An Examination of the Relationship Between Insomnia Symptoms and Treatment Outcome in Binge Eating Disorder

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Abstract

Background: Sleep has been shown to be associated with treatment outcome in addictive disorders, mood disorders and mixed eating disorders samples. To date no research has examined the relationship between insomnia symptoms and treatment outcome in binge eating disorder (BED). **Method:** The present study was a secondary analysis that evaluated whether insomnia symptoms were associated with Dialectical Behaviour Therapy (DBT) self-help treatment outcome in a community sample of 71 participants with BED. Binge eating frequency, eating disorder psychopathology, insomnia symptoms, as well as anxious and depressive symptoms were assessed at pre- and post-treatment and at 3-month follow-up. **Results:** Results showed an improvement in insomnia symptoms from pre- to post-treatment and that baseline clinical levels of insomnia symptoms were associated with poorer treatment outcome at a trend level. **Conclusion:** Results are preliminary and additional research is needed to determine the prognostic significance of sleep in BED. Clinical implications and future directions are discussed.

Key words: binge eating disorder, insomnia symptoms, treatment outcome

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List of Abbreviations

Binge Eating Disorder	BED
Diagnostic and Statistical Manual of Mental Disorders – Fifth Edition	DSM-5
American Psychiatric Association	APA
Diagnostic and Statistical Manual of Mental Disorders	DSM
Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition	DSM-IV
Eating Disorders Not Otherwise Specified	EDNOS
Bulimia Nervosa	BN
Anorexia Nervosa	AN
Body Mass Index	BMI
Health-Related Quality of Life	HRQoL
Athens Insomnia Scale	AIS
Cognitive Behavioural Therapy	CBT
Interpersonal Psychotherapy	IPT
Dialectical Behavioural Therapy	DBT
Randomized Controlled Trial	RCT
Guided Self-Help	GSH
Unguided Self-Help	USH
Self-Esteem	SE
Pittsburgh Sleep Quality Index	PSQI
Eating Disorder Examination Interview	EDE-17
Diagnostic and Statistical Manual of Mental Disorders – Third Edition – Revised	DSM-III-R
Rapid Eye Movement	REM
Symptom Checklist 90	SCL-90
Eating Disorder Examination Questionnaire	EDE-Q
Alcohol Use Disorders Identification Test	AUDIT
Drug Abuse Screening Test - 10	DAST-10
Insomnia Severity Index	ISI
Brief Symptom Inventory	BSI
Analysis of Variance	ANOVA
Standard Deviation	SD

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1.0 Introduction

1.1 Binge Eating Disorder

Binge Eating Disorder (BED) is defined by the Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-5) as an eating disorder characterized by recurring episodes of binge eating (on average once per week for three months) without the regular use of inappropriate compensatory behaviours such as vomiting, laxative misuse or fasting (American Psychiatric Association [APA], 2013a). Binge eating refers to eating an amount of food that is considered to be much larger than most people would normally eat during the same period of time and under analogous circumstances (APA, 2013a). Individuals with BED report a loss of control over their eating behaviours and feel unable to stop overeating, causing significant distress (e.g., shame and depressed mood) and functional impairment (Allison, Spaeth, & Hopkins, 2016).

BED affects 4-5% of women and 2-3% of men in the general population (Hudson, Hiripi, Pope, & Kessler, 2007; Kessler et al., 2013), and 30% of individuals with obesity (De Zwaan, 2001). Although other eating disorders such as Anorexia Nervosa (AN) and Bulimia Nervosa (BN) are three times more prevalent in women than men, the gender ratio is far less skewed in BED (Hudson et al., 2007).

BED occurs across all types of body shapes and sizes; however, it is significantly more common among obese individuals (Hudson et al., 2007). Obesity is a serious public health problem with adverse effects on health and quality of life (Huang, Frangakis, & Wu, 2006; Kolotkin et al., 2006). Similarly, BED also has a significant impact on health and quality of life. Factors associated with a BED diagnosis (i.e., medical complications, depressed mood, impairments in social functioning) reveals that BED seriously impairs

quality of life (Ágh et al., 2015). Moreover, individuals diagnosed with BED score significantly lower on health-related quality of life (HRQoL) measures compared to those without BED (Mond, Hay, Rodgers, Owen, & Beaumont, 2005; Perez & Warren, 2012; Rieger, Wilfley, Stein, Marino, & Crow, 2005; Singleton, Kenny, Hallett, & Carter, 2019).

Along with being the most common eating disorder, BED is often chronic with the highest mean lifetime durations among the eating disorders of approximately eight to 14 years, followed by BN (five to eight years) and AN (one to six years; Hudson et al., 2007; Pope et al., 2006). The median age of onset for BED ranges from 18-21 years and can begin as early as adolescence (i.e. 10 to 13 years old) and as late as later adulthood (i.e., 55 years old; APA, 2013a; Hudson et al., 2007). Little is known about the etiology of BED, however, several risk factors for developing the disorder have been identified. These include adverse childhood experiences, parental psychiatric disorders, childhood obesity, parental obesity and negative self-evaluation of weight and shape, and eating behaviours in adolescence (Fairburn et al., 1998). Additionally, being perceived as overweight by parent(s) during childhood has been shown to be a strong prospective predictor of developing an eating disorder (e.g., binge eating and purging disorders) in both early adolescence (e.g., by 14 years of age) and late adolescence to young adulthood (e.g., 15 to 20 years of age; Allen, Byrne, & Crosby, 2015; Allen, Byrne, Forbes, & Oddy, 2009; Allen, Byrne, Oddy, Schmidt, & Crosby, 2014).

1.1.1 Mental health disorders and BED. BED often co-occurs with other psychological disorders, including major depressive disorder and anxiety disorders (Grucza, Przybeck, & Cloninger, 2007; Hudson et al., 2007; Javaras et al., 2008). A study

conducted by Grissett and Fitzgibbon (1996), compared 144 participants with obesity who reported binge eating (72 of whom met diagnostic criteria for BED) to 48 obese individuals who did not report binge eating, and found that those who engaged in binge eating reported significantly higher levels of depression compared to their non-bingeing counterparts. Furthermore, a comparison of the 72 individuals who met diagnostic criteria for BED and the 72 binge eaters who did not, found that severe BED – defined as more frequent binge episodes – was associated with higher levels of depression (Grissett & Fitzgibbon, 1996). Similar findings were revealed in a study conducted by Grucza et al. (2007), that support BED's association with symptoms of major depression. Other mental health disorders and mental health related issues also found to have a significant association with BED include generalized anxiety disorder, panic attacks and a history of one or more suicide attempts (Grucza et al., 2007). These findings are consistent with a recent study that investigated depressive symptoms and HRQoL in a sample of women diagnosed with BED (Grenon et al., 2010). Furthermore, individuals clinically diagnosed with BED or BN experience a similar frequency of Axis I comorbidities, including mood disorders, anxiety disorders and substance abuse disorders (Striegel-Moore et al., 2001). Grilo, White, and Masheb (2009) revealed that 40-70% of individuals with DSM-IV BED presented with at least one additional lifetime and/or current psychiatric disorder, where the most common disorders were mood disorders (54%), anxiety-related disorders (37%) and disorders involved with substance abuse (25%).

Research evaluating treatment outcome of mood disorders (Chambless, Tran, & Glass, 1997; Steketee, Chambless, & Tran, 2001; Watanabe et al., 2010) and substance use disorders (Bradizza, Stasiewicz, & Paas, 2006; Compton, Cottler, Jacobs, Ben-

Abdallah, & Spitznagel, 2003; Ouimette, Gima, Moos, & Finney, 1999) have revealed that psychiatric comorbidities are associated with and predict poorer treatment outcome. Specifically, increased negative affect (Masheb & Grilo, 2008) or increased depressive symptoms as measured by the Beck Depression Inventory (Deumens, Noorthoorn, & Verbraak, 2012) were significant predictors of poor treatment outcome or increased attrition in BED treatment trials. These findings suggest that implementing treatment strategies that target both BED and comorbid conditions may help improve treatment outcomes.

1.1.2 Obesity and BED. In addition to psychiatric comorbidity, BED is strongly associated with weight gain and obesity (De Zwaan, 2001; Mustelin, Bulik, Kaprio, & Keski-Rahkonen, 2017; Sonneville et al., 2013; Striegel-Moore et al., 2001). According to the World Health Organization, overweight and obesity are defined as the accumulation of an abnormal or excessive amount of adipose tissue and may be a risk factor for severe health consequences ("Obesity", 2019). Obesity occurs when our daily energy intake (e.g., food consumption) exceeds our energy expenditure (e.g., exercise). Specifically, excess energy is stored by the body in the form of fat, leading to weight gain (Sizer, Whitney, & Piché, 2012). Weight gain and obesity are commonly measured using body mass index (BMI), an indicator of body fat that is calculated by dividing an individual's weight in kilograms by their squared height in metres (Vorona et al., 2005). BMIs in the range of 25 to 29.9 are considered to be overweight, and obesity is officially defined as having a BMI greater than or equal to 30 (Sizer et al., 2012). A comparison in a sample of women found that obesity was more commonly associated with BED than either subtype of BN (purging or non-purging; Striegel-Moore et al., 2001).

Upon examination of the different components of the diagnostic criteria for BED, both the behavioural and cognitive components were strongly associated with BMI (Mustelin et al., 2017). The more features of BED participants reported experiencing (e.g., "I stuff myself with food," "I eat when I am upset," "I eat or drink in secrecy") the higher their mean BMI. This suggests that individuals with more severe cases of BED, tend to gain more weight (Mustelin et al., 2017).

Due to the adverse effects that BED has on an individual's HRQoL, it is crucial for individuals to receive effective treatment for this disorder. The following section will summarize different evidence-based treatment approaches that have been developed to address the diverse symptoms of BED.

1.2 Evidence-Based Treatment for BED

BED consists of cognitive, emotional and behavioural symptoms and it is important that psychological treatment addresses all of these areas (Grilo, 2017). The three main evidence-based psychological treatments for BED are: cognitive behavioural therapy (CBT), interpersonal psychotherapy (IPT) and dialectical behavioural therapy (DBT; Iacovino, Gredysa, Altman, & Wilfley, 2012). These three treatment approaches will now be briefly reviewed, including the conceptual model of BED on which the intervention is based and the research evidence for each type of treatment.

1.2.1 CBT and IPT for BED. Originally CBT was developed as a treatment model for BN (Fairburn, Marcus, & Wilson, 1993). Due to the effectiveness of CBT for BN and because binge eating is a core feature of both BN and BED, researchers were interested in the effectiveness of using CBT for treating BED (Fairburn et al., 1993; Grilo, 2017). According to Grilo (2017), CBT is the treatment for BED with the strongest

evidence base. CBT focuses on addressing distorted beliefs and attitudes that individuals with BED may have regarding eating, body weight and body shape as well as interrupting disturbed eating patterns (Fairburn, 2008; Wilson, Grilo, & Vitousek, 2007). CBT conceptualizes BED as a disorder that may develop among individuals who focus excessively on body weight and shape as determinants of their self-worth, and who are high in dietary restraint (Fairburn, 2008; Iacovino et al., 2012; Wilson et al., 2007; Wilson, Wilfley, Agras, & Bryson, 2010). This may then develop into a cycle of dieting and binge eating; in which, the CBT model is designed to break the "diet-binge" cycle (Iacovino et al., 2012).

The intervention process starts with identifying an individual's current eating behaviours which is followed by identifying triggers for binge eating, such as determining one's self-worth based on their weight, or stressful events that may lead to disordered eating (Fairburn, 2008; Wilson et al., 2007). Subsequently, methods such as cognitive restructuring to monitor and challenge negative thoughts about body weight/shape and eating are implemented with a goal of reducing binge eating and eventually establishing healthy eating behaviours (Fairburn, 2008; Iacovino et al., 2012). The final phase of CBT is to help an individual with BED develop methods that will aid in coping with future triggers and help reduce potential risks of relapse from occurring (Fairburn, 2008). As previously mentioned, the CBT model is designed to treat BED by breaking the "diet-binge" cycle that is commonly seen in BN (Fairburn et al., 1993; Grilo, 2017; Iacovino et al., 2012). However, individuals with BED do not commonly engage in extreme dietary restraint or restriction like individuals with BN (APA, 2013a). Therefore, the CBT model may not be as applicable to BED as it is to BN. In addition, only about half of individuals

with BED recover with CBT (Iacovino et al., 2012; Wilson et al., 2007; Wilson et al., 2010). This led researchers to investigate other psychological treatment approaches.

The second psychological intervention for BED that has received a significant amount of research attention is IPT. In contrast to CBT, IPT focuses on improving interpersonal functioning that contributes to BED (Ansell, Grilo, & White, 2012; Striegel-Moore, Fairburn, Wilfley, & Pike, 2005). Interpersonal functioning includes behaviours within four domains: interpersonal deficits, role conflicts, role transiting and grief and loss (Grilo, 2017). IPT is based on the idea that individuals are more inclined to rely on unhealthy coping mechanisms such as binge eating if they cannot effectively express negative emotions, possess poor interpersonal functioning skills and are surrounded by negative social environments (Rieger et al., 2010). The process of IPT is used to teach individuals with BED how to identify and cope with their negative social environments, to effectively express their feelings and address future conflicts without using unhealthy eating behaviours as coping mechanisms (Rieger et al., 2010). IPT seeks to reduce binge eating pathology by identifying and improving interpersonal skills, enhance psychosocial functioning and promote positive self-image (Grilo, 2017; Wolfe, Baker, Smith, & Kelly-Weeder, 2009).

Overall, the effectiveness of CBT and IPT for BED has been found to be moderate, resulting in remission rates from binge eating of about 50% at the end of treatment (Iacovino et al., 2012; Wilson et al., 2007). At follow-up, both CBT and IPT produced reliable long-term reductions in binge eating, with a 50-60% remission rate maintained one to three years after completing the intervention programs (Hilbert et al., 2012; Wilfley et al., 2002). However, this means that about 50% of individuals do not

recover from BED with CBT or IPT. Subsequently, researchers examined whether DBT might be of help for BED.

1.2.2 DBT for BED. DBT is a form of CBT based on the affect regulation model of binge eating. This model argues that binge eating is a maladaptive coping strategy to modulate intense emotions among individuals who have not developed healthy emotion regulation strategies (Wiser & Telch, 1999). DBT was originally developed by Marsha Linehan as group skills training and individual psychotherapy and is considered the gold standard treatment for Borderline Personality Disorder (Linehan, 1987; Linehan, 1998). It was later modified for BED with a focus on DBT group skills training only (Wiser & Telch, 1999). While binge eating behaviour provides short-term relief from intense emotions, feelings of shame and guilt tend to occur following binge episodes, thereby creating further emotion dysregulation (Allison et al., 2016; Safer, Adler, & Masson 2018). Therefore, the primary goal of DBT is to help individuals with BED develop adaptive skills for regulating their emotions instead of binge eating. Specifically, the DBT approach consists of helping clients develop skills in four areas: mindfulness, distress tolerance, emotion regulation and interpersonal effectiveness (Grilo, 2017; Iacovino et al., 2012). DBT is thought to facilitate the development of more adaptive emotion regulation skills that can be incorporated into their daily life to reduce an individual's reliance on binge eating as a coping mechanism (Telch, Agras, & Linehan, 2001).

To date, five randomized controlled trials (RCT) have been conducted to evaluate the efficacy of DBT for BED (Carter, Kenny, Singleton, Van Wijk, & Heath, 2019; Masson, von Ranson, Wallace, & Safer, 2013; Rahmani, Omidi, Asemi, & Akbari, 2018; Safer, Robinson, & Jo, 2010; Telch et al., 2001). Three of the RCT's evaluated the

efficacy of DBT by comparing group DBT to an active comparison group therapy condition (Safer et al., 2010) or a waitlist control group (Rahmani et al., 2018; Telch et al., 2001). The findings revealed that group DBT resulted in BED remission rates of 64-89% (Rahmani et al., 2018; Safer et al., 2010; Telch et al., 2001). The long-term effectiveness of the group therapy DBT was established with a 56-65% remission rate at six-month follow-up (Safer et al., 2010; Telch et al., 2001) and a 60% remission rate at the 12-month follow-up (Safer et al., 2010). Furthermore, Safer et al. (2010) found that the group DBT condition achieved post-treatment reductions in binge eating more quickly than the active comparison group and that the DBT condition had a significantly lower dropout rate.

The fourth RCT evaluated the efficacy of guided self-help DBT treatment for BED compared to a waitlist control group (Masson et al., 2013). Self-help DBT consists of DBT skills training conducted individually via a self-help manual. One of the main advantages of self-help DBT is that it is easier to disseminate than group or individual DBT administered by a trained professional. With guided self-help DBT, participants are provided with a self-help manual and are asked to complete a number of sessions with a guided self-help therapist via a form of communication (i.e., telephone or videoconferencing). The guided self-help therapist is provided as a form of support to help the participant complete the program. Results revealed that the guided self-help DBT group had a significantly higher remission rate than the control condition (40% versus 3.3% for the waitlist control group; Masson et al., 2013). At six-month follow-up, the guided self-help group reported significant decreases in eating disorder psychopathology and a remission rate of 30% (Masson et al., 2013). Although this study revealed

beneficial findings for a DBT guided self-help program, the study also had certain limitations. For instance, the trial did not include an active comparison control group, rather it only used a waitlist control group which does not control for factors like attention and expecting to improve. Another limitation of the study was the small sample size (n=60), which likely contributed to a lack of power when evaluating predictors of treatment outcome (Masson et al., 2013). In addition, the study did not include an unguided self-help condition. Finally, the study design only included a six-month follow-up assessment and was not able to capture longer-term outcomes of participants who engaged in the intervention (Masson et al., 2013).

Recently, Carter and colleagues (2019) conducted an RCT of a self-help DBT skills training program for BED that was designed to improve upon some of the limitations of the Masson et al. (2013) study by including an active comparison condition and an unguided self-help intervention condition. A sample of 71 participants diagnosed with BED, based on answers provided on the Eating Disorder Examination Interview (EDE-17; Fairburn, Cooper, & O'Connor, 2014) were randomized to one of three conditions: DBT guided self-help (DBT-GSH), DBT unguided self-help (DBT-USH) or a self-esteem unguided self-help (active comparison) group (SE-USH) that did not address binge eating (Carter et al., 2019). Participants completed a series of self-report questionnaires at four time points (baseline, eight-week mid-treatment, 12-week post-treatment and 24-week follow-up) and completed the EDE-17 interview to assess changes in binge frequency at baseline (screening), post-treatment and follow-up (Carter et al., 2019). Across all three conditions, participants significantly decreased in binge eating frequency and eating disorder symptomology from baseline to post-treatment, with mean

percentage reduction rates ranging from 44% (DBT-USH) to 74% (DBT-GSH) at 12-weeks post-treatment (Carter et al., 2019). This reduction was maintained from post-treatment to follow-up (Carter et al., 2019). Furthermore, the mean reduction rates from baseline to three-month follow-up ranged from 59% (SE-USH) to 63% (DBT-USH; Carter et al., 2019).

Although there are evidence-based treatments for BED (e.g., CBT, IPT, DBT), the remission rates are approximately 50-60%. Thus, a significant proportion of individuals do not recover fully with the developed treatment regimens. It is pertinent for researchers to investigate factors associated with treatment outcome and ways of improving overall treatment outcome, one of which is the presence of comorbidities (Deumens et al., 2012; Masheb & Grilo, 2008). One such comorbidity which has yet to be studied in BED is insomnia symptoms. If symptoms of insomnia are associated with treatment outcome, then that could represent one potential avenue for improving treatment of BED.

1.3 Eating Behaviours and Sleep

1.3.1 Consequences of short sleep duration. There is a strong association between short sleep duration – defined as sleeping six hours or less per night – and an increased risk of developing chronic illnesses (i.e., type II diabetes, hypertension and coronary heart disease; Ayas et al., 2003; Gangwisch et al., 2006; Yaggi, Araujo, & McKinlay, 2006). In addition, short sleep duration can also affect other health-related behaviours, including eating behaviour (Ayas et al., 2003; Gangwisch et al., 2006; Schmid, Hallschmid, Jauch-Chara, Born, & Schultes, 2008; Taheri, Lin, Austin, Young, & Mignot, 2004; Yaggi et al., 2006). This may be related to the development of disordered eating behaviours, particularly food overconsumption.

Insomnia, sometimes accompanied by short sleep duration (Fernandez-Mendoza, 2017), is a sleep-wake disorder where a person reports a dissatisfaction with their sleep quality or quantity and experiences difficulty maintaining or initiating sleep and/or early-morning awakenings (APA, 2013b). Symptoms of insomnia are assessed based on individuals' self-reported sleep continuity (e.g. latency to fall asleep or time spent awake during the night) rather than biological measures such as polysomnography (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). Moreover, insomnia requires that the dissatisfaction with sleep must be accompanied by clinical distress and impairment of everyday functions, such as mood and energy levels (APA, 2013b).

Sleep is instrumental in the regulation of biological systems including endocrine regulation and the production of hormones related to eating behaviours and weight gain (i.e., leptin and ghrelin; Ayas et al., 2003; Gangwisch et al., 2006; Schmid et al., 2008; Taheri et al., 2004; Yaggi et al., 2006). Ghrelin is a hormone that signals hunger and is secreted by the stomach to induce the desire for food consumption (Carlson, 2013). Its counterpart, leptin is released from adipose tissue and secreted when food is consumed, signaling satiation (Carlson, 2013). Thus, the two hormones work together to regulate food consumption. One night of short sleep duration (4.5 hours) or one night of total sleep deprivation is significantly related to increased ghrelin levels and increased feelings of hunger the next day, compared to more balanced levels among individuals with regular sleep duration (seven hours; Schmid et al., 2008). In healthy male adults, restricted sleep duration (defined as four hours of sleep) is associated with a 28% increase in circulating ghrelin hormone and a 18% decrease in leptin levels the next day (Spiegel, Tasali, Penev, & Van Cauter, 2004). Moreover, short sleep duration was also significantly associated

with increased hunger and appetite, particularly for calorie-dense and high carbohydrate foods (Spiegel et al., 2004). Similarly, an overnight study using the data collected for the Wisconsin Sleep Cohort Study (Young, 2009) showed that five or less hours of sleep predicted 14.9% higher ghrelin levels and 15.5% lower leptin levels the next morning, compared to eight hours of sleep (Taheri et al., 2004).

Another study examined endocrine regulation among a sample of 14 men who met DSM-IV diagnostic criteria for primary insomnia and an age and body-weight matched sample of 24 healthy males (Motivala, Tomiyama, Ziegler, Khandrika, & Irwin, 2009). Diagnosis of primary insomnia was determined by the administration of the structured sleep disorders interview and the structured clinical interview for DSM-III-R/DSM-IV (Motivala et al., 2009). Based on the results from polysomnography (to measure sleep), participants diagnosed with insomnia experienced significantly less total sleep time (four to six hours) and sleep efficiency compared to the seven hours of sleep in healthy controls (Motivala et al., 2009). Those with insomnia also experienced significantly lower levels of ghrelin throughout the night compared to the controls, which was found to be negatively correlated with the amount of stage one sleep (Motivala et al., 2009). A similar association was found by Dzaja and colleagues (2004), revealing decreased nocturnal ghrelin levels in healthy adults who underwent experimental sleep deprivation. These findings support the relationship between short sleep duration in both experimental sleep deprivation or natural insomnia and ghrelin dysregulation, which may relate to weight gain and obesity in this population.

Not only is short sleep duration associated with appetite and the amount of food consumed but also with the type of food consumed. Both natural short sleep duration

(Imaki, Hatanaka, Ogawa, Yoshida, & Tanada, 2002) and experimentally induced short sleep duration (Nedeltcheva et al., 2009) have been repeatedly associated with more frequent eating by displaying an increase in snacking behaviours in-between meals, as well as increased consumption of calorie-dense foods rather than nutrient dense foods. Similarly, clinical symptoms of insomnia, as measured by the Athens Insomnia Scale (AIS; Soldatos, Dikeos, & Paparrigopoulos, 2000), was strongly associated with increased snacking behaviours in a sample of 361 obese participants, suggesting that insomnia may be associated with weight gain (Wrzosek, Wojnar, Sawicka, Talalaj, & Nowicka, 2018). However, the exact sleep duration of participants with clinical levels of insomnia symptoms was not reported. Due to the influence of short sleep duration on hormone regulation and its impact on the type of food consumed, findings from previous research suggest that sleep loss may increase food consumption among male and female adults, thereby contributing to weight gain (Bosy-Westphal et al., 2008; Brondel, Romer, Nougues, Touyarou, & Davenne, 2010).

1.3.2 Obesity and short sleep duration. Findings on the metabolic alterations associated with short sleep duration and increased appetite stimulated research evaluating the link between short sleep duration and the development of obesity (Taheri, 2006). Cappuccio and colleagues (2008), conducted a meta-analysis of 30 cross-sectional studies (12 with children and 18 with adults) on short-term sleep duration and its relationship to obesity. The systematic review revealed that obese children and adults had an increased risk (55-90%) of experiencing short sleep duration compared to non-obese participants (Cappuccio et al., 2008).

Primary care patients who were overweight and obese also reported significantly less sleep time than patients with a BMI in the normal range (Vorona et al., 2005). Similarly, in a 13-year prospective study, cross-sectional associations were found between restricted sleep (i.e., sleeping less than six hours per night) and increased BMI, after controlling for various confounding variables (i.e., family history of weight gain, amount of physical activity; Hasler et al., 2004). Thus, converging research provides evidence of an association between short sleep-duration, increased appetite, and increased food-consumption resulting in weight gain. Therefore, it is possible that short-sleep duration may be associated with BED since this disorder is associated with overeating and weight gain (De Zwaan, 2001).

1.3.3 BED and sleep. As previously discussed, BED is commonly accompanied by weight gain and obesity (De Zwaan, 2001; Sonneville et al., 2013; Striegel-Moore et al., 2001). Short sleep duration has been shown to disrupt endocrine regulation (e.g., ghrelin and leptin hormones) which increases appetite and hunger, leading to increased food consumption and subsequent weight gain and obesity. Therefore, among individuals with BED, it is likely that short sleep duration may exacerbate their binge eating symptoms. Another way that short sleep duration may worsen symptoms of BED is that it allows for increased hours awake and therefore more awake time to engage in binge eating. BED is commonly accompanied by preoccupation with thoughts of food (Davis et al., 2007). Therefore, in comorbid cases of BED and short sleep insomnia, individuals are awake more hours of the day leading to more opportunity to obsess over food related thoughts and cravings.

Insomnia is a comorbid diagnosis in 25-30% of individuals with eating disorders such as BED (Aspen et al., 2014). To date, there have only been three cross-sectional studies that have examined sleep difficulties in a clinical sample of BED. In the first, Tzinschinsky, Latzer, Epstein, and Tov (2000) evaluated sleep characteristics in a sample of 18 obese females with BED, 13 obese females without BED and 16 women without BED in the normal weight range. Objective sleep measures included actigraphy and subjective sleep measures included self-report questionnaires (e.g., Mini-Sleep Questionnaire; Zomer, Peled, Rubin, & Lavie, 1985, Standard Technion Clinical Sleep Questionnaire; Lavie, Kremerman, & Wiel, 1982) and sleep diaries (Tzinschinsky et al., 2000). Obese BED and obese non-BED individuals reported significantly more sleep awakenings, lower sleep efficiency and increased sleep disturbances compared to the normal weight controls (Tzinschinsky et al., 2000). However, there were no significant differences between obese individuals with or without BED (Tzinschinsky et al., 2000).

In the second study, Vardar, Caliyurt, Arikan, and Tugly (2004) compared sleep quality in obese binge eaters and non-binge eaters. A sample of 36 treatment-seeking obese individuals, eight of whom met criteria for BED based on answers to a structured interview, were compared to a sample of 37 individuals of normal weight (Vardar et al., 2004). Obese individuals with BED reported significantly poorer sleep quality and increased sleep latency as measured by the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) compared to obese individuals without BED and normal weight controls (Vardar et al., 2004).

In the third study, Kenny, Van Wijk, Singleton, and Carter (2018) found that, after controlling for BMI, individuals diagnosed with BED reported significantly more

symptoms of insomnia, as measured by the Insomnia Severity Index (ISI; Bastien, Vallieres, & Morin, 2001), compared to individuals with no history of an eating disorder. Additionally, insomnia symptom severity was a predictor of binge frequency, demonstrating that more severe symptoms of insomnia are associated with more frequent binge episodes (Kenny et al., 2018). The relationship between insomnia symptom severity and BED diagnosis was partially mediated by anxiety and fully mediated by depression (Kenny et al., 2018). Seeing that insomnia is highly co-morbid with mood and anxiety disorders, it is important to consider these symptoms when looking at the development, maintenance, and treatment of BED. The next section discusses research on the association between symptoms of insomnia and treatment outcome in BED and related disorders.

1.4 Insomnia Symptoms and Treatment Outcome

Brower and Perron (2010) conducted a systematic review on sleep disturbance as a risk factor for relapse in substance use disorders. Studies on alcohol dependence showed that increased sleep latency or increased Rapid Eye Movement (REM) sleep are predictors of relapse following treatment for alcohol dependence (Brower, Aldrich, & Hall, 1998). A more recent study revealed that short-sleep insomnia (4.5 to six hours) predicted an increased risk of alcohol consumption during treatment and increased risk of relapse at post-treatment (Conroy et al., 2006). Although this study focused on alcohol dependence, the findings revealed by Brower et al. (1998) may also be relevant to individuals diagnosed with BED. Davis and Carter (2014) have suggested that BED can be conceptualized from an addiction viewpoint, otherwise known as the ‘food addiction’ model of BED. There are several parallels between overeating and substance addiction

disorders (Davis & Carter, 2014). Based on the addiction model of BED and the findings revealed by Brower et al. (1998), it can be suggested that individuals with BED who also experience sleep disturbance, may be at an increased risk of poorer BED treatment outcomes including increased risk of binge eating relapse.

The relationship between insomnia symptoms and treatment outcome has been investigated in the mood disorder literature (Dombrovski et al., 2007; Manber et al., 2008; Troxel el at., 2012). Insomnia was associated with worse treatment outcome for depression and a higher risk of relapse at follow-up (Dombrovski et al., 2007; Manber et al., 2008; Troxel el at., 2012). Given the high degree of comorbidity between mood disorders and eating disorders (Grucza et al., 2007; Hudson et al., 2007; Javaras et al., 2008), insomnia symptoms may similarly be associated with poorer treatment outcome for BED.

1.4.1 Insomnia symptoms and treatment outcome for eating disorders. To date, no studies have examined the relationship between insomnia and treatment outcome for BED. However, there have been two studies that evaluated the association between sleep and treatment outcome in eating disorders (Lombardo, Battagliese, Venezia, & Salvemini, 2015; Sauchelli et al., 2016). In one study, archival research conducted by Lombardo and colleagues (2015) examined clinical files collected from 1995 to 2013 of 562 female eating disorder patients receiving outpatient treatment. Researchers examined whether poor sleep– as measured by the sleep subscales of a self-report questionnaire, the Symptom Checklist 90 (SCL-90; Derogatis, Lipman, & Covi, 1973) – at baseline and after treatment, predicted the severity of eating disorder symptomology in a mixed sample of eating disorder patients (e.g., AN, BN, BED, EDNOS; Lombardo et al., 2015), 39% of

which were BED patients. Poor sleep, as measured by the sleep scale of the SCL-90, at baseline predicted an increase in severity of eating disorder symptoms six months after treatment and this relationship was mediated by depression (Lombardo et al., 2015). Furthermore, results showed that persistent poor sleep, as measured by the sleep scale of the SCL-90 at six months post-treatment predicted the severity of eating disorder symptomology at post-treatment after controlling for age and this relationship was also mediated by depression (Lombardo et al., 2015). These findings suggest that chronic sleep problems as well as depression may be related to treatment response among individuals with eating disorders. It has yet to be investigated whether treatment of sleep problems during treatment for an eating disorder leads to improved treatment outcomes.

Results from the study conducted by Lombardo and colleagues (2015) identified a relationship between poor sleep and poor treatment outcome for eating disorders. However, there were some limitations to this study. First, because the study used data extracted from clinical files rather than the community, the findings may not be generalizable to the general public (Lombardo et al., 2015). Second, the researchers did not use the best available measures to assess severity of eating disorders (Eating Disorder Inventory [EDI]; Garner, 1991) and sleep quality (SCL-90; Lombardo et al., 2015) which limits their results. For example, the sleep subscale of the SCL-90 administered to measure sleep did not address sleep duration, and as a result, it is unclear whether short-sleep duration may be associated with these findings.

1.5 Rationale for Current Study and Study Objectives

The current study was a secondary analysis of data from a recent randomized controlled trial (RCT) that compared DBT guided self-help (DBT-GSH), DBT unguided

self-help (DBT-USH) and an active control condition (SE) for BED (Carter et al., 2019). The goal was to examine the relationship between insomnia symptoms and DBT treatment outcome in BED by addressing four research objectives:

1. To examine whether at least mild or clinical levels of insomnia symptoms at baseline (i.e., pre-treatment) predicted (a) changes in binge eating frequency and (b) changes in global eating disorder psychopathology from pre- to post-treatment in the combined DBT conditions only (i.e., DBT-GSH and DBT-USH), while controlling for relevant covariates. It was hypothesized that higher insomnia symptom severity at baseline would be associated with less improvement in binge eating frequency and eating disorder symptomology (i.e., EDE-Q Global scores) from baseline to post-treatment in the DBT self-help conditions.

2. To examine whether insomnia symptom severity improved across the three time points (i.e., baseline, post-treatment and follow-up) in both the DBT and SE conditions. Given the positive association found between BED and insomnia symptoms at baseline (Kenny et al., 2018) and the reductions in binge eating frequency from baseline to post-treatment (Carter et al., 2019), it was hypothesized that there would be a significant improvement in insomnia symptoms from pre- to post-treatment.

3. To determine whether change in at least mild or clinical levels of insomnia symptoms from pre-treatment to post-treatment predicted improvement in (a) binge eating frequency and (b) eating disorder psychopathology, while controlling for relevant covariates in the combined DBT conditions. Based on previous literature revealing an association between insomnia symptoms and poorer treatment outcome in other mental disorders, it was hypothesized that less change in insomnia symptom severity (decreased

ISI total scores) from pre-treatment to post-treatment would predict less improvement in binge frequency and eating disorder psychopathology from baseline to post-treatment.

4. To evaluate whether individuals in the DBT and SE conditions who were in remission from binge eating at post-treatment (i.e., no episodes of binge eating over the previous 28 days) reported significantly different levels of insomnia symptom severity at baseline compared to individuals who were not in remission at post-treatment. Based on the positive association between insomnia symptoms and binge eating frequency at baseline (Kenny et al., 2018), it was hypothesized that individuals who were abstinent from binge eating at post-treatment would report lower ISI scores (less severe symptoms of insomnia) at baseline compared to individuals who were not abstinent from binge eating at post-treatment.

2.0 Method

The current study was a secondary analysis of data from a recent randomized controlled trial (RCT; Carter et al., 2019) that evaluated the effectiveness of a Dialectical Behaviour Therapy (DBT) self-help program for BED (Carter et al., 2019). The results of the trial indicated that binge frequency and eating disorder symptomology improved significantly from baseline to post-treatment and these changes were maintained from post-treatment to three-month follow-up in the three DBT-GSH, DBT-USH and SE-USH conditions.

2.1 Ethical Approval

This project received ethical approval from the institutional Health Research Ethics Board.

2.2 Participants

There were 71 participants between the ages of 19-65 years who took part in the RCT. They were recruited from the community across the province of Newfoundland and Labrador via posters and brochures posted in universities, hospitals, public buildings (e.g. coffee shops), and local doctor's offices. Other forms of recruitment included local radio stations and advertisements in rural church bulletins.

2.2.1 Inclusion criteria for the trial. Participants were male and female, aged 19-65 years who met the DSM-5 diagnostic criteria for BED, which was determined via the Eating Disorder Interview (EDE-17; Fairburn et al., 2014). Additional inclusion criteria consisted of: (1) minimum BMI of 18.5; (2) able to read English; (3) high school graduate or equivalent; and (4) access to a computer or tablet with Wi-Fi (because guided self-help was delivered via videoconferencing in the trial). Individuals on a stable dose of antidepressant medication and/or sleep medication for at least three months were eligible to take part in the RCT. Interested participants were directed to a website and were asked to complete a series of screening questionnaires via Qualtrics. The screening measures included: (1) a demographics questionnaire, (2) the SCOFF questionnaire (Luck et al., 2002) to screen for eating disorder psychopathology with a cutoff score of three or more yes responses, (3) the Alcohol Use Disorders Identification Test (AUDIT; Reinert & Allen, 2002) and the Drug Abuse Screening Test version 10 (DAST-10; Maisto, Carey, Carey, Gordon, & Gleason, 2000) to screen for symptoms of substance abuse.

2.2.2 Exclusion criteria for the trial. Potential participants were excluded from participating in the trial for the following reasons: (1) current psychological treatment for BED; (2) major medical illness known to influence eating behavior that could interfere

with treatment (e.g., cancer, hypothyroidism, Type II diabetes); (3) current pregnancy; (4) exceeding a cut-off of five on the DAST (Maisto et al., 2000) or 16 on the AUDIT (Reinert & Allen, 2002) screening questionnaires indicating possible substance use disorder; and (5) methylphenidate (e.g., Ritalin) or stimulant use (excluding caffeine use).

2.2.3 Recruitment for the RCT. In the larger RCT, 558 individuals responded to the recruitment procedures and completed a screening questionnaire to test for the initial eligibility criteria (Carter et al., 2019). Of these, a total of 175 individuals were eligible and completed a telephone interview to confirm the diagnosis of BED. Ninety-nine of these individuals were not eligible to take part in the study, mainly because they did not meet DSM-5 criteria for BED based on EDE interview. The remaining 76 individuals met inclusion criteria and were invited to take part in the trial. Recruitment and screening procedures took place from October 2016 to July 2017. A total of 74 individuals accepted the invitations and were enrolled in the larger trial. Of note, following the completion of the baseline questionnaires it was determined that three of the participants had to be excluded from the final sample for ineligibility reasons such as diagnoses of Type II diabetes, pregnancy, and a reading disability yielding a final sample of 71 participants.

2.3 Procedure

Participants who met the initial inclusion criteria for the trial – as indicated by their responses on the screening survey – were contacted by a researcher and asked to complete a telephone interview to confirm the diagnosis of BED using the EDE-17. Once the confirmation of a BED diagnosis based on the DSM-5 diagnostic criteria was made, participants were randomized to one of three conditions for 12 weeks: (1) DBT unguided self-help (DBT-USH), (2) DBT guided self-help (DBT-GSH) or (3) Self-Esteem

unguided self-help (SE-USH; active comparison group). In the DBT conditions participants were given a new DBT self-help manual for BED (Safer et al., 2018) not yet published. The manual focused on developing skills in four different modules to aid in development and improvement of coping mechanisms other than binge eating. Participants in the unguided DBT condition were directed to work through the manual on their own, whereas, the guided DBT condition were assigned to meet with a trained therapist (e.g., one of three psychology graduate students) via videoconference for six sessions. The active control condition involved a self-help program that focused on self-esteem (SE) but did not address binge eating. None of the treatments addressed insomnia symptoms.

2.4 Assessment Measures

In the trial, assessments took place at: baseline (week 0), post-treatment (week 12), and three-month follow-up (week 24). At each assessment, the following measures were given: (1) the EDE-17 interview (Fairburn et al., 2014) to assess binge frequency, (2) the Eating Disorder Examination – Questionnaire (EDE-Q; Fairburn, 2008) to assess other aspects of eating disorder psychopathology, (3) the ISI (Bastien et al., 2001) to assess severity of insomnia symptoms; and (4) the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983) to assess psychological distress and symptoms of psychological disorders. Additional measures were included in the trial; however, these measures were not part of the current research study and will not be reported here.

2.4.1 Eating Disorder Examination Interview (Version 17; EDE-17). (Fairburn et al., 2014). The EDE-17 is an investigator-based structured interview that is designed in order to make eating disorder diagnoses based on DSM-5 criteria (see Appendix B). It

examines the frequency and severity of symptoms over the past three months. The trial used a brief version of the EDE-17, consisting only of the items necessary for a diagnosis of BED. The EDE-17 demonstrates good test-retest and interrater reliability along with adequate internal consistency (Beaumont, Kopec-Schrader, Talbot, & Touyz, 1993; Byrne, Allen, Lampard, Dove, & Fursland, 2010; Cooper, Cooper, & Fairburn, 1989; Cooper & Fairburn, 1987; Grilo et al., 2010; Grilo, Masheb, Lozano-Blanco, & Barry, 2004; Rizvi, Peterson, Crow, & Agras, 2000). Furthermore, this measure has established good criterion and convergent validity (Barnes, Masheb, White, & Grilo, 2011; Cooper et al., 1989; Loeb, Pike, Walsh, & Wilson, 1994; Wilfley, Schwartz, Spurrell, & Fairburn, 2000; Wilson & Smith, 1989). Frequency of binge eating at baseline, post-treatment and follow-up was measured using the EDE interview because the interview has been shown to be a more accurate measure of binge eating frequency than the questionnaire (Berg, Peterson, Frazier, & Crow, 2012).

2.4.2 Eating Disorder Examination – Questionnaire 6.0 (EDE-Q 6.0). (Fairburn, 2008). The EDE-Q 6.0 is a 36-item self-report questionnaire that was based on the original EDE interview which demonstrates good test-retest reliability and internal consistency (Berg et al., 2012). The EDE-Q is a measure used to assess eating disorder psychopathology (Fairburn, 2008) and differentiates between individuals with and without an eating disorder (Elder et al., 2006; Engelsen & Laberg, 2001). It demonstrates good test-retest reliability, internal consistency and temporal stability (Bardone-Cone & Boyd, 2007; Luce & Crowther, 1999; Mond, Hay, Rodgers, Owen, & Beaumont, 2004; Peterson et al., 2007; Reas, Grilo, & Masheb, 2006).

The current study used a modified seven-item, three-factor version of the EDE-Q (see Appendix C for items; Grilo et al., 2010; Grilo, Reas, Hopwood, & Crosby, 2015). This version of the EDE-Q consists of three-subscales: 1) Dietary Restraint (e.g., Have you gone eight hours or more without eating any food as a method to influence your shape/weight?), 2) Body Dissatisfaction (e.g., How dissatisfied have you been with your shape and weight?), and 3) Overvaluation of shape and weight (e.g., Has your shape/weight influenced how you think about yourself as a person?; Grilo et al., 2015). A Global score is calculated by taking the average of the three subscale scores. The item scores range from 0 (not at all) to 6 (extremely). The higher the scores across all three subscales, the greater the eating disorder pathology. Berg and colleagues (2012) have established that the EDE-Q has good test-retest reliability and internal consistency. In the current study, internal consistency was good for the Global (*Cronbach's alpha* = .84), Dietary Restraint (*Cronbach's alpha* = .89), and Body Dissatisfaction (*Cronbach's alpha* = .89) scales, and excellent for the Overvaluation scale (*Cronbach's alpha* = .97).

2.4.3 Insomnia Severity Index (ISI). (Bastien et al., 2001). The ISI assesses the severity of insomnia symptoms (Bastien et al., 2001). The items focus on both subjective symptoms (e.g., difficulty initiating or maintaining sleep) as well as the consequences of insomnia symptoms on one's daily functioning (e.g., daytime fatigue, mood, concentration, memory, etc.). The ISI asks participants to report their sleeping patterns over the previous two weeks and contains seven items assessing: (1) difficulty of sleep onset, (2) difficulty of staying asleep, (3) problems associated with early morning awakening, (4) satisfaction with current sleep patterns, (5) noticeability of the sleep problems to others, (6) level of distress or concern associated with the sleep problems,

and (7) level of interference of the sleep problems with daily functioning. All items were scored on a five-point likert scale ranging from 0 “not at all” to 4 “very much/extremely” (see Appendix D). The total ISI score was used to determine the severity of insomnia symptoms. Established clinical cutoffs are: 0-7 no clinically significant insomnia symptoms; 8-14 subthreshold insomnia symptoms; 15-21 clinical (moderate) insomnia symptoms; and 22-28 clinical (severe) insomnia symptoms (Bastien et al., 2001). The ISI has adequate internal consistency and test-retest reliability. The researchers also found that the measure is sensitive to changes in perception of sleep difficulties during treatment (Bastien et al., 2001). In the current study, internal consistency was very good (*Cronbach’s alpha*= .90).

2.4.4 Brief Symptom Inventory (BSI). (Derogatis & Melisaratos, 1983). The BSI is a 53-item self-report questionnaire that assesses nine clinically relevant psychological symptoms: (1) somatization; (2) obsessive-compulsive; (3) interpersonal sensitivity; (4) depression; (5) anxiety; (6) hostility; (7) phobic anxiety; (8) paranoid ideation; and (9) psychoticism (see Appendix E for items). The questionnaire measures how often individuals experience various symptoms in the past week as well as the intensity of the symptoms. Items are scored on a five-point scale ranging from 0 “not at all” to 4 “extremely”. According to Derogatis and Melisaratos (1983), the BSI has established very good test-retest reliability and convergent and construct validity. The current study only used the BSI depression (Items #9, 16, 17, 18, 35, and 50) and anxiety (Items #1, 12, 19, 38, 45 and 49) subscale scores as a measure of depression and anxiety symptomology. In the present study, internal consistency was good for both the anxiety (*Cronbach’s alpha* = .87) and depression (*Cronbach’s alpha* = .88) subscales.

2.5 Data Analyses

Data were analyzed using IBM SPSS Software Version 24 and significance was determined at $p < .05$. The dataset from the RCT used in the current study involved intention-to-treat analysis (Carter et al., 2019). This means that data were included from all participants that were randomized in the study and not just participants who completed assessments at each timepoint, which increases the chances of unbiased results by limiting the possibility of bias due to attrition (McCleary, 2002). Multiple imputation was used to estimate missing data at post-treatment or follow-up time points. For the current study, descriptive characteristics for demographic and clinical variables were computed using means and standard deviations for continuous variables and frequencies for categorical variables.

1.a The first research objective – to examine whether insomnia symptoms at baseline predicted changes in binge eating frequency from pre- to post-treatment in the combined DBT conditions – was examined using two hierarchical multiple regression analysis. The first regression included participants in the combined DBT conditions who reported at least mild symptoms of insomnia (mild to severe as indicated by ISI scores of >7; *n*=30). The second regression was exploratory and included only participants who scored above the cut-off for clinical levels of insomnia symptoms (moderate or severe as indicated by ISI scores of >14; *n*=14). The model for both regression analyses included ISI total score at baseline as the predictor variable; BMI, BSI anxiety and depression scores at baseline as the covariates; and change in binge frequency from baseline to post-treatment as the criterion variable.

1.b To examine whether insomnia symptoms at baseline predicted change in eating disorder psychopathology (i.e., EDE-Q Global score) from baseline to post-treatment in the combined DBT conditions –two hierarchical multiple regression analyses were conducted. The first regression included participants in the DBT condition who reported at least mild symptoms of insomnia (mild to severe as indicated by ISI scores of >7; *n*=30). The second regression was exploratory and included only participants who scored above the cut-off for clinical levels of insomnia symptoms (moderate or severe as indicated by ISI scores of >14; *n*=14). Both hierarchical multiple regression analyses included ISI total score at baseline as the predictor variable; BMI, BSI anxiety and depression scores at baseline as the covariates; and change in eating disorder symptomology (measured using the EDE-Q Global score) from baseline to post-treatment as the criterion variable. The reason why BMI, BSI anxiety and depression scores were included as covariates in all of the analyses is because BMI and mood disorders such as depression and anxiety are commonly associated with increased severity of eating disorder psychopathology in BED (Kenny, Singleton, & Carter, 2017).

2. To test the second research objective a 3(Time) by 2(Group) Repeated Measures ANOVA was conducted to examine whether insomnia symptom severity improved across the three time points (i.e., baseline, post-treatment and follow-up) in both the DBT and SE conditions among individuals who reported at least mild insomnia symptoms at baseline (*n*=46). As appropriate, post-hoc analyses using paired samples t-tests were conducted to identify where significant changes occurred.

3.a. The third research objective – to examine whether changes in insomnia symptom severity from pre- to post-treatment predicted changes in binge eating

frequency and eating disorder psychopathology from pre- to post-treatment – was tested using two hierarchical multiple regression analyses. The first regression included participants in the DBT condition who reported at least mild symptoms of insomnia (mild to severe as indicated by ISI scores of >7; *n*=30). The second regression was exploratory and included only participants who scored above the cut-off for clinical levels of insomnia symptoms (moderate or severe as indicated by ISI scores of >14; *n*=14). The model for both hierarchical multiple regression analyses included change in ISI total scores from baseline to post-treatment as the predictor variable; change in BMI, BSI depression and anxiety scores as the covariates; and change in binge frequency (measured using the EDE-17) from baseline to post-treatment as the criterion variable.

3.b. The second part of the third research objective – to examine whether change in insomnia symptoms from baseline to post-treatment predicted change in eating disorder psychopathology (i.e., EDE Global score) from baseline to post-treatment – was analyzed using two hierarchical multiple regression analyses. The first regression included participants in the DBT condition who reported at least mild symptoms of insomnia (mild to severe as indicated by ISI scores of >7; *n*=30). The second regression was exploratory and included only participants who scored above the cut-off for clinical levels of insomnia symptoms (moderate or severe as indicated by ISI scores of >14; *n*=14). Both hierarchical multiple regression analyses included change in ISI total scores from baseline to post-treatment as the predictor variable; change in BMI, BSI depression and anxiety scores as the covariates; and changes in eating disorder symptomology (measured using the EDE-Q Global score) from baseline to post-treatment as the criterion variable.

4. Finally, because the larger trial revealed improvements in binge eating frequency and eating disorder symptomology from baseline to post-treatment in all three conditions, the final objective was to evaluate whether individuals who were in complete remission from binge eating at post-treatment (i.e., no episodes of binge eating over the previous 28 days) reported significantly different levels of insomnia symptom severity at baseline compared to individuals who did not meet complete remission criteria at post-treatment. This analysis included the full study sample (i.e., participants in both DBT and control conditions). Participants were categorized based on whether they were abstinent (i.e., no episodes of binge eating over the previous 28 days) at post-treatment and those who were not abstinent. An independent samples t-test was conducted to compare the abstinent versus non-abstinent groups on mean total ISI scores at baseline. Next, a chi-squared test of independence was conducted to compare the proportion of individuals in the two abstinent groups across the four categories of insomnia symptom severity at baseline.

3.0 Results

3.1 Baseline Characteristics of the Sample

The sample consisted of 71 participants who met DSM-5 criteria for BED and took part in the larger trial. The majority of participants were female (*n*=66; 93%) and Caucasian (*n*=69; 97%). Most participants identified as either married/common law (*n*=42; 59%) or single (*n*=24; 34%) and had an education level of a bachelor's degree (*n*=26; 37%) or college diploma (*n*=28; 39%). Furthermore, the sample reported a mean age of 40.70 years (*SD* = 11.46) and a mean BMI of 37.34 (*SD* = 9.47). Additional details regarding the descriptive characteristics of the sample are displayed in Table 1. In terms

of clinical characteristics, the 71 participants reported an average of 17.11 (*SD* = 17.02) binge episodes over the past 28 days before intervention was implemented. The sample displayed a mean of 4.44 (*SD* = 0.92) on the EDE-Q Global, which is in the clinical range. The baseline means and standard deviations for the Dietary Restraint, Body Dissatisfaction and Overvaluation of Weight and Shape subscales are presented in Table 2. As measured by the BSI, the sample reported an average depression score of 1.34 (*SD* = 0.80) and an average anxiety score of 1.12 (*SD* = 0.81). Compared to the average score that is often seen in healthy individuals of 0.28, and the average score in psychiatric patients of 1.70+ (Derogatis & Melisaratos, 1983), the mean raw scores in this sample indicates moderate levels of both anxiety and depressive symptoms. The sleep characteristics of the whole sample were considered to be in the subthreshold range of insomnia symptoms with a mean ISI score of 10.91 (*SD* = 6.56) that ranged from 0 to 24. The majority of the sample fell in the no clinical insomnia symptoms or subthreshold insomnia symptoms range. Whereas, 31% of the sample (*n*=22) reported clinical levels of insomnia symptoms at baseline (moderate or severe). The percentages of individuals across all four clinical categories are displayed in Table 2.

Further analyses were conducted to compare the combined DBT treatment conditions and the SE control condition on descriptive and clinical characteristics. Independent samples t-tests were conducted for continuous variables and chi-squared analysis for categorical variables. Results revealed that the two groups did not differ significantly on clinical characteristics (i.e., binge eating, EDE-Q Global, EDE-Q subscales, BSI anxiety and depression or insomnia symptom severity) or descriptive characteristics with the exception of ethnicity. The DBT conditions consisted of

significantly more Caucasian individuals (n=48; 100%) compared to the SE condition (n=21; 91%), $\chi^2(1, n=71) = 4.30$, $p = .04$, Cramer's $V = .25$. Results regarding the baseline descriptive variables for both DBT and SE conditions are reported in Table 3 and results for baseline clinical variables are reported in Table 4.

3.2 Does Insomnia Symptom Severity at Baseline Predict Change in Binge Frequency from Baseline to Post-Treatment in DBT Self-Help?

The first research objective was to evaluate whether insomnia symptom severity at baseline predicted treatment outcome in the combined DBT treatment condition, after controlling for relevant covariates. As indicated in Table 4, 30 participants in the combined DBT conditions reported experiencing at least mild (subthreshold) symptoms of insomnia at baseline – exceeding a cut-off of seven on the ISI. Insomnia severity at baseline did not account for incremental variance in binge frequency above and beyond BMI, anxiety and depression, $R^2change = .03$, F (4, 25) = 1.86, $p = .35$, see Table 5. A second hierarchical multiple regression was then conducted with participants who met criteria for clinical levels of insomnia symptoms at baseline – exceeding a clinical cut-off of 14 on the ISI (n=14). Among those with clinical levels of insomnia, insomnia severity predicted change in binge frequency from baseline to post-treatment at a trend level with a moderate effect size, $R^2change = .23$, F (4, 9) = 1.40, $p = .10$ after controlling for baseline BMI, anxiety and depression scores, see Table 6.

Next, two hierarchical multiple regression analyses were conducted to evaluate whether insomnia symptoms at baseline predicted change in eating disorder symptomatology (i.e., EDE-Q Global score) from baseline to post-treatment. The first analysis included the 30 participants in the combined DBT condition who experienced at

least mild symptoms of insomnia at baseline (exceeding a cut-off of seven on the ISI). Insomnia symptom severity at baseline did not account for incremental variance in eating disorder symptomology (e.g., EDE-Q Global) above and beyond BMI, anxiety and depression, $R^2change$ = .04, F (4, 25) = 1.14, p = .28, see Table 7. A second exploratory hierarchical multiple regression analysis was then conducted on participants who experienced clinical levels of insomnia symptoms at baseline – exceeding a clinical cut-off of 14 on the ISI (n=14). Among those with clinical levels of insomnia symptoms, insomnia symptom severity at baseline did not account for incremental variance in eating disorder symptomology (e.g., EDE-Q Global) above and beyond BMI, anxiety and depression $R^2change$ = .01, F (4, 9) = 0.10, p = .77, see Table 8.

3.3 Did Insomnia Symptoms Improve from Baseline to Post-Treatment in the DBT and Active Control Conditions?

To test the second research objective of the current study – whether there were reductions in insomnia symptoms across time among participants in the DBT and active control conditions who reported at least mild symptoms of insomnia at baseline (exceeding the ISI cut-off of seven; n=46) – a 3(Time) X 2(Group) repeated measures ANOVA was conducted. The results revealed a significant main effect for Time, F (2, 82.03) = 7.28, $p < .01$, $\eta^2 = 0.19$. However, there was no significant main effect of Group, F(1, 39.82) = 0.85, p = 0.36 and no significant Interaction, F(2, 83.23) = 0.24, p = 0.79. This suggests that insomnia symptoms improved significantly across time (i.e., pre-treatment, post-treatment and follow-up), with no differences between the two treatment conditions (i.e., control versus DBT). Post-hoc paired samples t-tests were conducted to

assess where the significant changes occurred. After applying the Bonferroni correction for two comparisons (0.05/2=0.025) to control for Type I error, pairwise comparisons revealed significant reductions in insomnia symptoms in both DBT and control conditions (n=46) from pre-treatment (M=14.67) to post-treatment (M=9.42), $t(45) = 6.47$, $p < .01$, which were maintained at follow-up, with no significant changes from post-treatment (M=9.42) to follow-up (M=10.28), $t(45) = -0.92$, $p = 0.36$. These results are displayed in Figure 1.

3.4 Do Changes in Insomnia Symptom Severity from Baseline to Post-Treatment Predict Changes in Binge Frequency or Eating Disorder Symptomatology from Baseline to Post-Treatment in the DBT Conditions?

The third research objective was to examine whether change in insomnia symptom severity from baseline to post-treatment predicted changes in binge frequency from baseline to post-treatment in the DBT condition. The first hierarchical multiple regression analysis included individuals who reported at least mild insomnia symptoms (n=30) at baseline with ISI change scores from pre- to post-treatment as the predictor variable, change in BMI, BSI anxiety and BSI depression as the covariates and change in binge frequency as the criterion variable. Contrary to the hypothesis, results revealed that change in insomnia symptoms (mild to severe; n=30) did not account for incremental variance in change in binge frequency from baseline to post-treatment, $R^2change = .00$, $F(4, 25) = 1.65$, $p = .98$, after controlling for changes in BMI, anxiety and depression, see Table 9. Next, a second exploratory hierarchical multiple regression was conducted on participants (n=14) who met criteria for clinical levels of insomnia symptoms at baseline – scoring at the ISI clinical cut-off of 15 or above. The model included ISI change scores

from pre- to post-treatment as the predictor variable, change in BMI, BSI anxiety and BSI depression as the covariates, and change in binge frequency from pre- to post-treatment as the criterion variable. Change in clinical levels of insomnia symptoms, moderate or severe (n=14), did not account for incremental variance in change in binge eating frequency from pre-to post-treatment, $R^2change$ = .03, F (4, 9) = 0.55, p = .58, after controlling for changes in BMI, anxiety and depression, see Table 10.

Next, two hierarchical multiple regression analyses were conducted to evaluate whether change in insomnia symptoms predicted change in eating disorder symptomatology (i.e., EDE-Q Global score) from baseline to post-treatment. The first analysis included participants who experienced at least mild symptoms of insomnia at baseline (n=30), with the model containing ISI change scores from baseline to post-treatment as the predictor variable, change scores for BMI, anxiety and depression scores as the covariates, and change in EDE-Q global scores from baseline to post-treatment as the criterion variable. Contrary to the hypothesis, results indicated that change in at least mild insomnia symptoms (n=30) did not account for incremental variance in change in eating disorder psychopathology (i.e., EDE-Q Global) from baseline to post-treatment, $R^2change$ = .07, F (4, 25) = 1.41, p = .15 after controlling for baseline BMI, anxiety and depression scores, see Table 11. A second exploratory hierarchical multiple regression analysis was then conducted on participants who experienced clinical levels of insomnia symptoms at baseline (n=14), with a model including ISI change scores from baseline to post-treatment as the predictor variable, change scores for BMI, anxiety and depression scores as the covariates, and change in EDE-Q global scores from baseline to post-treatment as the criterion variable. Findings revealed that clinical levels of insomnia

symptoms at baseline did not account for incremental variance in change in eating disorder psychopathology from baseline to post-treatment, $R^2change = .08$, F (4, 9) = 0.25, $p = .41$, after controlling for baseline BMI, anxiety and depression scores, see Table 12.

3.5 Was Insomnia Symptom Severity at Baseline Associated with Abstinence from Binge Eating?

The fourth and final research objective was to examine whether participants who were in remission from binge eating at post-treatment (n=30) – defined as no episodes of binge eating over the previous 28 days – differed in terms of baseline insomnia symptom severity from participants who did not achieve abstinence from binge eating (n=41) at post-treatment. Out of the full sample (N=71), 42% met criteria for abstinence at post-treatment and the remaining 58% did not. An independent samples t-test demonstrated that the abstinent (M=11.18) and non-abstinent (M=10.71) groups did not differ significantly on baseline insomnia symptom severity total scores, t(185.13) = -0.24, p = .81. Next, a chi-squared test of independence was conducted to compare the abstinent and non-abstinent groups across the four categories of insomnia symptom severity, (i.e., no clinically significant insomnia symptoms (0-7), subthreshold insomnia symptoms (8-14), clinically significant insomnia symptoms moderate intensity (15-21) and clinically significant insomnia symptoms severe intensity (22-28). Findings revealed that the proportion of the individuals in the four insomnia categories between the abstinent and non-abstinent groups approached statistical significance with a moderate effect size, χ^2(3, n=70) = 6.88, p = .08, Cramer's V = 0.31. As illustrated in Figure 2, the majority (69%) of

individuals reported no clinical insomnia symptoms or subthreshold levels of insomnia symptoms at baseline. However, among the individuals who reported clinically relevant insomnia symptoms at baseline (i.e., scoring at 15 or above), more of them fell into the non-abstinent group (59%) than the abstinent group (41%); however, this difference was not statistically significant, $\chi^2(2, n=70) = 1.34$, $p = .25$, Cramer's $V = 0.14$, but the effect size was moderate.

4.0 Discussion

4.1 Main Findings

This was the first study to evaluate insomnia symptoms as a predictor of DBT treatment outcome in BED. The first finding was that among individuals who reported at least mild insomnia symptoms at baseline, severity of insomnia symptoms did not significantly predict change in binge frequency or eating disorder symptomology from pre- to post-treatment. Furthermore, among individuals with clinical levels of insomnia symptoms at baseline, insomnia symptom severity did not significantly predict change in eating disorder symptomology from pre- to post-treatment. However, among those who reported clinical levels of insomnia symptoms at baseline, there was a trend for symptom severity at baseline to predict binge frequency reduction from pre- to post-treatment with a moderate effect size. However, the regression analysis was statistically underpowered due to the small sample size of this subgroup (n=14) and requires further examination with a larger sample. It may be interpreted that clinically relevant levels of insomnia symptoms (moderate or severe) may be related to treatment outcome for BED with

regards to binge frequency only. However, further research is required to better understand this relationship.

These findings are consistent with the only previous study that assessed poor sleep and treatment outcome in a sample of mixed eating disorders based on DSM-IV criteria, the majority of which had BED (Lombardo et al., 2015). Persistent poor sleep from pre-treatment to six months post-treatment predicted severity of eating disorder symptoms (Lombardo et al., 2015). However, the current study found preliminary evidence of a relationship between insomnia symptoms and binge frequency outcome at a trend level, but not eating disorder symptomology outcome. One methodological reason that may account for this discrepancy in results is the differences in measures implemented to measure eating disorder symptomology in the two studies. The current study used the gold standard EDE-Q (Fairburn, 2008) to measure eating disorder psychopathology, whereas Lombardo et al. (2015) used the EDI (Garner, 1991). Moreover, the current study looked specifically at insomnia symptoms measured by the ISI (Bastien et al., 2001) in a sample of BED, while Lombardo et al. (2015) used the sleep subscale from the SCL-90 (Derogatis et al., 1973) to measure sleep in a sample with mixed eating disorders. The reliability of the sleep subscale from the SCL-90 has not been assessed and the subscale holds uncertain construct validity (Lombardo et al., 2015). Nonetheless, findings from both studies suggest that sleep may contribute to the treatment outcome of eating disorders.

The second main finding was consistent with the hypothesis that there would be a significant decrease in insomnia symptoms from pre- to post-treatment with the decrease in number of binge eating episodes. Among individuals with at least mild symptoms of

insomnia, insomnia symptoms improved significantly from pre- to post-treatment in both DBT and SE conditions, without targeted treatment for insomnia symptoms. This improvement was maintained at follow-up. However, the size of the change was not clinically significant, and the mean score remained above the cut-off score of eight, indicating mild insomnia.

Although no previous research has evaluated the relationship between insomnia symptoms and treatment outcome of BED, the current findings are consistent with those of Kenny and colleagues (2018) which revealed a positive relationship between BED and insomnia symptoms at baseline. Specifically, it was found that more severe symptoms of insomnia predicted more frequent binge episodes in BED (Kenny et al., 2018), whereas findings from the current study revealed that reductions in insomnia symptom severity from pre- to post-treatment were concurrent with reductions in binge eating frequency from pre- to post-treatment. This suggests that not only may insomnia symptoms be positively related to BED at baseline, but also throughout and after treatment. This provides additional support for the relationship between the two variables and the importance of assessing and considering sleep quality in the assessment and treatment of BED.

The third main finding was that change in insomnia symptom severity was not a statistically significant predictor of change in either binge frequency or eating disorder symptomology from baseline to post-treatment, after controlling for changes in BMI, anxiety and depression. As previously mentioned, the current results suggest that, while both binge eating and insomnia improved during treatment, there was no evidence of an association between these two variables. This may reflect low statistical power due to the

small sample size on which the analyses were based. However, the effect sizes for these analyses were small. Furthermore, results from the current study are not consistent with the results of Lombardo and colleagues (2015) in that they found a significant association between severity of poor sleep and the severity of eating disorder symptoms at baseline and post-treatment, whereas this was not found in the present study. However, the previous study did not find improvements in sleep quality from pre-treatment to six months post-treatment of "standard therapy" for eating disorders (Lombardo et al., 2015). Therefore, further research with larger samples is required to better understand the relationship between sleep and treatment outcome for eating disorders.

Finally, there were no significant differences in insomnia symptom severity at baseline between the abstinent and non-abstinent groups. However, the proportion of the individuals in the four insomnia categories (no clinical insomnia symptoms, subthreshold insomnia symptoms, clinically moderate insomnia symptoms and clinically severe insomnia) between the abstinent and non-abstinent groups approached statistical significance with a moderate effect size. This finding indicates that insomnia symptom severity at baseline may be related to BED treatment outcome. It is possible that this difference did not reach statistical significance because the current study was statistically underpowered due to small group sizes in these analyses. Regardless, this finding should be interpreted with caution. Additionally, a greater proportion of individuals who were not abstinent from binge eating at post-treatment reported clinical levels of insomnia symptoms at baseline compared to the proportion of individuals who were abstinent from binge eating at post-treatment. Although this trend did not reach statistical significance, it suggests that more severe levels of insomnia symptoms at baseline may be related to

poorer treatment outcome in BED. However, further research is required to confirm this relationship. Similar trends have been found in other studies involving mixed eating disorder samples (Lombardo et al., 2015), mood disorders (Dombrovski et al., 2007; Manber et al., 2008; Troxel el at., 2012), and addictive disorders (Brower et al., 1998; Brower, Aldrich, Robinson, Zucker, & Greden, 2001; Conroy et al., 2006) indicating that poor sleep and insomnia symptoms are associated with poorer treatment outcome. Thus, the current findings are comparable to those from studies evaluating insomnia and treatment outcome in disorders related to BED (Dombrovski et al., 2007; Manber et al., 2008; Troxel el at., 2012).

4.2 Strengths, Limitations, and Future Research Directions

4.2.1 Strengths and future directions. The current study had a number of methodological strengths. First, unlike most research studies on BED, the data used for this secondary analysis were from a sample that was recruited directly from the community as opposed to a clinic sample. A community sample is likely to be more generalizable compared to clinical samples. Secondly, intention-to-treat analysis was used rather than completer analysis. This means that data were included from all participants that were randomized in the study and not just participants who completed assessments at each time point, which increases the chances of unbiased results and limits the possibility of bias due to attrition (McCleary, 2002). Multiple imputation was used to estimate missing data at post-treatment or follow-up time points. A third methodological strength is the longitudinal study design as opposed to a cross-sectional design. A longitudinal experiment provides the ability to observe and measure a sequence of events that occur

over time. Furthermore, longitudinal study designs provide more reliable results as it can account for changes that may impact the observed relationships occurring over time.

Another strength of this study is that a well-validated and reliable investigator-based structured interview was used to diagnose BED (Fairburn et al., 2014). Furthermore, all the self-report measures utilized were well validated and demonstrated good reliability (Bastien et al., 2001; Berg et al., 2012; Derogatis & Melisaratos, 1983). A direction for future research is to look at the relationship between insomnia as a predictor of treatment outcome for BED using other evidence-based treatments apart from DBT (e.g., CBT and IPT). Both CBT and IPT have been well established as effective treatments for BED with moderate levels of short and long-term remission rates (Hilbert et al., 2012; Iacovino et al., 2012; Wilfley et al., 2002; Wilson et al., 2007). Therefore, it may be fruitful to evaluate whether insomnia symptoms predict outcome for other treatment approaches.

Additionally, among BED patients with clinical levels of insomnia, another direction for future studies is to treat the insomnia first using CBT-I, a first line of treatment for patients with insomnia (Qaseem, Kansagara, Forciea, Cooke, & Denberg, 2016; Riemann et al., 2017), and then follow it with treatment for BED. Improvement in sleep quality before initiating treatment for BED may allow researchers and clinicians to assess how patients respond to a treatment that is not confounded by sleep quality and can potentially eliminate it as a predictor of treatment outcome.

4.2.2 Limitations and future directions. This study also had a number of limitations. First and foremost, this study focused on a small sample of participants who reside in the province of Newfoundland and Labrador, the majority of whom were

Caucasian, female and highly educated. This limits the generalizability of the findings. Additionally, the small sample size resulted in underpowered statistical analyses and therefore may have failed to reveal significant relationships, limiting confidence in the findings. Thus, further research employing larger sample sizes is recommended to further evaluate the association between insomnia symptoms and BED treatment outcome. Furthermore, a larger sample size containing participants from provinces across Canada will allow for greater statistical power as well as findings that are more generalizable.

Secondly, only one self-report measure was used to evaluate insomnia symptoms. Although the ISI is a validated self-report measure for assessing insomnia symptoms, further research should consider a structured interview to confirm an insomnia diagnosis. A direction for future research is for studies to include more than one measure of sleep, such as sleep diaries or an objective measure. Studies should also include food diaries in combination with sleep diaries to gain a more comprehensive understanding between the association of sleep and BED (e.g., when bingeing occurs with relation to sleep time). Lastly, self-report measures were used in-order to assess eating disorder psychopathology (EDE-Q), anxiety and depression psychopathology (BSI). Therefore, participants were susceptible to response bias (e.g., answering questions untruthfully).

4.3 Clinical Implications

Poor sleep and insomnia have been shown to hinder treatment outcome and increase susceptibility to relapse in various disorders that are commonly related to each other, such as mood disorders (Dombrovski et al., 2007; Manber et al., 2008; Troxel el at., 2012) substance use disorders (Brower et al., 1998; Brower et al., 2001; Conroy et al., 2006) and eating disorders (Lombardo et al., 2015). This was the first study to assess the

relationship between insomnia symptoms and treatment outcome in BED. Among individuals with clinical levels of insomnia symptoms, insomnia severity predicted change in binge frequency from baseline to post-treatment at a trend level. This provides preliminary evidence of an association between moderate or severe insomnia symptoms and BED treatment outcome. Therefore, it may be important to implement treatment for insomnia symptoms first or alongside BED treatment. Examples of treatment for insomnia symptoms may include stimulus control, psychoeducation for sleep hygiene or cognitive behavioral therapy for insomnia. However, further research with larger samples is required to confirm this finding.

Another trend-level finding showed that among individuals who reported clinical levels of insomnia symptoms at baseline (i.e., scoring > 14), the majority of them did not achieve abstinence from binge eating at post-treatment. This suggests that moderate or severe insomnia symptom severity at baseline may be related to poorer treatment outcome in BED. However, further research with larger samples is needed to confirm this relationship.

This research provides a new avenue for future studies on how insomnia symptoms may relate to the treatment outcome of mental health conditions such as BED. If further research reveals a relationship between insomnia symptoms and treatment outcome for BED, it may be pertinent for clinicians working with individuals with BED to include assessment and treatment for insomnia symptoms when seeing clients with BED to improve overall treatment outcome. However, further research is imperative to better understand the role sleep plays in BED treatment outcome.

5.0 Conclusion

In conclusion, the findings from this study provide preliminary evidence that there might be a relationship between insomnia symptoms and DBT treatment outcome for BED. However, further research is required to confirm this relationship. Further investigation of these research questions may determine whether clinicians need to consider sleep quality when working with individuals who have BED, and when developing treatment plans for BED.

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Tables.

Table 1. Descriptive characteristics of the sample.

	BED (*n*=71) Mean (*SD*) or *n* (%)
BMI	37.34 (9.47)
Age	40.70 (11.46)
Biological Sex	
Male	5 (7%)
Female	66 (93%)
Marital Status	
Single	24 (34%)
Married/Common Law	42 (59%)
Divorced	3 (4%)
Widowed	0 (0%)
Separated	2 (3%)
Ethnicity	
Caucasian/White	69 (97%)
Hispanic	0 (0%)
Black	0 (0%)
Asian	0 (0%)
Other	2 (3%)
Highest level of Education	
High School Diploma or Equivalent	6 (8%)
College Diploma	28 (39%)
Bachelor's Degree	26 (37%)
Graduate Degree	11 (16%)

Note. BMI = Body Mass Index; SD = Standard Deviation

Table 2. Clinical characteristics of the sample.

	BED (*n*=71) Mean (*SD*) or *n* (%)
EDE-Q	
Dietary Restraint	3.28 (2.06)
Overvaluation	4.63 (1.52)
Body Dissatisfaction	5.42 (0.86)
ISI	
No Clinical Insomnia	24 (34%)
Subthreshold Insomnia	25 (35%)
Clinical Moderate Insomnia	15 (21%)
Clinical Severe Insomnia	7 (10%)

Note. EDE-Q = Eating Disorder Examination Questionnaire; ISI = Insomnia Severity Index

Table 3. Descriptive characteristics of the DBT and SE groups.

	Group	
	DBT (*n*=48) Mean (*SD*) or *n* (%)	**SE (*n*=23)** Mean (*SD*) or *n* (%)
BMI	36.30 (8.82)	39.50 (10.60)
Age	40.54 (11.91)	41.04 (10.73)
Biological Sex		
Male	4 (8%)	1 (4%)
Female	44 (92%)	22 (96%)
Marital Status		
Single	14 (29%)	10 (44%)
Married/Common Law	29 (61%)	13 (56%)
Divorced	3 (6%)	0 (0%)
Widowed	0 (0%)	0 (0%)
Separated	2 (4%)	0 (0%)
Ethnicity		
Caucasian/White	48 (100%)	21 (91%) *
Hispanic	0 (0%)	0 (0 %)
Black	0 (0%)	0 (0 %)
Asian	0 (0%)	0 (0 %)
Other	0 (0%)	2 (9 %)
Highest level of Education		
High School Diploma or Equivalent	6 (12%)	0 (0%)
College Diploma	18 (38%)	10 (44%)
Bachelor's Degree	17 (35%)	9 (39%)
Graduate Degree	7 (15%)	4 (17%)

Note. BMI = Body Mass Index; SD = Standard Deviation
Note. * indicates $p < .05$

Table 4. Clinical characteristics of the DBT and SE groups.

	Group				
	DBT (*n*=48) Mean (*SD*) or *n* (%)	**SE (*n*=23)** Mean (*SD*) or *n* (%)	*t* or χ^2	Cohen's *d* or *Cramer's V*	*p*
Binge Episodes (past 4 weeks)	14.90 (10.53)	21.74 (25.53)	-1.24	0.35	0.23
EDE-Q					
Restraint	3.44 (2.14)	2.94 (1.90)	0.95	0.25	0.35
Overvaluation	4.45 (1.57)	5.00 (1.36)	-1.45	0.37	0.15
Body Dissatisfaction	5.36 (0.80)	5.54 (0.99)	-0.82	0.20	0.42
Global	4.42 (0.96)	4.50 (0.86)	-0.33	0.09	0.74
BSI					
Depression	1.28 (0.81)	1.46 (0.76)	-0.89	0.23	0.38
Anxiety	1.14 (0.86)	1.09 (0.71)	0.23	0.06	0.82
ISI	10.34 (6.69)	12.09 (6.25)	-1.05	0.27	0.30
ISI Clinical Categories					
No Clinical	18 (37%)	7 (30%)	0.47	0.08	0.93
Subthreshold	16 (33%)	8 (35%)			
Clinical Moderate	10 (21%)	5 (22%)			
Clinical Severe	4 (9%)	3 (13%)			

Note. EDE-Q = Eating Disorder Examination Questionnaire; BSI = Brief Symptom Inventory; ISI = Insomnia Symptom Index; SD = Standard Deviation

Table 5. Results of multiple regression analysis of individuals with insomnia symptoms in the DBT condition (*n*=30), evaluating insomnia symptoms at baseline as a predictor of change in binge frequency from pre- to post-treatment, after controlling for BMI, anxiety and depression.

		Unstandardized coefficients		Standardized coefficients	
Change in Binge Frequency	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		-0.03	0.02	-0.42	-1.70
Anxiety		-0.02	0.20	0.09	-0.10
Depression		-0.06	0.20	-0.16	-0.32
Total Model	.20				
2					
BMI		-0.03	0.02	-0.45	-1.83
Anxiety		0.01	0.20	0.13	0.03
Depression		-0.05	0.20	-0.15	-0.26
Insomnia Symptoms		-0.02	0.02	-0.18	-0.91
Total Model	.23				

Note. BMI = Body Mass Index

Table 6. Results of multiple regression analysis of individuals with clinical levels of insomnia symptoms in the DBT condition (*n*=14) evaluating insomnia symptoms at baseline as a predictor of change in binge frequency from pre- to post-treatment, after controlling for BMI, anxiety and depression.

		Unstandardized coefficients		Standardized coefficients	
Change in Binge Frequency	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		-0.02	0.03	-0.39	-0.80
Anxiety		-0.16	0.29	-0.16	-0.56
Depression		0.16	0.35	0.24	0.44
Total Model	.15				
Block 2					
BMI		-0.02	0.02	-0.43	-0.95
Anxiety		-0.15	0.28	-0.13	-0.53
Depression		0.12	0.33	0.17	0.35
Insomnia Symptoms		-0.07	0.05	-0.49	-1.37
Total Model	.38				

Note. BMI = Body Mass Index

Table 7. Results of a multiple regression analysis of individuals with insomnia symptoms in the DBT condition (*n*=30) evaluating insomnia symptoms at baseline as a predictor of change in eating disorder psychopathology from pre- to post-treatment, after controlling for BMI, anxiety and depression.

		Unstandardized coefficients		Standardized coefficients	
Change in EDE-Q Global	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		-0.02	0.04	-0.17	-0.57
Anxiety		0.34	0.52	0.37	0.65
Depression		-0.35	0.52	-0.23	-0.67
Total Model	.11				
2					
BMI		-0.02	0.04	-0.14	-0.44
Anxiety		0.29	0.53	0.33	0.55
Depression		-0.37	0.52	-0.25	-0.70
Insomnia Symptoms		0.04	0.07	0.22	0.64
Total Model	.15				

Note. BMI = Body Mass Index; EDE-Q Global = Measure of eating disorder psychopathology

Table 8. Results of a multiple regression analysis of individuals with clinical levels of insomnia symptoms in the DBT condition (*n*=14) evaluating insomnia symptoms at baseline as a predictor of change in eating disorder psychopathology from pre- to post-treatment, after controlling for BMI, anxiety and depression.

		Unstandardized coefficients		Standardized coefficients	
Change in EDE-Q Global	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		-0.04	0.09	-0.19	-0.47
Anxiety		0.02	1.00	0.05	0.02
Depression		-0.09	1.15	0.06	-0.08
Total Model	.03				
Block 2					
BMI		-0.04	0.09	-0.18	-0.43
Anxiety		0.00	1.04	0.05	0.00
Depression		-0.06	1.19	0.07	-0.05
Insomnia Symptoms		0.06	0.19	0.10	0.31
Total Model	.04				

Note. BMI = Body Mass Index; EDE-Q Global = Measure of eating disorder psychopathology

Table 9. Results of a multiple regression analysis of individuals with insomnia symptoms in the DBT condition (*n*=30) evaluating change in insomnia symptoms from pre- to post-treatment as a predictor of change in binge frequency from pre- to post-treatment, after controlling for changes in BMI, anxiety and depression.

		Unstandardized coefficients		Standardized coefficients	
Change in Binge Frequency	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		0.00	0.04	0.34	0.04
Anxiety		-0.20	0.22	-0.37.	-0.91
Depression		0.26	0.24	0.28	1.07
Total Model	.21				
2					
BMI		0.00	0.04	0.34	0.04
Anxiety		-0.20	0.22	-0.37.	-0.91
Depression		0.26	0.26	0.27	1.02
Insomnia Symptoms		0.00	0.02	0.00	0.03
Total Model	.21				

Note. BMI = Body Mass Index

Table 10. Results of a multiple regression analysis of individuals with clinical levels of insomnia symptoms in the DBT condition (*n*=14) evaluating change in insomnia symptoms from pre- to post-treatment as a predictor of change in binge frequency from pre- to post-treatment, after controlling for changes in BMI, anxiety and depression.

		Unstandardized coefficients		Standardized coefficients	
Change in Binge Frequency	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		-0.06	0.10	0.37	-0.60
Anxiety		-0.08	0.29	-0.45	-0.28
Depression		0.28	0.35	0.21	0.79
Total Model	.17				
Block 2					
BMI		-0.06	0.10	0.41	-0.58
Anxiety		-0.09	0.31	-0.54	-0.28
Depression		0.27	0.38	0.16	0.73
Insomnia Symptoms		0.00	0.03	0.21	0.07
Total Model	.20				

Note. BMI = Body Mass Index

Table 11. Results of a multiple regression analysis of individuals with insomnia symptoms in the DBT condition (*n*=30) evaluating changes in insomnia symptoms from pre- to post-treatment as a predictor of change in eating disorder psychopathology from pre- to post-treatment, after controlling for changes in BMI, anxiety and depression.

		Unstandardized coefficients		Standardized coefficients	
Change in EDE-Q Global	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		0.04	0.09	0.16	0.48
Anxiety		-0.19	0.55	-0.36	-0.34
Depression		0.24	0.58	0.27	0.41
Total Model	.11				
Block 2					
BMI		0.05	0.09	0.21	0.61
Anxiety		-0.29	0.53	-0.41	-0.55
Depression		0.05	0.58	0.18	0.09
Insomnia Symptoms		0.09	0.06	0.30	1.42
Total Model	.18				

Note. BMI = Body Mass Index; EDE-Q Global = Measure of eating disorder psychopathology

Table 12. Results of a multiple regression analysis of individuals with clinical levels of insomnia symptoms in the DBT condition (*n*=14) evaluating changes in insomnia symptoms from pre- to post-treatment as a predictor of change in eating disorder psychopathology from pre- to post-treatment, after controlling for changes in BMI, anxiety and depression.

		Unstandardized coefficients		**Standardized coefficients**	
Change in EDE-Q Global	R^2	**B**	**Standard Error**	β	*t*
Block 1					
BMI		-0.02	0.27	-0.12	-0.09
Anxiety		0.12	0.92	-0.03	0.13
Depression		0.21	1.01	0.16	0.21
Total Model	.02				
Block 2					
BMI		0.00	0.27	-0.05	0.01
Anxiety		-0.11	0.96	-0.18	-0.11
Depression		-0.00	1.07	0.08	-0.00
Insomnia Symptoms		0.08	0.10	0.33	0.81
Total Model	.10				

Note. BMI = Body Mass Index; EDE-Q Global = Measure of eating disorder psychopathology

Figures.

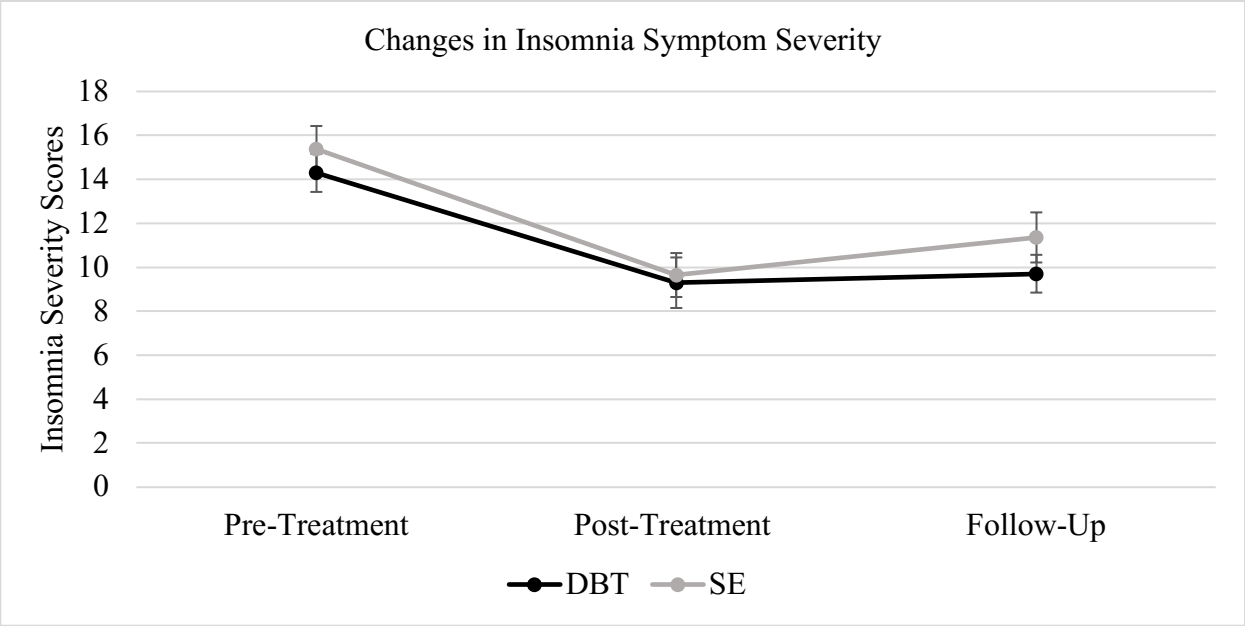


Figure 1. Improvement in insomnia symptom severity with improvement to binge eating frequency differed significantly from pre- to post-treatment and was maintained from post-treatment to follow-up in both DBT and SE conditions without targeted treatment of insomnia symptoms.

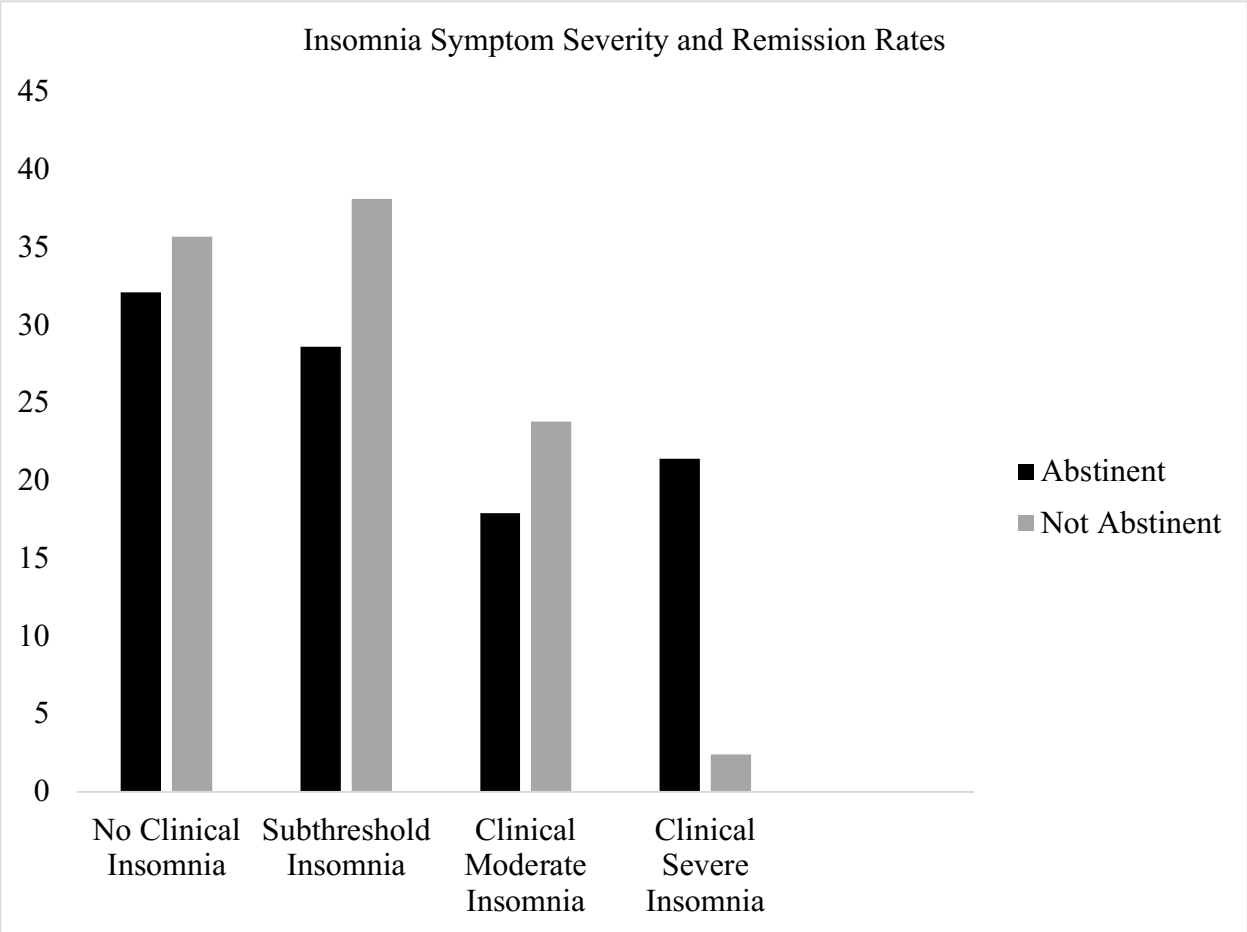


Figure 2. Proportion of individuals reporting scores of no clinically significant insomnia symptoms: 0-7, subthreshold insomnia symptoms: 8-15, clinical insomnia-moderate severity: 16-22 and clinical insomnia-severe symptoms: 23-28 at baseline were nearly significantly different across the abstinence and no abstinence groups at post-treatment.

Appendix A

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 B

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)

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?**

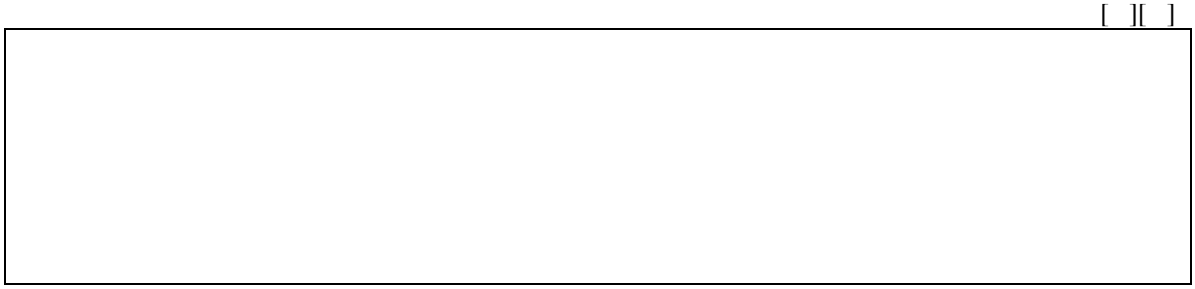
Typically, what form of exercise have you done? How hard have you exercised? Have you pushed yourself?

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.]

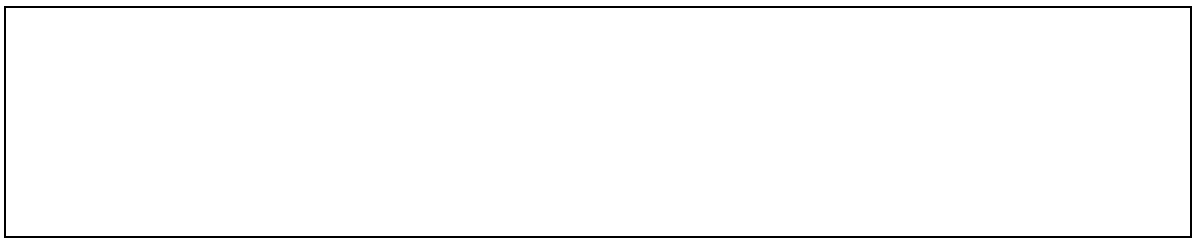
month 2 [ ][ ]
month 3 [ ][ ]

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 C

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 the 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 (i.e., 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 D

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)

Appendix E

Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983)

Below is a numbered list of problems and complaints. For each of these numbered statements circle the number on the right which best describes how much discomfort the problem has caused you over the **PAST FOUR WEEKS**. Please do not skip any items. Thank you

0 = Not at all
1 = A little bit
2 = Moderately
3 = Quite a bit
4 = Extremely

OVER THE PAST FOUR WEEKS HOW MUCH HAVE YOU BEEN DISTRESSED BY:

1. Nervousness or shakiness inside	0	1	2	3	4
2. Faintness or dizziness	0	1	2	3	4
3. The idea that someone else can control your thoughts	0	1	2	3	4
4. Feeling others are to blame for most of your troubles	0	1	2	3	4
5. Trouble remembering things	0	1	2	3	4
6. Feeling easily annoyed or irritated	0	1	2	3	4
7. Pains in the heart or chest	0	1	2	3	4
8. Feeling afraid in open spaces or on the streets	0	1	2	3	4
9. Thoughts of ending your life	0	1	2	3	4
10. Feeling that most people cannot be trusted	0	1	2	3	4
11. Poor appetite	0	1	2	3	4
12. Suddenly scared for no reason	0	1	2	3	4
13. Temper outbursts that you could not control	0	1	2	3	4
14. Feeling lonely even when you are with people	0	1	2	3	4
15. Feeling blocked in getting things done	0	1	2	3	4
16. Feeling lonely	0	1	2	3	4

17. Feeling blue	0	1	2	3	4
18. Feeling no interest in things	0	1	2	3	4
19. Feeling fearful	0	1	2	3	4
20. Your feelings being easily hurt	0	1	2	3	4
21. Feeling that people are unfriendly or dislike you	0	1	2	3	4
22. Feeling inferior to others	0	1	2	3	4
23. Nausea or upset stomach	0	1	2	3	4
24. Feeling that you are watched or talked about by others	0	1	2	3	4
25. Trouble falling asleep	0	1	2	3	4
26. Having to check and double-check what you do	0	1	2	3	4
27. Difficulty making decisions	0	1	2	3	4
28. Feeling afraid to travel on buses, subways, or trains	0	1	2	3	4
29. Trouble getting your breath	0	1	2	3	4
30. Hot or cold spells	0	1	2	3	4
31. Having to avoid certain things, places, or activities because they frighten you	0	1	2	3	4
32. Your mind going blank	0	1	2	3	4
33. Numbness or tingling in parts of your body	0	1	2	3	4
34. The idea that you should be punished for your sins	0	1	2	3	4
35. Feeling hopeless about the future	0	1	2	3	4
36. Trouble concentrating	0	1	2	3	4
37. Feeling weak in parts of your body	0	1	2	3	4
38. Feeling tense or keyed up	0	1	2	3	4
39. Thoughts of death or dying	0	1	2	3	4
40. Having urges to beat, injure, or harm someone	0	1	2	3	4
41. Having urges to break or smash things	0	1	2	3	4

42. Feeling very self-conscious with others	0	1	2	3	4
43. Feeling uneasy in crowds, such as shopping or at a movie	0	1	2	3	4
44. Never feeling close to another person	0	1	2	3	4
45. Spells of terror or panic	0	1	2	3	4
46. Getting into frequent arguments	0	1	2	3	4
47. Feeling nervous when you are left alone	0	1	2	3	4
48. Others not giving you proper credit for your achievements	0	1	2	3	4
49. Feeling so restless you couldn't sit still	0	1	2	3	4
50. Feelings of worthlessness	0	1	2	3	4
51. Feeling that people will take advantage of you if you let them	0	1	2	3	4
52. Feeling of guilt	0	1	2	3	4
53. The idea that something is wrong with your mind	0	1	2	3	4

Beck Depression Inventory II – Question 9 (Beck, Steer, Ball, & Ranieri, 1996)

Please pick out the one statement that best describes the way you have been feeling during the past two weeks, including today. If several statements in the group seem to apply, pick the highest number. Be sure that you do not choose more than one statement.

Suicidal Thoughts or Wishes	0 I don't have any thoughts of killing myself	1 I have thoughts of killing myself, but I would not carry them out	2 I would like to kill myself	3 I would kill myself if I had the chance