies that have demonstrated an association between depression and binge eating symptoms (Adamus-Leach et al., 2013; Mazzeo, Saunders, & Mitchell, 2006; Paxton & Diggins, 1997; Wheeler, Greiner, & Boulton, 2005). In an examination of the escape model of BED (Heatherton & Beaumeister, 1991), Paxton and Diggins (1997) found that depression was the sole predictor of binge eating, accounting

for the association between coping and binge eating. Similarly, depression was a significant predictor of binge eating symptoms in men with BED, although self-esteem also explained unique variance in women (Mazzeo et al., 2006). In conjunction with the current report, these findings suggest that increased depression is associated with greater binge frequency – in particular OBE frequency – an argument which is strengthened by the fact that the current and previous studies have used different measures of binge eating and depression. Despite (unavoidable) mild variations in self-report measures, the relation between depression and binge eating persists. Thus, while the relationship between difficulties in emotion regulation and binge frequency appears to be sensitive to differences in measurement tools, the relationship between depression and binge frequency is robust, highlighting an important role for depression in BED.

**4.5 Depression Moderates the Association Between DERS and Binge Frequency**

While the current study presents compelling evidence for a positive relationship between depression and binge eating disorder symptoms, the association between these variables does not occur in isolation and is likely to interact with related variables, such as emotion regulation. The current study is the first to document that the relationship between emotion regulation and binge frequency is moderated by depression. Namely, there is a significant relationship between difficulties in emotion regulation and binge frequency, with greater difficulties in emotion regulation predicting more binge episodes, in individuals who report high (+1SD) but not low (-1SD) depression scores on the BSI. One explanation for this finding is that individuals who experience greater depression are more likely to require emotion regulation skills, exacerbating the difference between those who do and do not have difficulties in emotion regulation. For example, consider

two individuals: Anne reports difficulties in emotion regulation while Barb does not. If Anne and Barb do not experience intense emotions such as depression then we would not expect to see a difference in binge frequency according to the emotion regulation model because neither Anne nor Barb needs to use these skills. If however, Anne and Barb experience high levels of depression, then we would expect Anne to report more binge eating episodes given that she has more difficulty accessing and implementing emotion regulation skills. Thus although not anticipated, the findings from the moderation analysis support the emotion regulation model, suggesting that individuals who experience more distress (i.e., depression) are more affected by differences in emotion regulation skills (see Figure 8). This could also, partially, explain why the relationship between difficulties in emotion regulation and binge frequency did not emerge. If the relationship between emotion regulation skills and binge eating is dependent on the presence of emotionality, this association would not appear in the whole sample where we would expect to see variance in experiences of emotionality.

In general, previous studies have reported similar results, suggesting associations between emotionality, negative affect, and emotion regulation in eating disorders. For instance, neuroticism (the tendency to have a strong negative reaction to stress) was found to moderate the effect of negative affect lability (the tendency for emotions to change quickly in terms of strength and valence) on week-to-week binge variance in individuals with BED and BN (Zander & de Young, 2014). Increased negative affect lability was only associated with variance in binge frequency in individuals who also reported increased neuroticism scores. Interestingly, this analysis appears to be opposite from, but complementary to, the current study. While we examined emotion regulation, Zander and

de Young (2014) examined neuroticism, which can also be conceptualized as negative emotional reactivity, a related but distinct construct. Davidson (1998) describes emotional reactivity as how readily one experiences an emotion, with what intensity, and for what duration. In the context of BED, emotional reactivity would be the vulnerability to experiencing intense emotions, whereas emotion regulation reflects the ability to modulate those emotions. Further, we looked at depression as a pervasive state of negative affect while Zander and de Young (2014) examined negative affect lability, or *changing* negative affect. The results from the two studies suggest that both emotion regulation and reactivity are related to binge frequency but interact with pervasive and changing negative mood states, respectively highlighting the complexity of the relationship between negative affect and binge eating.

Other moderation analyses in BED have focused primarily on negative urgency (Anestis et al., 2007; Emery et al., 2013; Manjrekar et al., 2015). These, like the current study, suggest an interaction between emotion regulation (i.e., negative urgency) and affect (e.g., positive emotions; Emery et al., 2013), as well as interactions between different aspects of emotion regulation (Anestis et al., 2007; Manjrekar et al., 2015). For instance, Manjrekar et al. (2015) reported that the relationship between negative urgency and binge frequency was moderated by emotional awareness (clarity and awareness). Evidently, the relationship between emotion regulation, affect, and binge eating is complex and multifaceted. Future research should consider that emotion regulation is not a single construct and that different aspects of emotion regulation may be differentially related to depression/negative affect.

**4.6 Difficulties in Emotion Regulation Predict Eating Disorder Psychopathology**

Despite the association between binge frequency and depression, the latter did not predict eating disorder psychopathology. Rather, only difficulties in emotion regulation explained unique variance in EDE-Q Global score after controlling for BMI, age, depression, and overvaluation of weight and shape. These findings are consistent with previous studies examining emotion regulation and eating disorder psychopathology in a range of eating disorders (Haynos, Roberto, & Attia, 2015; Woltz et al., 2015). In a sample of individuals with unspecified eating disorders, emotional awareness, emotional clarity, and access to emotion regulation strategies were significantly associated with EDE-Q Global score, although multiple regression revealed that only the access to strategies contributed unique variance to EDE-Q Global score (Pietsky, Haynos, Lavendar, Crow, & Peterson, 2017). Moreover, changes in difficulties in emotion regulation predicted changes in EDE-Q total score in a sample of inpatients with AN above and beyond weight gain (Rowsell, MacDonald, & Carter, 2016). Rowsell and colleagues (2016) also reported that depression was not associated with changes in EDE-Q total score from admission to discharge, paralleling the current finding that DERS total score but not depression was associated with EDE-Q Global score. This report (Rowsell et al., 2016) is important because it suggests possible clinical implications for the relationship between DERS total score and EDE-Q Global score presented here. Namely, it is possible that changes in difficulties in emotion regulation would also be associated with changes in EDE-Q Global scores following treatment for BED as in AN (Rowsell et al., 2016), highlighting the importance of emotion regulation training in treatment for eating disorders.

In contrast, DERS total score did not predict binge frequency after controlling for BMI, age, and depression. Instead, as discussed above we found that BSI depression score was the only significant predictor of binge frequency and that depression moderated the association between difficulties in emotion regulation and binge frequency. While related, binge frequency and EDE-Q Global score clearly capture different aspects of the psychopathology of BED, indicating that the role of emotion regulation likely differs across symptoms. This was evidenced by differential relations between difficulties in emotion regulation and OBEs compared to loss of control overeating (for full discussion see the section 4.3). Nevertheless, the current findings do provide evidence for an emotion regulation model of BED, highlighting the complexities of the relationship between emotion regulation strategies and binge eating.

### 4.7 The Severity of Insomnia Symptoms Does Not Moderate the Association Between DERS and Binge Frequency

Having provided compelling evidence for the emotion regulation model of BED, the final goal of this study was to examine the role of associated factors, namely sleep difficulties. Recently sleep has been shown to play a role in emotion regulation (Gruber & Cassoff, 2014). One theory suggests that the connectivity between emotion centers in the brain (i.e., the amygdala and prefrontal cortex [PFC]) is dependent on sleep. The connectivity between these two areas is higher when rested, allowing for more efficient executive functioning and inhibition of impulses, and lower when sleep deprived, resulting in poor executive function capacity and impulsivity (Gruber & Cassoff, 2014). Importantly, the association between emotion regulation and sleep disturbance suggests that sleep may also play a role in clinical disorders (such as BED) that are characterized

by emotion regulation difficulties. Studies have confirmed this hypothesis demonstrating a moderating effect of sleep on emotion regulation in BPD symptoms (Selby, 2013) and remission from bipolar disorder (Boudebesse & Henry, 2012). We therefore sought to examine whether insomnia symptoms would have a moderating effect on the relationship between emotion regulation and eating disorder pathology in BED, similar to BPD (Selby, 2013).

In contrast to previous studies (Boudebesse & Henry, 2012; Selby, 2013), the severity of insomnia symptoms did not moderate the association between DERS total score and binge frequency or EDE-Q Global score. Since the proposed relationship between sleep and emotion regulation is biological (i.e., connectivity between brain areas; Gruber & Cassoff, 2014), it is possible that a significant relationship between ISI and DERS did not emerge in the current study because the ISI is a measure of perceived sleep difficulty rather than actual sleep duration. This explanation would be consistent with reports demonstrating that decreased sleep duration is associated with increased emotional reactivity in children (Reidy, Hamann, Inman, Johnson, & Brennan, 2016) and that sleep extension, facilitating recovery from sleep debt, may improve emotion regulation (Motumura et al., 2014), as these studies argue that sleep duration (rather than perceived sleep difficulties) contribute to emotion regulation deficits. Other studies however have reported that scores on the ISI are correlated with polysomnography data (Sadeghniiat-Haghighi, Yazdi, & Firoozeh, 2014), indicating that while the ISI does not provide a numerical value for sleep duration, ISI total score converges with biological measures. Moreover, two recent studies have reported an association between perceived sleep quality and insomnia with emotion regulation in veterans with post traumatic stress

disorder and individuals with anxiety (Kriwan, Pickett, & Jarrett, 2017; Short et al., 2016). Neither of the aforementioned studies used a biological measure of sleep duration and still reported an association between sleep and emotion regulation skills, including self-reported distress and frustration on a task. In fact, Kirwan and colleagues (2017) reported that emotion regulation moderated the relationship between insomnia and anxiety similar to what was reported in BPD (Selby, 2013). Namely, insomnia increased anxiety only in individuals who reported poor emotion regulation skills. Thus, the use of perceived insomnia symptoms as a measure of sleep disturbance likely does not account for the lack of moderation in the current analysis.

Alternatively, the difference in results between the current study and those examining BPD (Selby, 2013), bipolar disorder (Boudebesse & Henry, 2012), and anxiety (Kirwan et al., 2017) could be attributed to the absence of data on SBEs in the current study (as was discussed in section 4.3). Previous research suggests that there are minimal differences between individuals who exhibit SBEs and those who exhibit OBEs (Brownstone et al., 2013; Fitzsimmons-Craft et al., 2014; Palavras et al., 2013), and has argued that binge eating may be better characterized by loss of control rather than the amount of food consumed (Fitzsimmons-Craft et al., 2014). It is possible that insomnia severity and emotion regulation are related to loss of control episodes of eating and not limited to objectively large episodes. If this is correct, we would expect to find an effect when both OBE and SBEs are considered but not just for OBEs (as was assessed in the current study). Having said this, unlike in the DERS analysis, the interaction between ISI total score and DERS total score did not account for additional variance in EDE-Q Global score suggesting that the effect of difficulties in emotion regulation on eating disorder

psychopathology is not dependent on insomnia symptoms. Clearly, additional research is necessary to examine the relationship between insomnia, emotion regulation, and eating disorder pathology in BED.

It must also be noted that the current analysis neglected the role of depression in insomnia and emotion regulation. As previously discussed, the current study found that BSI depression score moderated the relationship between difficulties in emotion regulation and binge frequency. Research has also indicated an association between depression and insomnia (Herrick & Sateia, 2016). In fact, sleep disturbance is a diagnostic criterion for major depressive disorder (APA, 2013), and both long (≥ 10 hours/night) and short (≤6 hours/night) sleep duration are predictors of subsequent depressive episodes (Perlman, Johnson, & Mellman, 2006). Since this analysis was the first, to our knowledge, to assess moderators of the relationship between emotion regulation and eating disorder pathology, the relationships between emotion regulation and (1) insomnia and (2) depression were considered independently for ease of interpretation. Considering the overlap between depression, insomnia, and emotion regulation (Borges et al., 2002; Gruber & Cassoff, 2014; Fontenelle et al., 2003; Herrick & Sateia, 2016; Smith et al., 1998; Telch & Stice, 1998) however, future studies should consider the interaction between all three variables.

Ultimately results from the current study do not point towards a role for insomnia symptoms as a moderator of emotion regulation difficulties in BED. Having said this, the current study was the first to evaluate such a relationship. These findings should be replicated before any conclusions can be drawn. Moreover, even if insomnia symptoms are not related to emotion regulation, this does not preclude their importance in BED.

Unpublished data from our lab has found that individuals with BED report greater insomnia severity scores and are more likely to report clinically significant insomnia compared to individuals with NED (Van Wijk, 2017). These results persist even after controlling for BMI and age, but are no longer significant once depression has been added to the model. Further, ISI total score is a significant predictor of binge frequency until depression has been added to the model. Taken together, these findings suggest a possible mediating role for insomnia symptoms between depression and binge frequency, and future studies should test this hypothesis. Sleep disturbance has also been cited as a risk factor for relapse in addition to psychoactive substances (Brower & Perron, 2010). Given the overlap between BED and addiction (Davis & Carter, 2014), including the role of difficulties in emotion regulation, sleep disturbance may also be a risk factor for relapse in BED, though this hypothesis remains to be tested. Despite the tentativeness of these hypotheses, these arguments suggest two possible roles for insomnia in BED that are not directly related to difficulties in emotion regulation. Thus, while the current study does not suggest a role for insomnia symptoms in the emotion regulation model of BED, the role of sleep and sleep disturbance in this disorder should not be overlooked.

### 4.8 Clinical Implications

The current findings support an emotion regulation model of BED and have some potentially important clinical implications. First, the findings highlight the importance of addressing emotion regulation difficulties in treating BED. Since individuals with BED report greater difficulties in emotion regulation that are related to the severity of eating disorder psychopathology, this suggests that increasing emotion regulation capabilities should help reduce eating disorder psychopathology. In support of this argument, Rowsell

and colleagues (2016) demonstrated that change in DERS score predicted change in eating disorder psychopathology in a sample of inpatients with AN above and beyond the contribution of weight gain. This finding suggests that facilitating emotion regulation strategies in eating disorder samples may be effective in reducing psychopathology.

One type of therapy that has shown promise in facilitating emotion regulation strategies is dialectical behaviour therapy (DBT). DBT, currently the gold standard for BPD treatment (Linehan, 1993), focuses on developing efficacy in four areas: (1) mindfulness; (2) emotion regulation; (3) distress tolerance; (4) interpersonal relations (Linehan, 1993; Wiser & Telch, 1999). Findings from the current study suggest that DBT may be effective for treating BED as well. Preliminary reports support this assertion (Masson et al., 2013; Safer et al., 2010; Telch, 1997; Telch et al., 2000; Telch et al., 2001) demonstrating that DBT is relatively effective in reducing binge frequency. It is not yet clear whether changes in emotion regulation skills mediate this relationship (Masson et al., 2013) although this could be the consequence of using binge frequency as the outcome variable. In the current study, DERS total score was associated with EDE-Q Global score and not binge frequency; however, the association between DERS and binge frequency was moderated by depression level. Although previous studies have not found change in DERS score to be predictive of change in binge frequency (e.g., Masson et al., 2013), these findings suggest that including other variables (i.e., depression) and looking at other measures of eating disorder pathology (e.g., EDE-Q Global score) may enhance our understanding of the role of emotion regulation in DBT treatment for BED.

The current findings also suggest that slight alterations in DBT administration could increase its efficacy in BED. It was found that BED was associated with negative

and positive urgency. Currently increasing distress tolerance is the most effective treatment for reducing negative urgency (Cyders & Smith, 2008). Putting a greater focus on distress tolerance at the beginning of DBT treatment could allow individuals to gain control of impulsive behaviours early in treatment. Additionally, serotonin and dopamine have been linked to negative urgency (for review see Cyders & Smith, 2008), suggesting that selective serotonin reuptake inhibitors (SSRIs), the first line of treatment for BPD (Grossman, 2002; Soloff, 2000), could be used to facilitate the efficacy of distress tolerance training. Combining these two approaches (i.e., distress tolerance and SSRIs) could thus be a good first step in treating BED by reducing negative urgency behaviours. This is particularly important as Manasse and colleagues (2016) have reported that negative urgency, as opposed to non-emotion related facets of impulsivity, predicted treatment outcome in BED. In contrast, there is currently no treatment that focuses on reducing positive urgency (Cyders & Smith, 2008), likely because positive emotions are typically considered to be "good". That individuals with BED report greater positive (as well as negative) urgency indicates that clinicians and health professionals need to be mindful of how clients react to positive emotions. Distress tolerance training could easily be modified to focus on positive emotions if clinicians are aware that this is an issue for the client.

Finally, understanding the moderating effect of depression on DERS total score and binge frequency could also increase the efficacy of DBT for BED. Namely, clinicians could screen for depression to determine whether DBT would be the most effective treatment, or whether another therapeutic approach may be best suited to the individual. Since emotion regulation was associated with eating disorder pathology across the entire

sample but was only associated with binge frequency in individuals with higher depression, it follows that DBT may be most effective for individuals reporting greater depression scores. Specifically, these individuals are likely to see a reduction in both eating disorder psychopathology *and* binge frequency following DBT treatment, ultimately translating to better quality of life. While still hypothetical at this point, this argument highlights an important point: in order to provide the most effective treatment we need to understand which individuals will be most likely to benefit from any given treatment. For instance research has suggested that individuals with higher pre-treatment depression and weight concern, and lower general health and vitality are less likely to complete guided self-help treatment for BN (Jones et al., 2012). Similarly, depression and body dissatisfaction are negative predictors of clinically significant change in eating disorder pathology in adolescents who are hospitalized for AN (Schlegl et al., 2016). Our findings are consistent with this research, indicating that clinicians should consider pre-treatment depression in both treatment planning and implementation.

## 4.9 Strengths and Limitations

**4.9.1 Strengths.** The current study had a number of strengths. All of the hypotheses and analyses presented here were grounded in theory and empirical research. This study contributes to a growing body of literature in support of the emotion regulation model of BED, presenting convergent analyses to examine the model from different perspectives. First the hypothesis that individuals with BED would exhibit greater difficulties in emotion regulation was tested using one-way ANOVAs. After confirming that individuals with BED did report greater difficulties in emotion regulation, a second set of analyses were conducted regressing binge frequency and eating disorder

psychopathology on DERS total score. Doing so allowed us to characterize the nature of the relationship between difficulties in emotion regulation and eating disorder pathology. The results from this analysis aligned with the first providing convergent evidence for an emotion regulation model. Including both the DERS and UPPS-P also strengthened the findings by providing convergent results on two different, but related, measures of emotion regulation. In this way, the current study provides one of the most comprehensive (and first) analyses of the emotion regulation model in BED. Furthermore, BMI, age, depression, and overvaluation of weight and shape were included as covariates for all analyses increasing internal validity, allowing us to conclude with confidence that the results reported here cannot be better accounted for by differences on any of these variables. It is also worth noting that all of the measures used in the current study were well-validated and considered to be the gold standard, enhancing confidence in the findings and allowing them to be easily compared with previous studies.

In addition to methodological strengths, this study exhibits features that may be advantageous to the implementation of treatment programs within the province. Although the NL sample will be discussed later as a possible limitation, the use of such a homogeneous sample could also be considered a strength. Currently, there are no publicly funded services for BED in NL, despite our lab's estimate that 7,000 to 14,000 individuals in the province suffer from the disorder. In order to establish reliable and cost-effective treatment options for these individuals, it is essential that health professionals and policy-makers understand how BED presents in *this* province. It would be unwise and costly to implement programs that are not relevant to individuals suffering from BED here. While the use of a NL sample does limit the generalizability of the findings to

provinces in other parts of Canada, it will likely be instrumental in establishing treatment centers and policies across NL. That our ethnic breakdown is similar to that in NL provides further support for the utility of this project as the basis for establishing treatment programs within the province. Moreover, we recruited participants from the community, rather than clinical practices, further suggesting the generalizability of our findings to the NL population.

**4.9.2 Limitations.** As with all studies, this study had some potential methodological limitations. First, the generalizability of the current findings should be interpreted with caution as the sample was a NL sample, and primarily female and Caucasian. Moreover, less than 50% of individuals eligible for a telephone interview accepted the invitation suggesting a possible selection bias. In terms of internal validity, we noted that the three groups were not matched on BMI, age, and depression suggesting that factors other than BED could have accounted for the differences reported here; however, these variables were controlled in the analyses. This study was also a cross-sectional analysis with a correlational design indicating that we cannot determine directionality. Finally, we acknowledge the limitations of using self-report measures and the lack of data regarding SBEs

***4.9.2.i Generalizability.*** Since the sample was recruited from NL, it is possible that the findings from this study may not be generalizable to other provinces within Canada or countries outside of Canada. As mentioned previously, NL exhibits a culture that is distinct from the rest of the country. Consequently, one must be cautious when making inferences about the generalizability of this sample, as it is possible that cultural differences may influence the presentation and characteristics of BED. The

generalizability of these findings is also limited in that our sample was primarily white and female. Hudson and colleagues (2007) reported that the ratio of men to women with BED is 2.5:3.0. The ratio in the current study was 1:14.5 (93.5% female), a twelve-fold increase from Hudson et al.'s (2007) estimate, suggesting that the current findings may not reflect the experience of men living with BED. The sample was also primarily Caucasian (96.8%); however this estimate is similar to the percentage of individuals in NL who reported White/Caucasian ethnicity in the 2006 census (94.2%; Statistics Canada, 2006).

There was also a possible selection bias in the current study. Namely, only 60% of individuals who were eligible to take part in the NED study completed the questionnaires, and less than half of individuals who were eligible for a telephone interview in the BED group accepted (46.5%). It is possible that completers and non-completers differed in some meaningful way limiting the generalizability of the sample. For example, those who completed the NED questionnaires were willing to sit down and complete a 1-hour questionnaire indicating motivation and/or willingness to help with research compared to non-completers. This is consistent with analyses comparing completers and non-completers. While there were no differences in terms of BMI, alcohol consumption, drug use, or number of positive responses on the SCOFF, a Chi Squared test of independence revealed a significant difference in level of education across completers and non-completers. Namely, there was a trend towards completers being more likely to have completed graduate work, $p = .058$, suggesting that these individuals may be more familiar with the research process and therefore likely to participate in research. In the BED group, not completing a telephone interview could suggest unwillingness to discuss

binge eating with a stranger over the phone. Furthermore, individuals in the BED group were recruited for a treatment study. Thus, completing the interview could be an indicator of willingness to change. When individuals who accepted and did not accept the interview were compared however, there were no significant differences in BMI, age category, level of education, binge frequency, number of binge characteristics endorsed, or compensatory behaviours suggesting that there were no clinically significant differences between the groups.

***4.9.2.ii Interpretability.*** One goal in the current study was to be able to extend on previous findings by including an OW control group. Unfortunately, even though the OW group reported a mean BMI in the obese category, the average BMI in the BED group was still significantly higher. Moreover, the three groups (BED, NW, OW) differed in terms of age and BSI depression scores, suggesting that differences in difficulties in emotion regulation or impulsivity could attributed to differences in factors other than the presence or absence of BED. To address this concern, we conducted analyses of covariance including age, BMI, and BSI depression score as covariates. Since the differences persisted even after controlling for these covariates we are confident that the results reported here cannot be better accounted for by differences in age, BMI, and/or depression. In addition, all analyses were rerun including overvaluation of weight and shape in the model given the argument for its importance in BED (Grilo, 2013), producing near identical results. While many steps were taken to maximize internal validity in the current study, it is not possible to include all possible covariates or to have groups matched on all variables. One factor that was not included in the present study, and should be considered in the future, is anxiety. Given the role of depression in the

current study, the overlap between anxiety and depression (Hirschfield, 2001), and anxiety and BED (Telch & Stice, 1998), future analyses will include BSI anxiety score as a covariate to ensure that differences on this variable cannot account for the findings reported here.

***4.9.2.iii Directionality.*** In addition to limitations affecting the generalizability and interpretability of the findings, this study was a cross-sectional analysis examining emotion regulation in BED at a single time point. Thus, it is not possible to determine whether emotion regulation and urgency deficits preceded binge eating or whether binge eating precipitated difficulties in emotion regulation. Longitudinal and prospective studies are necessary to address this question. Similarly, the analyses presented here are correlational suggesting that causation cannot be inferred. Although emotion regulation theory posits that difficulties in emotion regulation lead to binge eating, it is also possible that binge eating leads to emotion regulation deficits, or that the two factors interact reciprocally. Examining the trajectory of emotion regulation deficits and binge eating over the course of the larger DBT trial may help to clarify some of these uncertainties.

***4.9.2.iv Measures.*** Finally, we noted two limitations related to data collection. As data for this study was collected via self-report questionnaires, there is always the issue of recall bias or variability in interpretation of the questions. This limitation might have been exacerbated in the current study because the questionnaires were completed online. Although the individual could complete the questionnaires at their convenience increasing feasibility of the study, they were not able to ask the researcher questions to clarify meaning. To minimize these issues and to maximize interpretability, the current study administered questionnaires that are considered to be the gold standard and whose

reliability and validity have been established. For instance, the EDE-Q has been shown to correlate highly with the EDE interview (Berg et al., 2012) and ISI scores are associated with biological measures of sleep (Sadeghniiat-Haghighi et al., 2014) suggesting that these are reliable measures of eating disorder pathology and insomnia symptom severity respectively. Furthermore, individuals in the BED group completed the EDE interview allowing us to confirm BED diagnosis and also to obtain an accurate report of objective binge frequency.

Although having an interviewer determined measure of OBE frequency was a strength in the current study, the lack of data regarding SBEs was a limitation. As has been reiterated throughout this discussion, no significant differences have yet been identified between OBEs and SBEs to suggest that one is more indicative of eating disorder pathology (Brownstone et al., 2013; Fitzsimmons-Craft et al., 2014; Palavras et al., 2013), and researchers have argued that loss of control may be a better indicator of pathology in eating disorders characterized by binge eating (Fitzsimmons-Craft et al., 2014). In retrospect, including a measure of both OBE and SBE frequency would therefore have been useful in the current study. Having said this, the EDE-Q contains a self-report question about the frequency of binge eating, reminiscent of the measure employed by Whiteside et al. (2007) so it would be possible in the future to (1) compare self-report to interviewer gathered responses and (2) examine the relationship between DERS and self-reported binge frequency. Since we included the EDE-Q Global score as a measure of eating disorder psychopathology in the current study, it is unlikely that any new information would emerge from such analyses. Nevertheless, it is an important point to consider and will be addressed in future studies.

### 4.10 Future Directions

Having described the clinical implications, limitations, and strengths of this project, this final section addresses possible future research directions. Numerous future directions have emerged from this work some of which have been briefly discussed earlier in this section. For instance, examining the trajectory of DERS Clarity subscale scores across DBT treatment may provide valuable insight as to how individuals with BED perceive their emotions. Furthermore, additional research is necessary to better delineate the role of insomnia symptoms and sleep disturbance in BED as this could be influential in recovery maintenance. Here however I will focus in particular on three future directions emerging from this study. First, throughout this discussion it has become clear that future studies should include measures of both subjective *and* objective binge episodes. Failing to do so here limited the interpretability of the findings and led to a number of possible explanations that could not be directly tested. Next, the role of depression has emerged as an important predictor and moderator in the current study. It will be important for future studies to consider models of BED that conceptualize BED with negative affect as a more disturbed variant of the disorder (Grilo, 2004). Finally, the role of urgency in BED was only briefly examined in the current study. Future studies should focus on understanding how these factors could contribute to eating disorder pathology.

**4.10.1 Focus on SBEs.** While not a direct aim of this study, previous research examined during the writing of this discussion has suggested that the distinction between OBEs and SBEs should be a point of consideration in future studies on BED. As research suggests that there is no clinically or statistically significant difference in

psychopathology between individuals who report OBEs and SBEs (Brownstone et al., 2013; Fitzsimmons-Craft et al., 2014; Palavras et al., 2013), it is recommended that future studies include measures of both OBE and SBE frequency. Considering OBE and SBE binge frequency separately and together will provide a more comprehensive understanding of the nature of binge eating in BED, allowing researchers to differentiate between loss of control overeating and objectively large binge episodes. This line of research will aid not only in our theoretical understanding of BED, but will also inform future treatment studies to improve the efficacy of treatment for BED.

**4.10.2 Depression subtypes.** Similarly, considering the role of depression in BED may be paramount in determining effective treatments and predictors of treatment outcome for BED. In the current study, findings suggested that difficulties in emotion regulation were associated with binge frequency in individuals who reported higher but not lower depression scores. This finding is consistent with a conceptualization of BED that subtypes individuals based on pure dietary and dietary-negative affect dimensions (Stice et al., 2001). Individuals who fall in the dietary-negative affect dimension report increased eating and weight concerns, poorer social maladjustment, and tend to have a worse treatment response (Stice et al., 2001) with more binge eating episodes at the end of treatment (Masheb & Grilo, 2008), as well as greater negative urgency (Carrard, Crepin, Ceschi, Golay, & Van der Linden, 2012) compared to individuals in the dietary restraint cluster. Consequently, Grilo (2004) has suggested that the dietary-negative affect dimension represents a more disturbed variant of eating disorders. This two-cluster model has been replicated many times in individuals with eating disorders (Masheb & Grilo, 2008; Stice et al., 2001), including in child and adolescent samples (Goldschmidt et al.,

2008; Grilo, 2004), but did not emerge in an overweight/obese sample (Gagnon-Girouard, 2010), suggesting that it is specific to an eating disorder population. Future studies should consider this distinction when examining difficulties in emotion regulation, impulsivity, and the relationship between these variables and eating disorder pathology. If consistent with the current study, it is hypothesized that individuals reporting the negative affect subtype of BED should show a stronger relationship between DERS total score and binge frequency, as was indicated by the moderation analysis here. Importantly, this could possibly be indicative of differential treatment outcomes and point to different treatment plans for the two subtypes.

**4.10.3 Negative and positive urgency.** Finally, it will be important for future studies to elaborate on the role of negative and positive urgency in BED. This study was the first to our knowledge to demonstrate that positive urgency is elevated in BED. Replication studies are therefore necessary to confirm this finding. Moreover, in the current study urgency (both negative and positive) was compared across groups using ANOVAs but multiple regression analyses were not conducted to examine the relationship between these variables and eating disorder pathology. Since the current study has demonstrated an association between DERS total score and eating disorder psychopathology, future studies should investigate whether negative and/or positive urgency are also associated with these variables, and whether one contributes unique variance above the other. Importantly, examining changes in negative and positive urgency across treatment and how this relates to treatment outcome are necessary to strengthen the assertion presented here that negative and positive urgency should be a focus of BED treatment.

## 5.0 Conclusion

Understand the etiology of BED is crucial to developing and implementing effective treatment programs. The current study provides evidence in support of an emotion regulation model of BED, demonstrating that individuals with BED report greater difficulties in emotion regulation, as well as negative and positive urgency, compared to individuals with NED. These differences persist even after controlling for BMI, age, depression, and overvaluation of weight and shape. Moreover, we report a positive association between DERS total score and EDE-Q Global score, indicating that individuals with greater difficulties in emotion regulation also reported greater eating disorder psychopathology. While DERS total score did not predict binge frequency after controlling for BMI, age, and depression, a moderation analysis revealed a significant DERS by BSI interaction. These findings suggest that the relationship between emotion regulation and binge frequency is strongest in individuals who report greater depression. Although not anticipated, this finding is consistent with the emotion regulation model suggesting that the effect of difficulties in emotion regulation is most apparent for individuals who experience the highest levels of emotionality. Finally, there was no evidence for an interaction between insomnia symptoms and difficulties in emotion regulation, though it is noted that this was the first analysis of this nature in BED and future research is necessary to corroborate the result. The findings presented here add to a growing body of literature supporting a role for emotion regulation in the development and maintenance of BED. Future research directions include focusing on subjective, as well as objective, binge episodes, examining the importance of depression subtypes in BED, and extending the role of positive and negative urgency, while clinical implications

suggest the development and implementation of DBT programs for BED. Ultimately, the current study provides comprehensive evidence for the emotion regulation model of BED and a foundation for the development of targeted treatment programs.

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**Tables.**

*Table 1*. Reasons for ineligibility following screening in the NED group.

| Reason for Ineligibility | Frequency (%) |
|---|---|
| **Reported binge episodes** | 55 (49.5) |
| **Reported previous or current ED diagnosis** | 15 (13.5) |
| **Exceeded SCOFF cutoff** | 13 (11.7) |
| **Exceeded DAST or AUDIT cutoff** | 9 (8.1) |
| **Not between 19-65** | 6 (5.4) |
| **Hypothyroidism** | 3 (2.7) |
| **On antidepressants for less than 3 months** | 2 (1.8) |
| **Less than Gr. 12 education** | 1 (0.9) |
| **Other** | 1 (0.9) |
| **Met two exclusion criteria** | 6 (5.4) |

*Note.* SCOFF: screening questionnaire for likelihood of eating disorder. DAST: Drug abuse screening questionnaire. AUDIT: Alcohol use disorder identification test.

*Table 2.* Reasons for ineligibility following screening in the BED group.

| Reason for Ineligibility | Frequency (%) |
|---|---|
| **Did not report binge episodes** | 64 (26.3) |
| **Reported not having access to technology or wifi in a private location** | 27 (11.1) |
| **Not between 19 and 64** | 24 (9.9) |
| **Reported Type II Diabetes** | 23 (9.5) |
| **Reported Hypothyroidism** | 23 (9.5) |
| **Reported compensatory behaviours** | 14 (5.8) |
| **Gastric bypass** | 10 (4.1) |
| **On antidepressants for less than 3 months** | 8 (3.3) |
| **Less than Gr. 12 education** | 7 (2.9) |
| **Currently taking a stimulant for ADHD** | 5 (2.1) |
| **Exceeded the cutoff on the DAST or AUDIT** | 3 (1.2) |
| **Reported current ED diagnosis other than BED and/or currently receiving treatment at the Hope ED program** | 3 (1.2) |
| **Did not report binge eating at least once per week over the last three months on average** | 3 (1.2) |
| **Reported a serious medical condition (e.g., MS)** | 1 (0.4) |
| **Other** | 9 (3.7) |
| **Met two exclusion criteria** | 18 (7.4) |
| **Met three exclusion criteria** | 1 (0.4) |

*Note.* SCOFF: screening questionnaire for likelihood of eating disorder. DAST: Drug abuse screening questionnaire. AUDIT: Alcohol use disorder identification test.

*Table 3.* Reasons for ineligibility following telephone interview in BED group.

| Reason for Ineligibility | Frequency (%) |
|---|---|
| **Did not meet EDE criteria for BED** | |
| **Did not reported objectively large binge eating episodes** | 31 (44.9) |
| **Reported regular compensatory behaviour** | 21 (30.4) |
| **Did not report loss of control** | 6 (8.7) |
| **Objective binge episodes occurred less than once per week over the last three months on average** | 4 (5.8) |
| **Did not report distress about binge eating** | 1 (1.4) |
| **Did not endorse 3/5 binge characteristics** | 1 (1.4) |
| **Currently seeing a registered psychologist for problems related to binge eating** | 2 (2.9) |
| **Diabetes** | 1 (1.4) |
| **Other** | 1 (1.4) |
| **Did not complete interview** | 1 (1.4) |

*Table 4.* Sex, marital status, and ethnicity for the NW, OW, and BED groups.

| | Group | | |
|---|---|---|---|
| | **NW** | **OW** | **BED** |
| | Frequency (%) | Frequency (%) | Frequency (%) |
| **Biological Sex** | | | |
| **Male** | 8 (21.1) | 5 (11.9) | 4 (6.5) |
| **Female** | 30 (78.9) | 37 (88.1) | 58 (93.5) |
| **Marital Status** | | | |
| **Single** | 20 (52.6) | 16 (38.1) | 22 (35.5) |
| **Married/Common Law** | 17 (44.7) | 24 (57.1) | 36 (58.1) |
| **Divorced** | 0 (0) | 2 (4.8) | 3 (4.8) |
| **Widowed** | 0 (0) | 0 (0) | 0 (0) |
| **Separated** | 1 (2.6) | 0 (0) | 1 (1.6) |
| **Ethnicity** | | | |
| **Caucasian/White** | 34 (89.5) | 41 (97.6) | 60 (96.8) |
| **Hispanic** | 0 (0) | 1 (2.4) | 0 (0) |
| **Black** | 1 (2.6) | 0 (0) | 0 (0) |
| **Asian** | 1 (2.6) | 0 (0) | 0 (0) |
| **Other** | 2 (5.3) | 0 (0) | 2 (3.2) |

*Table 5.* Age and BMI for individuals in the NW, OW, and BED groups.

| | Group | | |
|---|---|---|---|
| | **NW** | **OW** | **BED** |
| | Mean (SD) | Mean (SD) | Mean (SD) |
| **Age** | 31.34 (12.796)[a] | 37.55 (14.681)[ab] | 40.29 (11.576)[b] |
| **BMI** | | | |
| **Current** | 21.984 (1.815)[a] | 31.023 (4.575)[b] | 37.600 (9.550)[c] |
| **Lowest adult** | 19.750 (2.544)[a] | 24.655 (2.126)[b] | 25.9516 (6.558)[b] |
| **Highest adult** | 23.949 (3.089)[a] | 34.251 (5.380)[b] | 39.936 (9.535)[c] |

*Note.* Different superscripts (i.e., a, b, c) indicate differences between groups.

*Table 6.* Number of individuals who self reported being overweight and having gone on a diet across groups.

| | Group | | |
|---|---|---|---|
| | **NW** | **OW** | **BED** |
| | Frequency (%) | Frequency (%) | Frequency (%) |
| **Do you currently consider yourself to be overweight?** | | | |
| **Yes** | 2 (5.3)[a] | 37 (88.1)[b] | 59 (95.2)[b] |
| **No** | 36 (94.7)[a] | 5 (11.9)[b] | 3 (4.8)[b] |
| **Have you previously gone on diets to control your weight?** | | | |
| **Yes** | 19 (50)[a] | 36 (85.7)[b] | 59 (95.2)[b] |
| **No** | 19 (50)[a] | 6 (14.3)[b] | 3 (4.8)[b] |

*Note.* Different superscripts (i.e., a, b, c) indicate differences between groups.

*Table 7.* Proportion of individuals who reported an EDE-Q score ≥ 4.

| | Group | | |
|---|---|---|---|
| | **NW** | **OW** | **BED** |
| | Frequency (%) | Frequency (%) | Frequency (%) |
| **Reported an EDE-Q Global score ≥ 4?** | | | |
| **Yes** | 4 (10.5)[a] | 15 (35.7)[a] | 45 (72.6)[b] |
| **No** | 34 (89.5)[a] | 27 (64.3)[a] | 17 (27.4)[b] |

*Note.* Different superscripts (i.e., a, b, c) indicate differences between groups.

*Table 8.* Clinical characteristics of the NW, OW, and BED groups.

| | Group | | |
|---|---|---|---|
| | **NW** | **OW** | **BED** |
| | Mean (SD) | Mean (SD) | Mean (SD) |
| **EDE-Q** | | | |
| **Global** | 2.003 (1.383)[a] | 3.303 (1.476)[b] | 4.482 (0.918)[c] |
| **Dietary Restraint** | 1.746 (1.889)[a] | 2.873 (2.077)[b] | 3.269 (2.063)[b] |
| **Overvaluation of Weight and Shape** | 2.026 (1.896)[a] | 2.976 (2.092)[a] | 4.694 (1.535)[b] |
| **Body Dissatisfaction** | 2.237 (1.663)[a] | 4.060 (1.839)[b] | 5.484 (0.815)[c] |
| **BSI** | | | |
| **Depression** | 0.781 (.721)[a] | 0.889 (.930)[a] | 1.393 (0.811)[b] |

*Note.* Different superscripts (i.e., a, b, c) indicate differences between groups.

*Table 9.* DERS total and subscale scores.

| | Group | | |
|---|---|---|---|
| | **NW** | **OW** | **BED** |
| | Mean (SD) | Mean (SD) | Mean (SD) |
| **DERS** | | | |
| **Total*** | 75.308 (21.679)[a] | 74.805 (23.369)[a] | 99.332 (23.815)[b] |
| **Non-acceptance*** | 11.505 (4.828)[a] | 11.638 (5.229)[a] | 17.081 (6.458)[b] |
| **Goals*** | 15.250 (5.055)[ab] | 13.143 (4.905)[a] | 16.407 (5.048)[b] |
| **Impulsivity*** | 9.684 (4.954)[a] | 10.357 (4.848)[a] | 14.694 (5.355)[b] |
| **Awareness** | 14.079 (5.247)[a] | 14.762 (5.122)[a] | 17.919 (5.570)[b] |
| **Strategies*** | 14.395 (6.502)[a] | 14.619 (6.336)[a] | 21.489 (7.698)[b] |
| **Clarity** | 10.395 (3.553) | 10.286 (3.604) | 11.742 (4.012) |

*Note.* Different superscripts (i.e., a, b, c) indicate significant differences between scores. * indicates that the difference remained significant after controlling for age, BMI, and BSI depression score.

*Table 10.* Effect sizes (Cohen's d) for comparisons across DERS total and subscale scores.

| | Comparison | |
|---|---|---|
| | **BED v. NW** | **BED v. OW** |
| **DERS** | | |
| **Total** | 1.054*** | 1.048*** |
| **Non-acceptance** | 0.863*** | 0.843*** |
| **Goals** | 0.231 | 0.660** |
| **Impulsivity** | 0.926*** | 0.839*** |
| **Awareness** | 0.712** | 0.591* |
| **Strategies** | 0.986*** | 0.966*** |
| **Clarity** | 0.354 | 0.382 |

*Note.* * indicates $p < .05$; ** indicates $p < .01$; *** indicates $p < .0005$

*Table 11.* Effect sizes (Cohen's d) for comparisons across UPPS-P total and subscale scores.

| | Comparison | |
|---|---|---|
| | **BED v. NW** | **BED v. OW** |
| **UPPS-P** | | |
| **Total** | 0.794** | 0.609** |
| **Negative Urgency** | 1.497*** | 0.943*** |
| **Premeditation** | 0.446 | 0.448* |
| **Perseverance** | 0.567* | 0.562** |
| **Sensation Seeking** | 0.668** | 0.332 |
| **Positive Urgency** | 0.958*** | 0.645*** |

*Note.* * indicates $p < .05$; ** indicates $p < .01$; *** indicates $p < .0005$

*Table 12.* UPPS-P total and subscale scores.

| | Group | | |
|---|---|---|---|
| | **NW** | **OW** | **BED** |
| | Mean (SD) | Mean (SD) | Mean (SD) |
| **UPPS-P** | | | |
| **Total*** | 1.943 (0.317)[a] | 1.959 (0.418)[a] | 2.254 (0.433)[b] |
| **Negative Urgency*** | 2.026 (0.569)[a] | 2.206 (0.670)[a] | 2.836 (0.528)[b] |
| **Premeditation** | 1.854 (0.372)[ab] | 1.823 (0.381)[a] | 2.070 (0.544)[b] |
| **Perseverance** | 1.921 (0.499)[a] | 1.883 (0.496)[a] | 2.227 (0.567)[b] |
| **Sensation Seeking** | 2.518 (0.613)[a] | 2.341 (0.625)[ab] | 2.105 (0.626)[b] |
| **Positive Urgency *** | 1.943 (0.317)[a] | 1.582 (0.564)[a] | 2.046 (0.676)[b] |

*Note.* Different superscripts indicate significant differences between scores. * indicates that the difference remained significant after controlling for age, BMI, and BSI depression score.

*Table 13.* Coefficients for the bivariate and hierarchical regression analyses with binge frequency as the criterion.

| | **Unstandardized Coefficients** | | **Standardized Coefficients** | |
|---|---|---|---|---|
| | **B** | **Standard error** | **Beta** | **t** |
| **Model** | | | | |
| **1** | | | | |
| **DERS (1)** | 0.940 | 0.281 | 0.396 | 3.341** |
| **2** | | | | |
| **Age (1)** | 0.987 | 0.571 | 0.202 | 1.729 |
| **BMI (1)** | -0.513 | 0.673 | -0.087 | -0.763 |
| **BSI (2)** | 27.611 | 9.996 | 0.396 | 2.762** |
| **DERS (3)** | 0.514 | 0.347 | 0.217 | 1.482 |
| **3** | | | | |
| **Age (1)** | 0.979 | 0.578 | 0.201 | 1.693 |
| **BMI (1)** | -0.523 | 0.682 | -0.088 | -0.768 |
| **Overvaluation (2)** | 0.752 | 4.956 | 0.020 | 0.152 |
| **BSI (3)** | 27.296 | 10.294 | 0.392 | 2.652* |
| **DERS (4)** | 0.496 | 0.379 | 0.209 | 1.340 |

*Notes.* Numbers in brackets indicate the block in which each predictor was added. * indicates $p < .05$. ** indicates $p < .01$.

*Table 14.* Coefficients for the bivariate and hierarchical regression analyses with EDE-Q Global score as the criterion.

| | **Unstandardized Coefficients** | | **Standardized Coefficients** | |
|---|---|---|---|---|
| | **B** | **Standard error** | **Beta** | **t** |
| **Model** | | | | |
| **1** | | | | |
| **DERS (1)** | 0.017 | 0.004 | 0.446 | 3.859*** |
| **2** | | | | |
| **Age (1)** | -0.001 | 0.010 | -0.107 | -0.107 |
| **BMI (1)** | 0.013 | 0.011 | 0.131 | 1.104 |
| **BSI (2)** | 0.120 | 0.169 | 0.106 | 0.709 |
| **DERS (3)** | 0.014 | 0.006 | 0.360 | 2.360* |

*Notes.* Numbers in brackets indicate the block in which each predictor was added. * indicates $p < .05$. ** indicates $p < .01$. *** indicates $p < .0005$.

*Table 15.* Coefficients for BSI x DERS moderation analysis with binge frequency as the criterion.

| | Unstandardized Coefficients | | Standardized Coefficients | |
|---|---|---|---|---|
| | **B** | **Standard error** | **Beta** | **t** |
| **Block** | | | | |
| **1** | | | | |
| **DERS** | 0.378 | 0.343 | 0.159 | 1.105 |
| **BSI** | 26.501 | 10.060 | 0.380 | 2.634* |
| **2** | | | | |
| **DERS** | 0.330 | 0.329 | 0.139 | 1.004 |
| **BSI** | 17.995 | 10.246 | 0.258 | 1.752 |
| **DERS x BSI** | 0.772 | 0.311 | 0.301 | 2.479* |

*Note.* * indicates $p < .05$.

*Table 16.* Coefficients for BSI x DERS moderation analysis with EDE-Q Global score as the criterion.

| | Unstandardized Coefficients | | Standardized Coefficients | |
|---|---|---|---|---|
| | **B** | **Standard error** | **Beta** | **t** |
| **Block** | | | | |
| **1** | | | | |
| **DERS** | 0.014 | 0.006 | 0.370 | 2.501* |
| **BSI** | 0.138 | 0.168 | 0.122 | 0.824 |
| **2** | | | | |
| **DERS** | 0.015 | 0.006 | 0.376 | 2.526* |
| **BSI** | 0.179 | 0.179 | 0.158 | 1.001 |
| **DERS x BSI** | -0.004 | 0.005 | -0.089 | -0.680 |

*Note.* * indicates $p < .05$.

*Table 17.* Coefficients for ISI x DERS moderation analysis with binge frequency as the criterion.

| | Unstandardized Coefficients | | Standardized Coefficients | |
|---|---|---|---|---|
| | **B** | **Standard error** | **Beta** | **t** |
| **Block** | | | | |
| **1** | | | | |
| **DERS** | 0.847 | 0.283 | 0.357 | 2.994** |
| **ISI** | 1.710 | 1.035 | 0.197 | 1.653 |
| **2** | | | | |
| **DERS** | 0.793 | 0.293 | 0.334 | 2.710* |
| **ISI** | 1.754 | 1.040 | 0.202 | 1.686 |
| **DERS x ISI** | 0.034 | 0.045 | 0.091 | 0.754 |

*Note.* * indicates $p < .05$. ** indicates $p < .01$.

*Table 18.* Coefficients for ISI x DERS moderation analysis with EDE-Q Global score as the criterion.

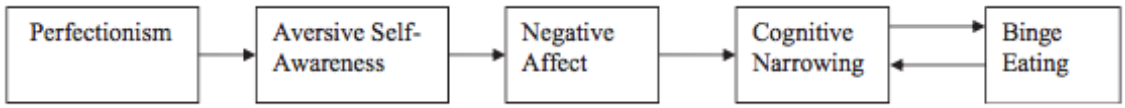
| | Unstandardized Coefficients | | Standardized Coefficients | |
|---|---|---|---|---|
| | **B** | **Standard error** | **Beta** | **t** |
| **Block** | | | | |
| **1** | | | | |
| **DERS** | 0.016 | 0.005 | 0.419 | 3.564** |
| **ISI** | 0.019 | 0.017 | 0.135 | 1.151 |
| **2** | | | | |
| **DERS** | 0.015 | 0.005 | 0.387 | 3.199** |
| **ISI** | 0.020 | 0.017 | 0.142 | 1.211 |
| **DERS x ISI** | 0.001 | 0.001 | 0.128 | 1.078 |

*Note.* * indicates $p < .05$. ** indicates $p < .01$.

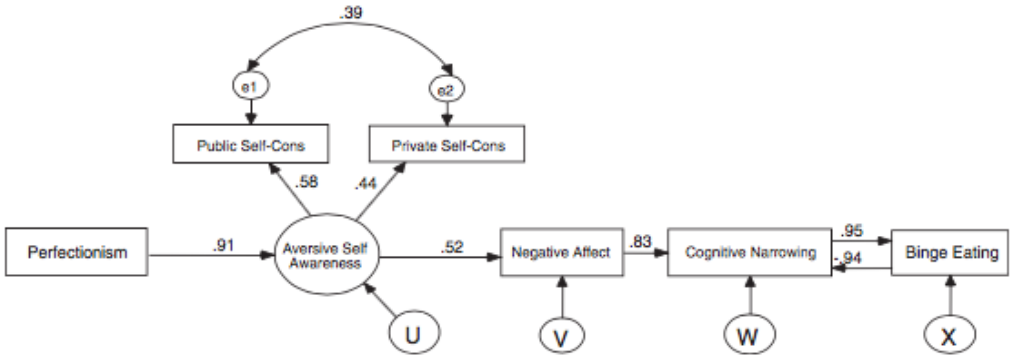
*Table 19.* Summary of pooled and unpooled error terms for Cohen's d calculation.

| Measure | Error Term |
|---|---|
| **Demographics** | |
| **Age** | Pooled |
| **Current BMI** | Not pooled |
| **Lowest adult BMI** | Not pooled |
| **Highest adult BMI** | Not pooled |
| **EDE-Q** | |
| **Global** | Not pooled |
| **Dietary Restraint** | Pooled |
| **Overvaluation** | Not pooled |
| **Body Dissatisfaction** | Not pooled |
| **BSI** | |
| **Depression** | Not pooled |
| **DERS** | |
| **Total** | Pooled |
| **Non-accept** | Not pooled |
| **Goals** | Pooled |
| **Impulse** | Pooled |
| **Aware** | Pooled |
| **Strategies** | Pooled |
| **Clarity** | Pooled |
| **UPPS-P** | |
| **Total** | Pooled |
| **Negative Urgency** | Pooled |
| **Premeditation** | Pooled |
| **Perseverance** | Pooled |
| **Sensation Seeking** | Pooled |
| **Positive Urgency** | Pooled |

**Figures.**




(a)




(b)

*Figure 1*. (a) Components of the escape theory of binge eating disorder. (b) Escape model of binge eating disorder as reported by Blackburn et al., (2006). Figure 1a and 1b have been taken from Blackburn et al., (2006).

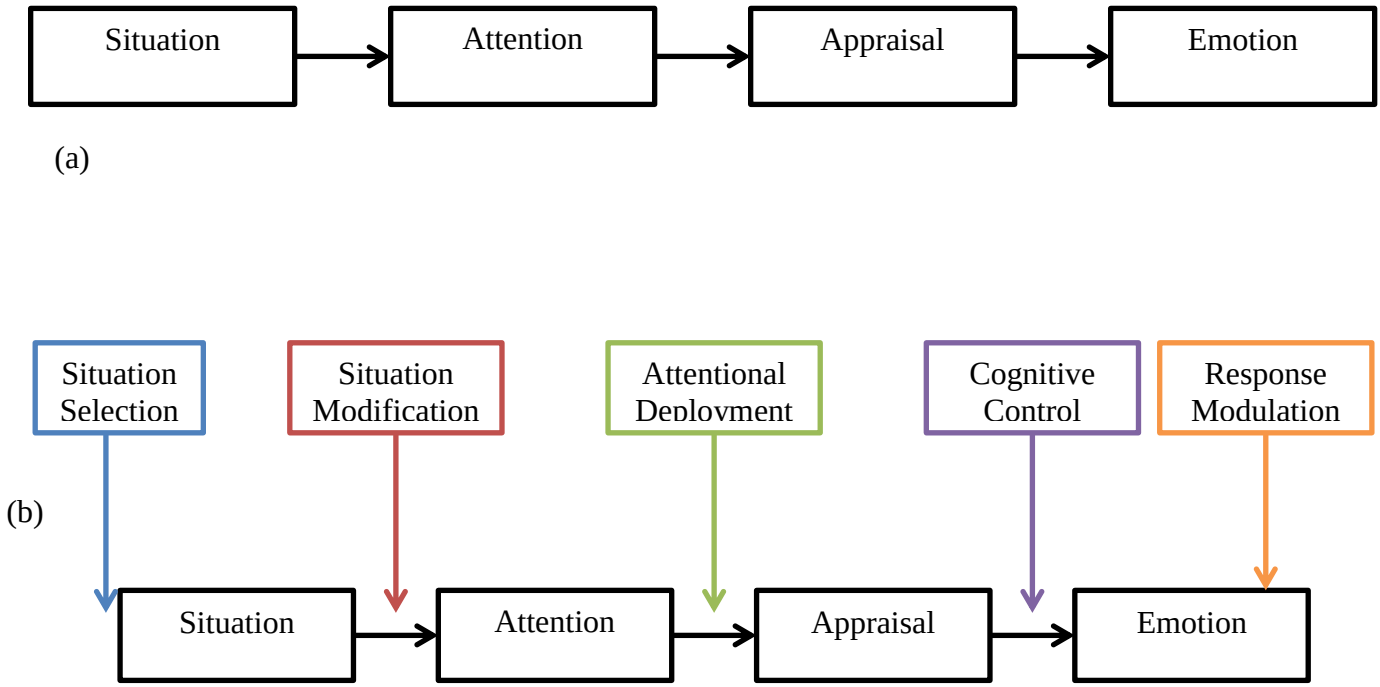



*Figure 2.* (a) The modal model of emotion. (b) Depiction of how the emotion regulation strategies map onto the modal model of emotion. Diagrams have been adapted from Gross and Thompson (2007).

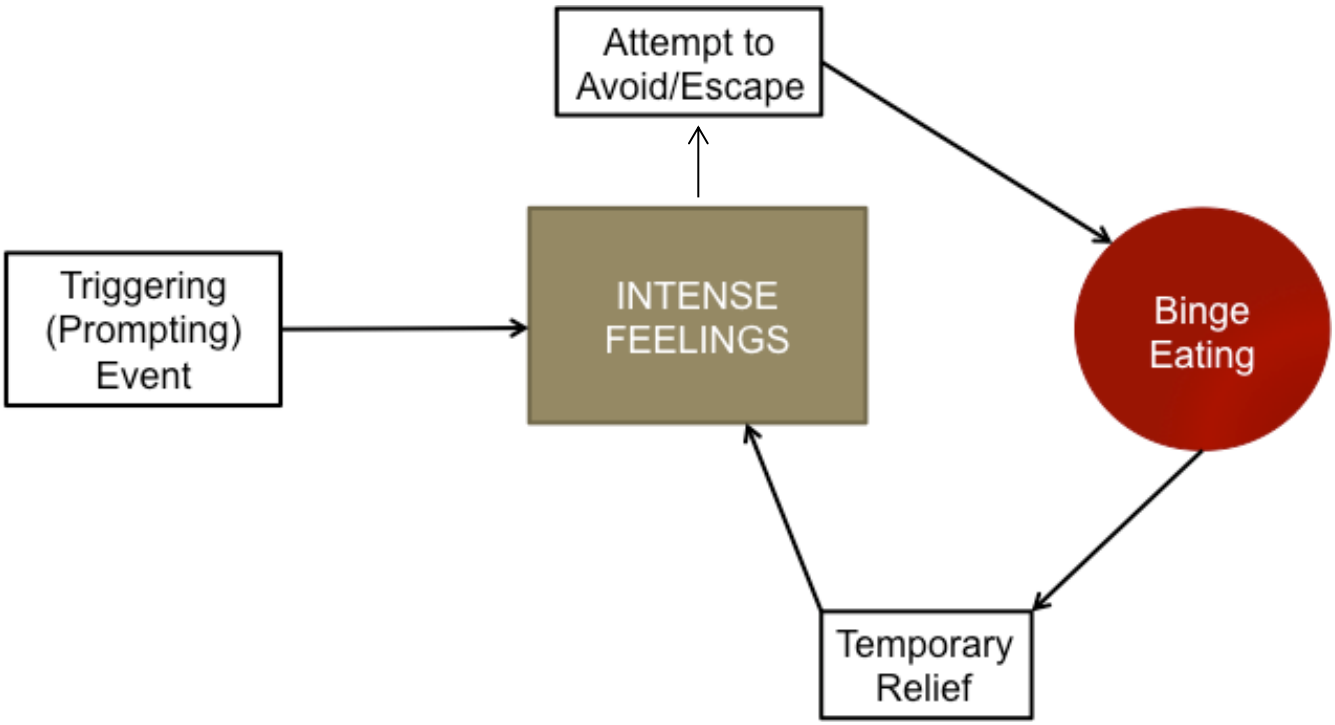
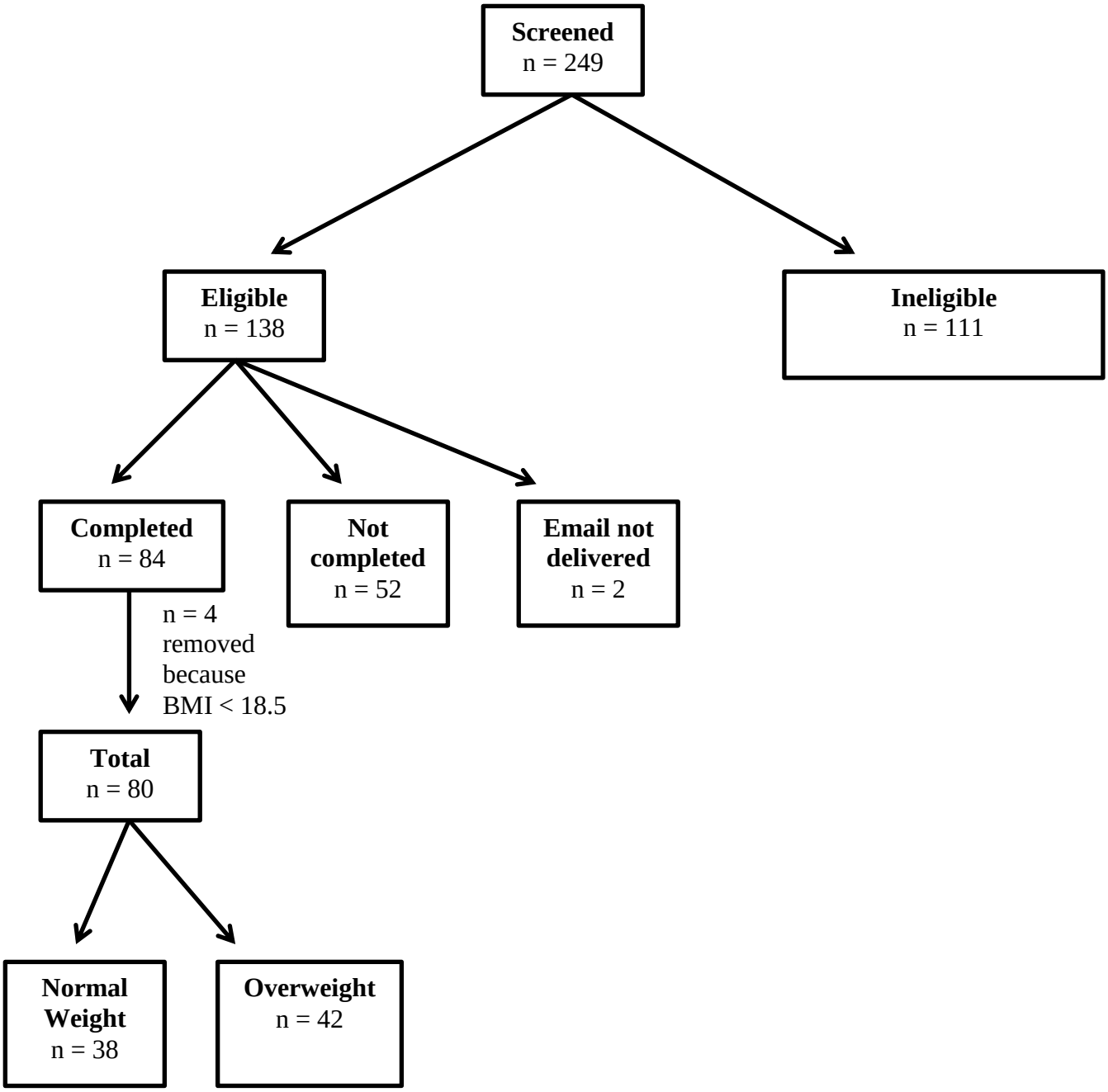



*Figure 3.* The Dialectical Behaviour Theory (DBT) model of binge eating (Safer et al., in press). The model proposes that external or internal events lead to intense emotions. The individual then turns to binge eating as a way of avoiding or escaping these uncomfortable emotions. While this leads to temporary relief, ultimately the individual experiences more intense negative emotions such as guilt or shame after the episode.

(a)


Screened
n = 249
Eligible
n = 138
Ineligible
n = 111
Completed
n = 84
Not completed
n = 52
Email not delivered
n = 2
n = 4 removed because BMI < 18.5
Total
n = 80
Normal Weight
n = 38
Overweight
n = 42

(b)

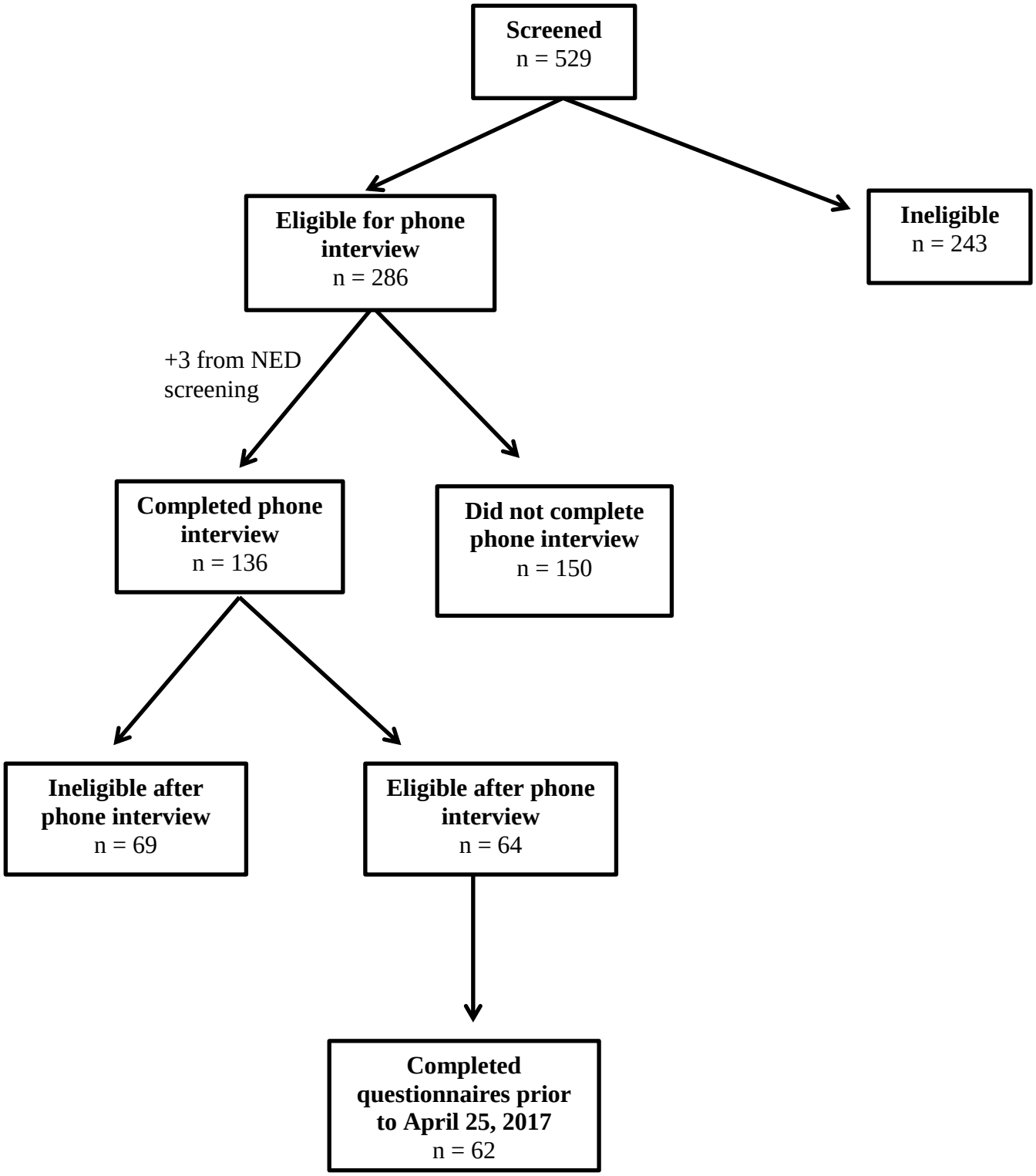
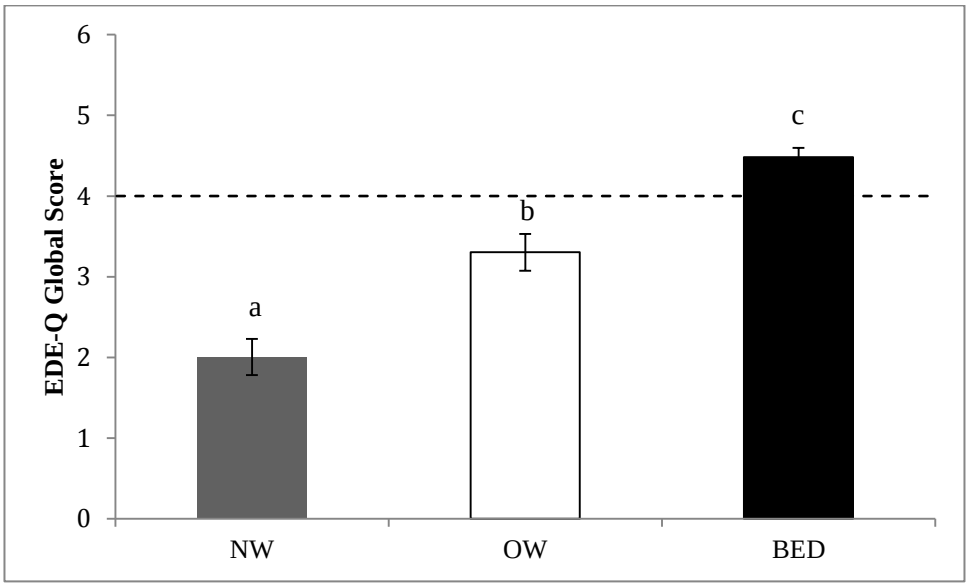
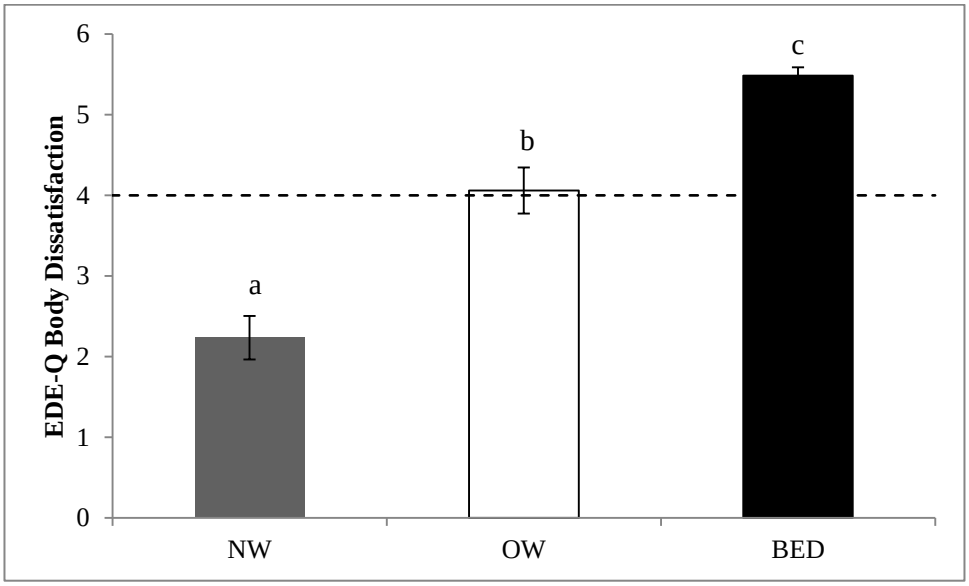

Screened
n = 529
Eligible for phone interview
n = 286
Ineligible
n = 243
+3 from NED screening
Completed phone interview
n = 136
Did not complete phone interview
n = 150
Ineligible after phone interview
n = 69
Eligible after phone interview
n = 64
Completed questionnaires prior to April 25, 2017
n = 62

*Figure 4.* Consort diagrams for the current study. (a) Consort diagram for the NED group. (b) Consort diagram for the BED group.

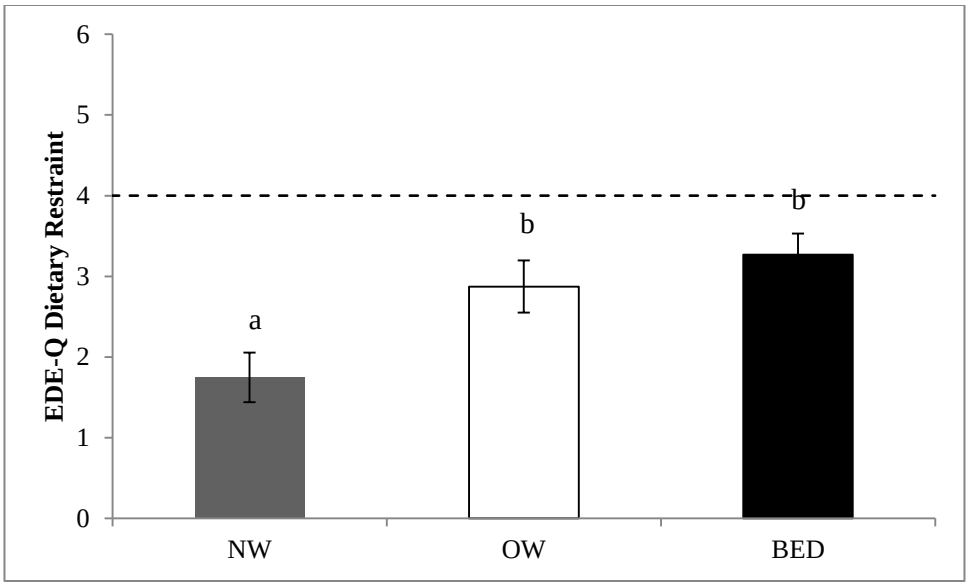
(a)


EDE-Q Global Score
6
5
4
3
2
1
0
a
b
c
NW
OW
BED

(b)


EDE-Q Body Dissatisfaction
6
5
4
3
2
1
0
a
b
c
NW
OW
BED

(c)




(d)

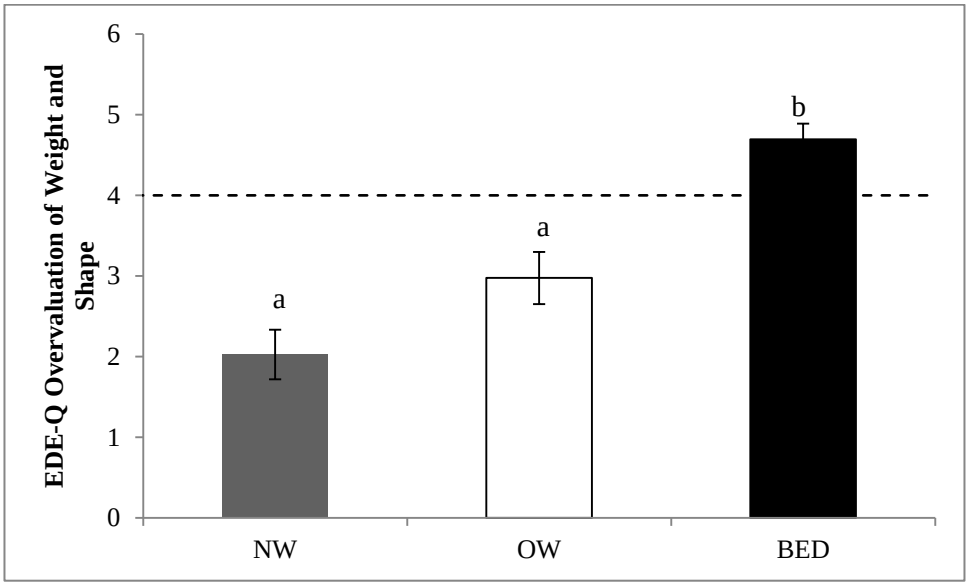



*Figure 5.* Global and EDE-Q subscale scores for individuals in the NW, OW, and BED groups. The clinical cutoff of 4 (Carter et al., 2001 as cited in Panelo et al., 2013) is indicated by the dashed line on each graph. The highest possible score for each

scale is 6. (a) Global score: individuals in the BED group reported greater scores than individuals in the OW group who in turn reported greater scores than those in the NW group. (b) Dietary restraint: Individuals in the NW group reported significantly lower scores than individuals in both the OW and BED groups. (c) Overvaluation: Individuals in the BED group reported greater scores than individuals in both the NW and OW groups. (d) Body dissatisfaction: individuals in the BED group reported greater scores than individuals in the OW group who in turn reported greater scores than those in the NW group.

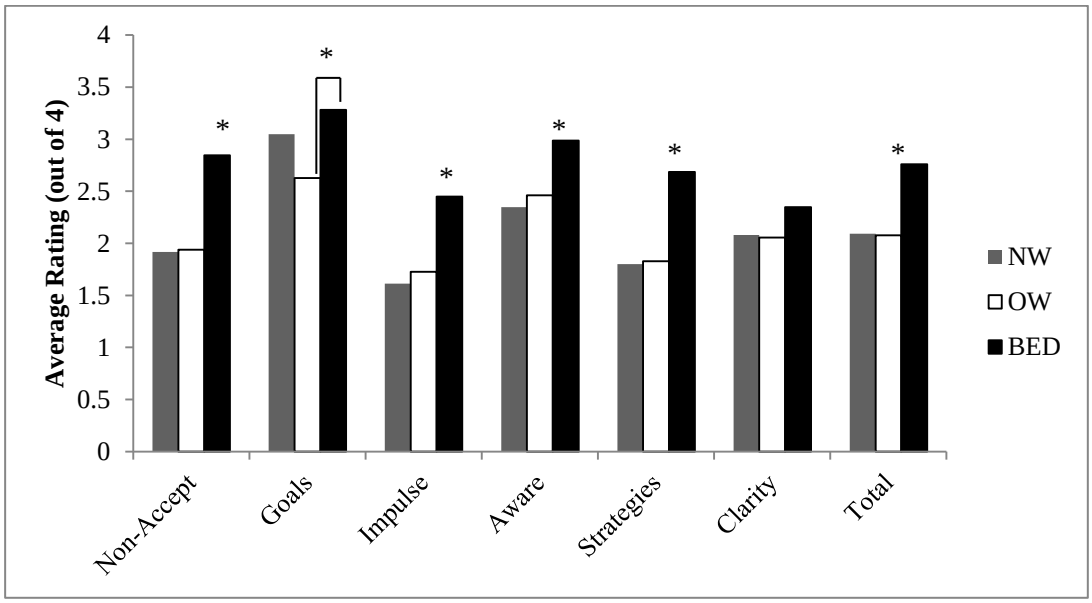



*Figure 6.* Average scores for the DERS subscales and total score. Typically DERS scores are reported as the sum of all the items on each scale. By presenting the average score however, it is possible to compare across subscales. Since the transformation from the total to average score is a linear transformation this does not affect the results of the statistical analysis. After controlling for BMI, age, depression, and overvaluation of weight and shape, differences persisted on the Total, Non-accept, Goals, Aware, and Strategies scales.

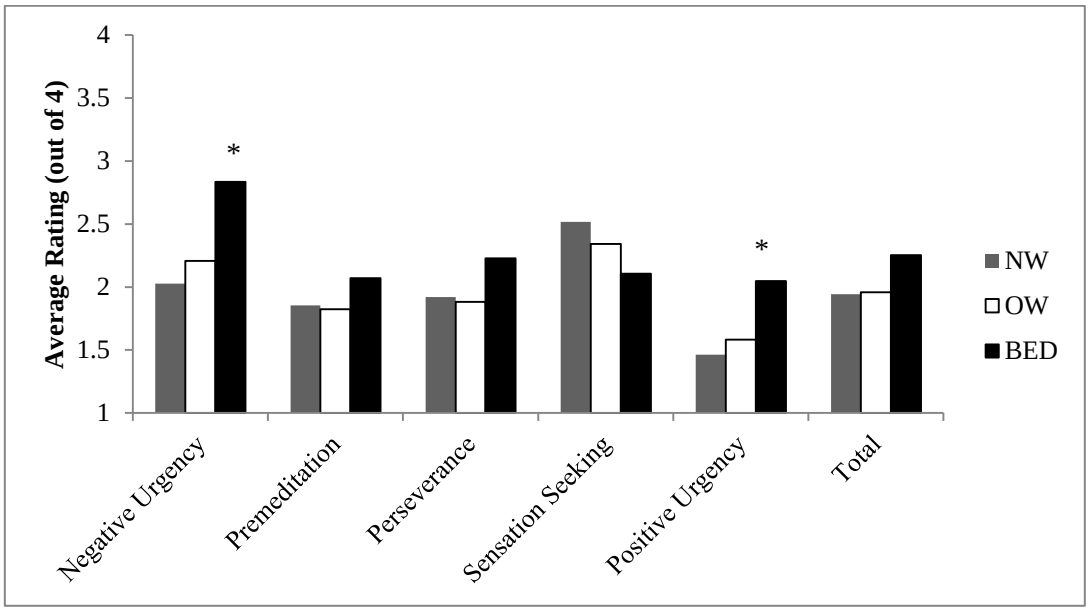



*Figure 7.* Average scores for UPPS-P total and subscales. After controlling for BMI, age, depression and overvaluation of weight and shape, differences persisted on the Negative and Positive Urgency subscales.

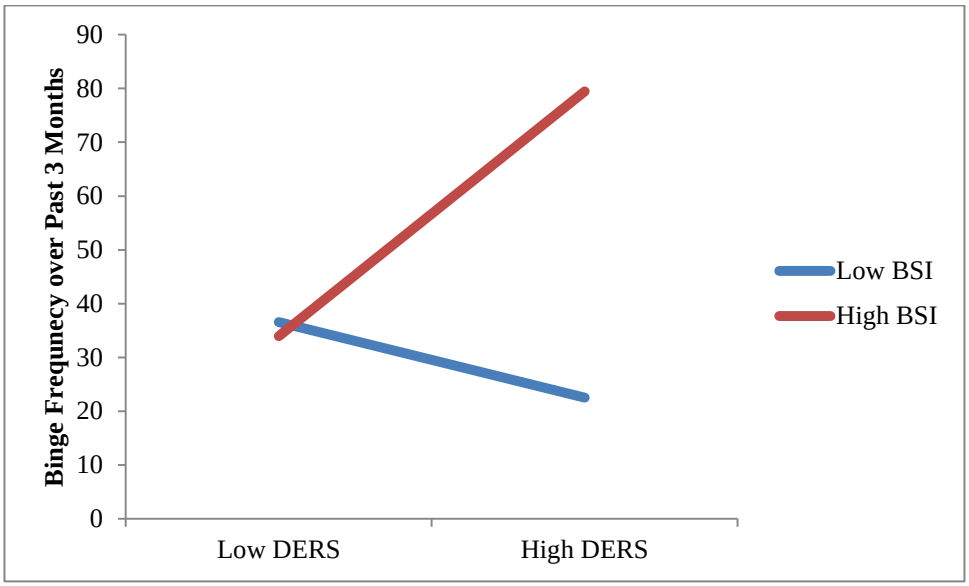



*Figure 8.* Results from the depression by difficulties in emotion regulation moderation analysis. Note that only the High BSI (red line) relationship is significant.

‘

Appendix A

**Telephone Screening Interview Script**
**EDE-17.0 (Fairburn et al., 2014)**

Hello [participant's name]. My name is [researcher's name], and I'm a researcher in the psychology department at Memorial University. I'm a part of the study on overeating that you have expressed interest in.

Thank you for your interest in taking part. Is this a convenient time for me to tell you a few things about the study and to make sure you match the criteria for the study?

Here's what we're going to do [today/this evening]. To see whether you meet the criteria to participate in this study, I am going to ask you a few questions about your eating habits, with a focus on overeating. I know that this can be difficult to discuss but it is really important that we get a clear picture of your eating habits to figure out if this study is suitable for you.

Please note: your participation in this interview is voluntary and you can discontinue at any time. Also, completing this interview does not commit you to taking part in this study. All information that you disclose in this interview is confidential, and will be not be shared with anyone except the research team. Do you have any questions before we begin?

As these interviews are designed in a certain way, it is important that I read through the full instructions as they have been written by the authors before we begin. Also, I just wanted to let you know that the interview will be very focused on specific episodes of overeating. Do you have any questions before we start?

**EATING DISORDER EXAMINATION (Edition 17.0D)**

Copyright 2014 by Christopher G Fairburn, Zafra Cooper and Marianne O'Connor

**THE INTERVIEW SCHEDULE ORIENTATION TO THE TIME PERIOD**

What we are going to do is an interview in which I will ask you about your eating habits. Because a standard set of questions is going to be asked, please note that some may not apply to you. The questions focus on the past four weeks, but there will be some that cover the previous three and six months. The past four weeks go from yesterday (day and date) to (day and date). And two months before that go from (date) to (date). And to help you remember these periods, I have noted down the recent holidays (e.g., Canada Day, Thanksgiving).

**QUESTIONS FOR IDENTIFYING BINGE EPISODES** [See preceding section "Guidelines for Proceeding Through the Overeating Section". The asterisked questions should be asked in every case.]

**Main Probe Questions** (to get the overall picture)

***To begin, I would like to get a sense of your typical eating habits. In the past 4 weeks [since DATE], what has a typical day of eating looked like for you?**

***What time would you get up in the morning? Do you typically eat breakfast? What would you usually have? And when would you eat next?...** [Get a clear picture of a typical day of eating including times and amounts. Might need to ask about week days versus weekends.].

**Overeating Episodes**

***Next, I would like to ask you about any episodes of *overeating*, or *loss of control over eating*, that you might have had over the past four weeks.**

***Different people mean different things by overeating. I would like you to describe any times when you have felt that you have eaten, or might have eaten, too much at one time.**

***And any times you have felt you have lost control over eating?**

**Subsidiary Probe Questions** (to classify any episodes of overeating) To assess the amount of food eaten:

**Typically what have you eaten at these times?**

**Did you view this amount as excessive?**

To assess the social context:

**What were the circumstances?**
**What were others eating at the time?**

To assess "loss of control":

**Did you have a sense of loss of control at the time?**
**Did you feel you could have stopped eating once you had started?**

**Did you feel you could you have prevented the episode from starting?**

[For objective binge episodes, the following two ratings should be made:

i) Over the past 4 weeks (28 days), on how many of the days did you have an overeating episode like this? number of days (rate 00 if none)
ii) number of episodes (rate 000 if none)

In general, it is best to calculate the number of days first and then the number of episodes. Rate 777 if the number of episodes is so great that their frequency cannot be calculated.

[Episodes of subjective overeating are not rated.]

Objective binge episodes

days [ ][ ]

episodes [ ][ ][ ]

[Ask about each of the preceding two months referring back to the relevant dates and any events of note. For objective binge episodes, rate the number of episodes over the preceding two months and the number of days on which they occurred. Rate 0s if none and 9s if not asked.]

Objective bulimic episodes

days - month 2 _____

month 3 _____

episodes – month 2 _____

month 3 _____

[Also rate the longest continuous period in weeks free (not due to force of circumstances) from objective binge episodes over the past three months. Rate 99 if not applicable.]
[ ][ ]

**BINGE EATING DISORDER MODULE (Diagnostic items)**

[Only enter this module if at least 12 objective binge episodes have been present over the preceding 12 weeks. Otherwise rate 9. Use a respondent-based interviewing style, rather than the investigator-based style of the EDE.]

[Rate each feature individually using the binary scheme below.]

0 - Feature not present

1 - Feature present **Features Associated with Binge Eating**
**During these episodes** (refer to objective binge episodes that are representative of those over the past three months)**, have you *typically* .....**

**... Eaten much more rapidly than normal? [ ]**
**... Eaten until you have felt uncomfortably full? [ ]**
**... Eaten large amounts of food when you haven't felt physically hungry? [ ]**
**... Eaten alone because you have felt embarrassed about how much**

**you were eating? [ ]**

**... Felt disgusted with yourself, depressed, or very guilty? [ ]**

**Distress about Binge Eating**

**In general, over the past three months how distressed or upset have you felt about these episodes** (refer to objective bulimic episodes that are representative of those over the past three months)**?** [Rate the presence of marked distress about the binge eating. This may stem from the actual behaviour itself or its potential effect on body shape and weight.]

0 – No marked distress

1 – Marked

**[ ]**

**SELF-INDUCED VOMITING (Diagnostic item)**

***Over the past six months have you made yourself sick or vomit as a means of controlling your shape or weight, or to compensate for overeating?**
[Rate the number of discrete episodes of self-induced vomiting. If the participant denies that the vomiting is under his or her control, determine whether it has the characteristics that would be expected were it not self-induced (e.g., unpredictability, occurrence in public). If the available evidence suggests that the vomiting is under the participant's control (i.e., it is self-induced), then rate it as such. Accept the participant's definition of an episode. Rate 777 if the number of episodes is so great that it cannot be calculated. Rate 000 if no vomiting.]

[ ][ ][ ]

**LAXATIVE MISUSE (Diagnostic item)**

***Over the past six months have you taken laxatives as a means of controlling your shape or weight, or to compensate for overeating?**
[Rate the number of episodes of laxative-taking as a means of controlling shape, weight or body composition. This should have been the *main* reason for the laxative-taking, although it may not have been the sole reason. Only rate the taking of substances with a true laxative effect. Rate 00 if there was no laxative use or there is doubt whether the laxative-taking was primarily to influence shape, weight or body composition.]

[ ][ ][ ]

**DIURETIC MISUSE (Diagnostic item)**

***Over the past six months have you taken diuretics as a means of controlling your shape or weight, or to compensate for overeating?**
[Rate the number of episodes of diuretic-taking as a means of controlling shape, weight or body composition. This should have been the *main* reason for the diuretic-taking, although it may not have been the sole reason. Only rate the taking of substances with a true diuretic effect. Rate 00 if there was no diuretic use or there is doubt whether the diuretic-taking was primarily to influence shape, weight or body composition.]

[ ][ ][ ]

**DRIVEN EXERCISING (Diagnostic item)**

***Over the past six months have you exercised as a means of controlling your weight, altering your shape or amount of fat, burning off calories, or to compensate for overeating?**

***Have you felt driven or compelled to exercise?**

**Typically, what form of exercise have you done? How hard have you exercised? Have you pushed yourself?**

**Have you exercised even when it might interfere with other commitments or do you harm?**

**Have there been times when you have been unable to exercise for any reason? How has this made you feel?**
[Rate the number of days on which the participant has engaged in "driven" exercising. Such exercising should have been intense in character and have had a "compulsive" quality to it. The participant may describe having felt compelled to exercise. Other indices of this compulsive quality are exercising to the extent that it significantly interferes with day-to-day functioning (e.g. such that it prevents attendance at social commitments or it intrudes on work or exercising when it might do one harm (e.g., when possibly injured). Another suggestive feature is having a strong negative reaction to being unable to exercise. **Only rate driven exercising that was *predominantly* intended to use calories or change shape, weight, or body composition.** Exercising that was

exclusively intended to enhance health or fitness should not be rated. Rate 00 if no such driven exercising.]

[ ][ ]

[Rate the *average* amount of time (in minutes) per day spent exercising in this way. Only consider days on which the participant has exercised. Rate 999 if no such exercising.]

[ ][ ][ ]

[Ask about the preceding two months. Rate the number of days on which the participant has exercised in this manner over each of the two preceding months. If not asked, rate 99.]

month 2 [ ][ ]
month 3 [ ][ ]

**OTHER EXTREME WEIGHT-CONTROL BEHAVIOUR ( Diagnostic item)**

***Over the past six months have you done anything else to control your shape or weight, or to compensate for overeating?**

[Rate other noteworthy (i.e., potentially effective) dysfunctional forms of weight-control behaviour (e.g., spitting, insulin under-use, thyroid medication misuse). Rate number of days and nature of the behaviour. Rate 99 if no such behaviour.]

month 1 [ ][ ]
month 2 [ ][ ]
month 3 [ ][ ]

**AVOIDANCE OF EATING (Restraint subscales) *Over the past six months have you gone for periods of eight or more waking hours without eating anything?**
**Has this been to influence your shape or weight or to compensate for overeating?**

[Rate the number of days on which there has been at least eight hours abstinence from eating food (soup and milkshakes count as food, whereas drinks in general do not) during waking hours. It may be helpful to illustrate the length of time (e.g., 9 a.m. to 5 p.m.). The abstinence must have been at least partly *self- imposed* rather than being due to force of circumstances. It should have been intended to influence shape, weight or body composition, or to avoid triggering an episode of overeating, although this may not have been the sole or main reason (i.e., fasting for religious or political reasons would not count). Note that the rating should be consistent with those made earlier for "Pattern of eating".]

[ ][ ][ ]

**END OF EDE**

Thank you for taking part in this interview today. All of your answers are confidential and will be stored securely and without any identifying information.
In terms of next steps, we will next review your answers and then we will then send you an email very soon to let you know whether you are eligible to take part in the study. If you are eligible to take part, then we will then give you more information about the study. Do you have any questions?
Thank you so much for your time today. If you have any questions that you didn't get to ask, don't hesitate to contact us at [PHONE]. Enjoy your [day/evening]. We'll be in touch soon.

**END OF TELEPHONE INTERVIEW**

# Appendix B

## EDE-17 Criteria for a Diagnosis of BED (Fairburn et al., 2014)

To obtain a diagnosis of BED the individual must meet the following five criteria:

A. Presence of "Objective Bulimic Episodes" described as consuming an amount of food over a time frame that would be considered large by another person.

B. Three or more of the following are present:

    a. Eating faster than usual

    b. Eating until uncomfortably full

    c. Eating large amounts of food when no physically hungry

    d. Eating alone because of feeling embarrassed about how much one eats

    e. Feeling disgusted with oneself, depressed or guilty after a binge

    At least three of the relevant items from the BED module on the EDE-17 must rated positively to meet this criterion.

C. Marked distress is present. The relevant item from the BED module is rated positively.

D. Binge eating must have occurred on average once a week for the past three months:

    a. The individual must report at least 12 OBEs over the past three months

    b. **AND** there cannot be a period of more than two weeks between OBEs (not due to force of circumstances)

E. The binge eating is not associated with the use of inappropriate compensatory behaviours and does not occur exclusively during a period of anorexia nervosa.

a. The bulimia nervosa criteria for compensatory behaviour are not met. Compensatory behaviour is defined as meeting at least one of the following criteria:
    i. At least 12 episodes of self induced vomiting over the past three months
    ii. At least 12 episodes of laxative misuse over the past three months
    iii. At least 12 episodes of diuretic misuse over the past three months
    iv. At least 12 days over the past three months on which the individual has engaged in driven exercise
    v. At least 12 days over the past three months on which the individual has engaged in other extreme weight control behaviour
    vi. Dietary restriction outside the bulimic episodes should be rated a 1 or 2 for each of the past three months
    vii. **AND** a rating of no higher than 2 on "Periods of absence of extreme weight-control behaviour"
b. **AND** the criteria for anorexia nervosa are not met

# Appendix C

## SCOFF Questionnaire (Morgan et al., 1999)

1. Do you make yourself **S**ick (vomit) because of feeling uncomfortably full?
2. Do you worry that you have lost **C**ontrol over how much you eat?
3. Have you recently lost more than **O**ne stone (15 lbs) in a 3-month period?
4. Do you believe yourself to be **F**at when others say you are thin?
5. Would you say that **F**ood dominates your life?

Appendix D

Drug Abuse Screening Test (DAST-10; Skinner, 1972)

| **In the past 12 months... Circle** | | | |
|---|---|---|---|
| 1. | Have you used drugs other than those required for medical reasons? | Yes | No |
| 2. | Do you abuse more than one drug at a time? | Yes | No |
| 3. | Are you unable to stop abusing drugs when you want to? | Yes | No |
| 4. | Have you ever had blackouts or flashbacks as a result of drug use? | Yes | No |
| 5. | Do you ever feel bad or guilty about your drug use? | Yes | No |
| 6. | Does your spouse (or parents) ever complain about your involvement with drugs? | Yes | No |
| 7. | Have you neglected your family because of your use of drugs? | Yes | No |
| 8. | Have you engaged in illegal activities in order to obtain drugs? | Yes | No |
| 9. | Have you ever experienced withdrawal symptoms (felt sick) when you stopped taking drugs? | Yes | No |
| 10. | Have you had medical problems as a result of your drug use (e.g. memory loss, hepatitis, convulsions, bleeding)? | Yes | No |
| **Scoring:** Score 1 point for each question answered "Yes," except for question 3 for which a "No" receives 1 point. | | **Score:** | |

Appendix E

Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993)

| Questions | 0 | 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| 1. How often do you have a drink containing alcohol? | Never | Monthly or less | 2-4 times a month | 2-3 times a week | 4 or more times a week |
| 2. How many drinks containing alcohol do you have on a typical day when you are drinking? | 1 or 2 | 3 or 4 | 5 or 6 | 7 to 9 | 10 or more |
| 3. How often do you have six or more drinks on one occasion? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 4. How often during the last year have you found that you were not able to stop drinking once you had started? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 5. How often during the last year have you failed to do what was normally expected of you because of drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 7 How often during the last year have you had a feeling of guilt or remorse after drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |

| | | | | | |
|---|---|---|---|---|---|
| 8. How often during the last year have you been unable to remember what happened the night before because of your drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 9. Have you or someone else been injured because of your drinking? | No | | Yes, but not in the last year | | Yes, during the last year |
| 10. Has a relative, friend, doctor, or other health care worker been concerned about your drinking or suggested you cut down? | No | | Yes, but not in the last year | | Yes, during the last year |

# Appendix F

## Demographics Questionnaire

Page 1

Hello! Thank you for taking part in the *MUN Stop Overeating Study*.

Before we can randomize you to one of the study self-help programs, we need you to please fill in the questionnaires below. These should take about 60 minutes to complete. Once you have submitted your responses you will get an email from a member of our team. This email will tell you more about the program you will be following for the next twelve (12) weeks.

We would like to take this time to remind you that all of your responses are ***confidential***, and will only be seen by the research team for this study.

We look forward to working with you to help you overcome your overeating.

Page 2

How old are you (in years)? _____

What was your biological sex at birth?

- ☐ Male
- ☐ Female

What gender do you identify as?

- ☐ Male
- ☐ Female
- ☐ Other

What is your marital status?

- ☐ Single
- ☐ Married/Common Law
- ☐ Divorced
- ☐ Widowed
- ☐ Separated

What is your ethnicity?

- ☐ Caucasian/White
- ☐ Hispanic
- ☐ Black
- ☐ Asian
- ☐ Other

Page 3

What is your current height? _____

What is your current weight? (please weigh yourself before answering) _____

What is the highest weight you've been as an adult? _____

When did you reach that weight? _____

How long did you stay at that weight? _____

What was your lowest adult weight? _____

When did you reach that weight? _____

How long did you stay at that weight? _____

Page 4

To the best of your memory, how old were you when you began binge eating? _____ (ONLY FOR BED GROUP)

Have you ever received treatment for binge eating? (ONLY FOR BED GROUP)

- ☐ Yes
- ☐ No

If yes, when? _____

Do you currently consider yourself to be overweight?

- ☐ Yes
- ☐ No

If yes, to the best of your memory how old were you when you were first overweight? _____

Have you previously gone on diets to control your weight (it does not matter if you consider them successful or not)?

- ☐ Yes
- ☐ No

If yes, to the best of your knowledge how old were you when you first went on a diet? _____

Appendix G

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004)

Please indicate how often the following 36 statements apply to you by writing the appropriate number from the scale above (1 – 5) in the box alongside each item.

| | 1<br>Almost never | 2<br>Sometimes | 3<br>About half the time | 4<br>Most of the time | 5<br>Almost always |
|---|---|---|---|---|---|
| I am clear about my feelings | | | | | |
| I pay attention to how I feel | | | | | |
| I have no idea how I am feeling | | | | | |
| I am attentive to my feelings | | | | | |
| I care about what I am feeling | | | | | |
| When I'm upset, I acknowledge my emotions | | | | | |
| When I'm upset, I become embarrassed for feeling that way | | | | | |
| I experience my emotions as overwhelming and out of control | | | | | |
| I have difficulty making sense out of my feelings | | | | | |
| I know exactly how I am feeling | | | | | |
| I am confused about how I feel | | | | | |
| When I'm upset, I become angry with myself for feeling that way | | | | | |
| When I'm upset, I have difficulty getting work done | | | | | |

| | 1<br>Almost never | 2<br>Sometimes | 3<br>About half the time | 4<br>Most of the time | 5<br>Almost always |
|---|---|---|---|---|---|
| When I'm upset, I become out of control | | | | | |
| When I'm upset, I believe that I will remain that way for a long time | | | | | |
| When I'm upset, I believe that I'll end up feeling very depressed | | | | | |
| When I'm upset, I believe that my feelings are valid and important | | | | | |
| When I'm upset, I have difficulty focusing on other things | | | | | |
| When I'm upset, I feel out of control | | | | | |
| When I'm upset, I can still get things done | | | | | |
| When I'm upset, I feel ashamed with myself for feeling that way | | | | | |
| When I'm upset, I know that I can find a way to eventually feel better | | | | | |
| When I'm upset, I feel like I am weak | | | | | |
| When I'm upset, I feel like I can remain in control of my behaviours | | | | | |
| When I'm upset, I feel guilty for feeling that way | | | | | |
| When I'm upset, I have difficulty concentrating | | | | | |
| When I'm upset, I have difficulty controlling my behaviours | | | | | |

| | 1<br>Almost never | 2<br>Sometimes | 3<br>About half the time | 4<br>Most of the time | 5<br>Almost always |
|---|---|---|---|---|---|
| When I'm upset, I believe that there is nothing I can do to make myself feel better | | | | | |
| When I'm upset, I start to feel very bad about myself | | | | | |
| When I'm upset, I lose control over my behaviours | | | | | |
| When I'm upset, I take time to figure out what I'm really feeling | | | | | |
| When I'm upset, my emotions feel overwhelming | | | | | |

# Appendix H

## UPPS-P Impulsivity Scale (Lynam et al., 2007)

Below are a number of statements that describe ways in which people act and think. For each statement, please indicate how much you agree or disagree with the statement. If you **Agree Strongly** circle **1**, if you **Agree Somewhat** circle **2**, if you **Disagree somewhat** circle **3**, and if you **Disagree Strongly** circle **4**. Be sure to indicate your agreement or disagreement for every statement below. Also, there are questions on the following pages.

| | Agree Strongly | Agree Some | Disagree Some | Disagree Strongly |
|---|---|---|---|---|
| 1. I have a reserved and cautious attitude toward life. | 1 | 2 | 3 | 4 |
| 2. I have trouble controlling my impulses. | 1 | 2 | 3 | 4 |
| 3. I generally seek new and exciting experiences and sensations. | 1 | 2 | 3 | 4 |
| 4. I generally like to see things through to the end. | 1 | 2 | 3 | 4 |
| 5. When I am very happy, I can't seem to stop myself from doing things that can have bad consequences. | 1 | 2 | 3 | 4 |
| 6. My thinking is usually careful and purposeful. | 1 | 2 | 3 | 4 |
| 7. I have trouble resisting my cravings (for food, cigarettes, etc.). | 1 | 2 | 3 | 4 |
| 8. I'll try anything once. | 1 | 2 | 3 | 4 |
| 9. I tend to give up easily. | 1 | 2 | 3 | 4 |
| 10. When I am in great mood, I tend to get into situations that could cause me problems. | 1 | 2 | 3 | 4 |
| 11. I am not one of those people who blurt out things without thinking. | 1 | 2 | 3 | 4 |
| 12. I often get involved in things I later wish I could get out of. | 1 | 2 | 3 | 4 |
| 13. I like sports and games in which you have to choose your next move very quickly. | 1 | 2 | 3 | 4 |
| 14. Unfinished tasks really bother me. | 1 | 2 | 3 | 4 |
| 15. When I am very happy, I tend to do things that may cause problems in my life. | 1 | 2 | 3 | 4 |
| 16. I like to stop and think things over before I do them. | 1 | 2 | 3 | 4 |
| 17. When I feel bad, I will often do things I later regret in order to make myself feel better now. | 1 | 2 | 3 | 4 |
| 18. I would enjoy water skiing. | 1 | 2 | 3 | 4 |
| 19. Once I get going on something I hate to stop. | 1 | 2 | 3 | 4 |
| 20. I tend to lose control when I am in a great mood. | 1 | 2 | 3 | 4 |
| 21. I don't like to start a project until I know exactly how to proceed. | 1 | 2 | 3 | 4 |

***Please go to the next page***

| | Agree Strongly | Agree Some | Disagree Some | Disagree Strongly |
|---|---|---|---|---|
| 22. Sometimes when I feel bad, I can't seem to stop what I am doing even though it is making me feel worse. | 1 | 2 | 3 | 4 |
| 23. I quite enjoy taking risks. | 1 | 2 | 3 | 4 |
| 24. I concentrate easily. | 1 | 2 | 3 | 4 |
| 25. When I am really ecstatic, I tend to get out of control. | 1 | 2 | 3 | 4 |
| 26. I would enjoy parachute jumping. | 1 | 2 | 3 | 4 |
| 27. I finish what I start. | 1 | 2 | 3 | 4 |
| 28. I tend to value and follow a rational, "sensible" approach to things. | 1 | 2 | 3 | 4 |
| 29. When I am upset I often act without thinking. | 1 | 2 | 3 | 4 |
| 30. Others would say I make bad choices when I am extremely happy about something. | 1 | 2 | 3 | 4 |
| 31. I welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional. | 1 | 2 | 3 | 4 |
| 32. I am able to pace myself so as to get things done on time. | 1 | 2 | 3 | 4 |
| 33. I usually make up my mind through careful reasoning. | 1 | 2 | 3 | 4 |
| 34. When I feel rejected, I will often say things that I later regret. | 1 | 2 | 3 | 4 |
| 35. Others are shocked or worried about the things I do when I am feeling very excited. | 1 | 2 | 3 | 4 |
| 36. I would like to learn to fly an airplane. | 1 | 2 | 3 | 4 |
| 37. I am a person who always gets the job done. | 1 | 2 | 3 | 4 |
| 38. I am a cautious person. | 1 | 2 | 3 | 4 |
| 39. It is hard for me to resist acting on my feelings. | 1 | 2 | 3 | 4 |
| 40. When I get really happy about something, I tend to do things that can have bad consequences. | 1 | 2 | 3 | 4 |
| 41. I sometimes like doing things that are a bit frightening. | 1 | 2 | 3 | 4 |
| 42. I almost always finish projects that I start. | 1 | 2 | 3 | 4 |
| 43. Before I get into a new situation I like to find out what to expect from it. | 1 | 2 | 3 | 4 |
| 44. I often make matters worse because I act without thinking when I am upset. | 1 | 2 | 3 | 4 |
| 45. When overjoyed, I feel like I can't stop myself from going overboard. | 1 | 2 | 3 | 4 |

***Please go to the next page***

| | Agree Strongly | Agree Some | Disagree Some | Disagree Strongly |
|---|---|---|---|---|

46. I would enjoy the sensation of skiing very fast down a high mountain slope.
47. Sometimes there are so many little things to be done that I just ignore them all.
48. I usually think carefully before doing anything.
49. When I am really excited, I tend not to think of the consequences of my actions.
50. In the heat of an argument, I will often say things that I later regret.
51. I would like to go scuba diving.
52. I tend to act without thinking when I am really excited.
53. I always keep my feelings under control.
54. When I am really happy, I often find myself in situations that I normally wouldn't be comfortable with.
55. Before making up my mind, I consider all the advantages and disadvantages.
56. I would enjoy fast driving.
57. When I am very happy, I feel like it is ok to give in to cravings or overindulge.
58. Sometimes I do impulsive things that I later regret.
59. I am surprised at the things I do while in a great mood.

# Appendix I

## Eating Disorder Examination Questionnaire 6.0 (EDE-Q; Fairburn & Belgin, 2008)

Instructions: The following questions are concerned with the past four weeks (28 days) only. Please read each question carefully. Please answer all the questions. Thank you. Questions 1 to 12: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days) only.

| On how many of the past 28 days have… | No days | 1-5 days | 6-12 days | 13-15 days | 16-22 days | 23-27 days | Every day |
|---|---|---|---|---|---|---|---|
| Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight (whether or not you have succeeded)? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you tried to exclude from your diet any foods that you like in order to influence your shape or weight (whether or not you have succeeded)? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you tried to follow definite rules regarding your eating (for example, a calorie limit) in order to influence your shape or weight (whether or not you have succeeded)? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you had a definite desire to have an empty stomach with the aim of influencing your shape or weight? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you had a definite desire to have a totally flat stomach? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Has thinking about food, eating or calories made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Has thinking about shape or weight made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you had a definite fear of losing control over eating? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you had a definite fear that you might gain weight? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you felt fat? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Have you had a strong desire to lose weight? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

Questions 13-18: Please fill in the appropriate number in the boxes on the right. Remember that the questions only refer to th past four weeks (28 days).

| | |
|---|---|
| Over the past 28 days, how many times have you eaten what other people would regards as an unusually large amount of food (given the circumstances)? | |
| ... On how many of these times did you have a sense of having lost control over your eating (at the time you were eating)? | |
| Over the past 28 days, on how many DAYS have such episodes of overeating occurred (i.e. you have eaten an unusually large amount of food and have had a sense of loss of control at the time)? | |
| Over the past 28 days, how many times have you made yourself sick (vomit) as a means of controlling your shape or weight? | |
| Over the past 28 days, how many times have you taken laxatives as a means of controlling your shape or weight? | |
| Over the past 28 days, how many times have you exercised in a “driven” or “compulsive” way as a means of controlling your weight, shape or amount of fat, or to burn off calories? | |

Questions 19 to 21: Please circle the appropriate number. Please note that for these questions the term “binge eating” means eating what others would regard as an unusually large amount of food for the circumstances, accompanied by a sense of having lost control over eating.

| | No days | 1-5 days | 6-12 days | 13-15 days | 16-22 days | 23-27 days | Every day |
|---|---|---|---|---|---|---|---|
| Over the past 28 days, on how many days have you eaten in secret (ie, furtively)? ... Do not count episodes of binge eating. | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| On what proportion of the times that you have eaten have you felt guilty (felt that you've done | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| wrong) because of its effect on your shape or weight? ... Do not count episodes of binge eating. | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Over the past 28 days, how concerned have you been about other people seeing you eat? ... Do not count episodes of binge eating. | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

Questions 22 to 28: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days).

| Over the past 28 days… | Not at all | Slightly | | Moderately | | Markedly | |
|---|---|---|---|---|---|---|---|
| Has your weight influenced how you think about (judge) yourself as a person? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Has your shape influenced how you think about (judge) yourself as a person? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| How much would it have upset you if you had been asked to weigh yourself once a week (no more, or less, often) for | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| the next four weeks? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| How dissatisfied have you been with your | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| weight? | | | | | | | |
| How dissatisfied have you been with your shape? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| How uncomfortable have you felt seeing your body (for example, seeing your shape in the mirror, in a shop window reflection, while undressing or taking a bath or shower)? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| How uncomfortable have you felt about others seeing your shape or figure (for example, in communal changing rooms, when swimming, or wearing tight clothes)? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

What is your weight at present? (Please give your best estimate.):

What is your height? (Please give your best estimate.):

If female:

Over the past three to four months have you missed any menstrual periods?: YES NO

If so, how many?:

Have you been taking the "pill"? YES NO

THANK YOU

Appendix J

Insomnia Severity Index (ISI; Bastien et al., 2001)

The Insomnia Severity Index has seven questions. The seven answers are added up to get a total score. When you have your total score, look at the 'Guidelines for Scoring/Interpretation' below to see where your sleep difficulty fits.

For each question, please CIRCLE the number that best describes your answer.

*Please rate the CURRENT (i.e. LAST 2 WEEKS) SEVERITY of your insomnia problem(s).*

| **Insomnia Problem** | **None** | **Mild** | **Moderate** | **Severe** | **Very Severe** |
|---|---|---|---|---|---|
| 1. Difficulty falling asleep | 0 | 1 | 2 | 3 | 4 |
| 2. Difficulty staying asleep | 0 | 1 | 2 | 3 | 4 |
| 3. Problems waking too early | 0 | 1 | 2 | 3 | 4 |

4. How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern?

| Very Satisfied | Satisfied | Moderately Satisfied | Dissatisfied | Very Dissatisfied |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

5. How NOTICEABLE to others do you think your sleep problem is in terms of impairing the quality of your life?

| Not at all Noticeable | A Little | Somewhat Much | Very Much | Noticeable |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

6. How WORRIED/DISTRESSED are you about your current sleep problem?

| Not at all Worried | A Little | Somewhat Much | Very Much | Worried |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?

| Not at all Interfering | A Little | Somewhat Much | Very Much | Interfering |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

**Guidelines for Scoring/Interpretation:**

Add the scores for all seven items (questions 1 + 2 + 3 + 4 + 5 +6 + 7) = _____ your total score

Total score categories: 0–7 = No clinically significant insomnia 8–14 = Subthreshold insomnia 15–21 = Clinical insomnia (moderate severity) 22–28 = Clinical insomnia (severe)