Change in eating behavior and eating pattern during treatment of Bulimia Nervosa and Binge Eating Disorder

A randomized, clinical trial comparing effects from cognitive behavioral therapy, and a combination of physical exercise and dietary therapy

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Master Thesis
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Master thesis

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This master project presents an important contribution to the knowledge on eating behavior, nutrient intake and food selection in BN and BED patients during CBT and PED-t treatment. The work of this study has been conducted between August 2016 and November 2017.

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Ane Korsvold

Abstract

Background and aims:

Bulimia Nervosa (BN) and Binge Eating Disorder (BED) are two Eating Disorders (EDs) with high rates of medical complications and comorbidity. Studies have found associations between BN and BED, and dysfunctional eating behavior and less healthful food choices. Around 90% of BN and BED patients never seek professional help, and the majority may go untreated. This study aimed to examine differences in treatment effects between Cognitive Behavior Therapy (CBT) and Physical Exercise and Dietary Therapy (PED-t) in female BN and BED patients, measured as changes in 1) eating behavior, and 2) eating pattern; nutrient intake and food selection.

Study design:

One hundred sixty-four female BN and BED patients were randomized to two treatment arms; CBT and PED-t. Mean ages were 28 years and 29 years, and mean BMI values were 25.5 kg/m^2 and 25.6 kg/m^2, in CBT and PED-t, respectively. Eating behavior was measured with the Binge Eating Scale (BES) and the cognitive restraint (CR), uncontrolled eating (UE) and emotional eating (EE) scales in the 21-item Three Factor Eating Questionnaire (TFEQ-R21). Eating behavior was also compared to a group of patients not receiving any treatment. Nutrient intake and food selection were assessed with 24-h dietary recalls. The intervention programs consisted of 20 sessions during a 16-week period. Changes were measured at baseline (week 0) and post treatment (week 17-18).

Results:

Participants had dysfunctional eating behavior at baseline, with high scores on BES, UE and EE. No differences in treatment effects were detected between CBT and PED-t. Mean score reductions in BES, UE and EE were -9.0, -4.3, and -2.5 for CBT, and -10.4, -5.9, and -3.7 for PED-t, respectively. Compared to control, CBT reported greater reductions in BES and UE, whereas PED-t reported greater reductions in BES, UE and EE. Within group comparison detected reduced CR score (-1.4) in PED-t only. Higher post treatment intakes of protein, dietary fiber, and carbohydrate staple foods were reported in PED-t compared to CBT.

Conclusion:

This trial established support for CBT and PED-t in treatment of BN and BED patients, with no difference in treatment effect between the interventions. Compared to control, greatest improvements in eating behavior was found in BN-PED-t. BN-PED-t and BED-PED-t reported greater improvements in nutrient intake and food selection compared to CBT patients.

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Abbreviations

ED	Eating disorder
AN	Anorexia Nervosa
BN	Bulimia Nervosa
BED	Binge Eating Disorder
CBT	Cognitive Behavior Therapy
PED-t	Physical Exercise and Dietary therapy
BES	Binge Eating Scale
TFEQ	Three Factor Eating Questionnaire
CR	Cognitive restraint
UE	Uncontrolled eating
EE	Emotional eating
24-h recall	24-hour dietary recall
PA	Physical activity
EI	Energy intake
BMI	Body Mass Index
RMR	Resting Metabolic Rate
PAL	Physical Activity Level

1 Background

1.1 Eating disorders

1.1.1 Eating disorders

The American Psychiatric Association (APA) recognizes four main Eating Disorders (EDs) in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5), with the three former being Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Binge-Eating Disorder (BED) (1). The diagnostic criteria are presented in Appendix 1. EDs that do not fit these criteria are classified as either Other Specified or Unspecified Feeding or Eating Disorder (1).

EDs is a collective term for a group of psychiatric illnesses associated with severe medical, cognitive, emotional and social complications (2), being reported by 1-4% of European women (3, 4). Estimated prevalence's of BN and BED are higher than AN (5, 6). Further on, this paper will only focus on BN and BED.

Median age of onset is somewhat younger for BN than BED, but both disorders appear in the late teens to early 20s (7, 8). The lifetime risk for having either BN or BED is higher among women than men (7), but studies indicate that EDs are more common among males than previously thought (3, 9). Despite this, this paper will only focus on the female population.

Reported lifetime prevalence's for BN and BED among Norwegian women is 1.6% and 3.2%, respectively (10), supported by updated surveys and reviews (3, 7). In 2002, around 18 000 (BN) and 28 000 (pathological overeating) Norwegian women needed treatment, the majority in the age of 20-34 years (11).

1.1.2 Risk factors for onset of EDs

Findings from genetic epidemiology conclude that BN and BED are heritable conditions, where both genes and the environment contribute to the risk of development (12). Instead of looking at single risk factors for development of EDs, the etiology is divided in predisposing, precipitating, and maintaining factors (13, 14). Predisposing factors include body dissatisfaction, depressive symptoms, dieting, familiar relationships, childhood obesity,

environmental pressure and expectations, and genetics (3, 15). A history of ED in either parent, parental mental illness, prenatal maternal stress, and high parental education are all factors associated with higher risk of ED in the offspring (3). Precipitating factors, or triggers, cover conflicts, bullying, early onset of puberty, a high pressure related to performance, changed living conditions (moving, studying abroad), injuries among athletes, and dieting (16). Maintaining factors include familiar conflicts derived from the ED, negative comments or reactions from the environment, psychological symptoms of malnutrition (depression, fatigue), and a favorable self-perception of symptoms, such as being extraordinary, reduced tension and inner turmoil, and amenorrhea (16). Also, clinical perfectionism, low core self-esteem, mood intolerance, and interpersonal difficulties, are maintaining factors of EDs (17).

In community samples, body dissatisfaction or dieting and unhealthy weight control methods has been reported by 24%-77% of adolescent girls (18). Among Norwegian 15-year old adolescents, 49% of the girls find themselves too fat, and 32% of them engage in dieting behaviors (19). These are worrying numbers because dieting behavior increases the risk of extreme weight loss behaviors and subsequent EDs (20).

1.1.3 ED characteristics

Although diagnostic distinctions are made, several symptoms are shared across the EDs, such as disrupted body image and eating patterns, body dissatisfaction, weight concerns and over evaluation of body shape and weight (2, 5). The tendency to overeat, especially among BN and BED patients, will reinforce the preoccupation with body weight and shape and create a vicious circle (21). People with EDs judge themselves largely, or exclusively, based on their shape, weight or eating habits (22). Their lives become focused on their ability to control these domains of life. Along with the predominant behavioral aspect, there is also a central dysfunctional system for evaluating self-worth among ED patients (22).

Studies indicate that most patients migrate among diagnostic categories over time (23, 24), proposing that common ‘transdiagnostic’ mechanisms may be involved in the maintenance of the EDs (25). Research suggest that between 25-56% of BN patients have a history of AN (26-28). In a US community sample with an 8-year follow-up, high transition rates from subthreshold BN to BED (23%) and subthreshold BED to BN (22%) was reported among female adolescents (8).

1.1.4 Characteristics of BN

BN is a disorder characterized by recurrent episodes of binge eating and inappropriate compensatory behaviors to prevent weight gain (1). A binge eating episode is defined as a discrete period of time with consumption of larger amounts of food than most healthy subjects would eat, accompanied with a sense of lack of control (1). BN subjects may use different forms of compensatory purging behavior, such as self-induced vomiting, misuse of laxatives, diuretics, or other medications, fasting, or excessive exercise. According to DSM-5, these episodes of binge eating and purging both occur, on average, at least once a week for three months, but there is wide variability in these types of behavior (29). Individuals with BN unwarranted base their self-evaluation on body shape and weight (1).

1.1.5 Complications related to BN

The mode and frequency of purging are deciding factors for the medical complications of BN (29). Complications associated with excessive vomiting are dental caries and gastroesophageal reflux (29). Abuse of laxative drugs may lead to severe constipation. Recurrent purging behavior, such as vomiting, and misuse of laxatives and diuretics, can also result in serious fluid and electrolyte disturbances (29). Compensatory behaviors are associated with metabolic alkalosis, metabolic acidosis and hypokalemia (30). Hypokalemia is a predisposing factor for cardiac arrhythmias, and may affect metabolic parameters that increases the risk for developing type-2 diabetes (29, 31). BN patients are also at a higher risk of heart attack and stroke (7). Mehler et al. reported a normal bone mass density (BMD) in BN patients, unless they have a former history of AN (29). However, recent data show that BN results in lower BMD in women compared to healthy controls (32). Hormonal alterations, energy deficit and changed body composition may contribute to reduced BMD (32). A combination of excessive exercise and a restrictive diet, and thereby low energy availability, increases the risk of low bone mineral density, and over time osteoporosis (33, 34). Up to 50% of BN patients have irregular menses, but the future ability to conceive is not affected in recovered BN patients (21, 35). Alcohol abuse is reported by 50% of BN patients (5).

1.1.6 Characteristics of BED

Diagnostic criteria for BED include recurrent episodes of binge eating, without any purging behavior (36). A binge eating episode is defined similarly as for BN, but also include three or more of five additional features (see Appendix 1). As for BN, the binge episodes occur at least once per week for three or more months (1). The binge episodes must also be associated with a sense of loss of control and marked distress (1). Contrary to the DSM-5 diagnostic criteria for AN and BN, overvaluation of body shape and weight is not included for BED, despite its association with increased distress (37).

1.1.7 Complications related to BED

Morbidity rates are higher among BED patients than healthy individuals (38). BED is associated with obesity and related complications such as type-2 diabetes and cardiovascular disease, psychiatric co-morbidity, reduced quality of life and impaired social functioning (38). An assessment of the lifetime prevalence for mood, anxiety and substance use disorder among BED patients found rates of 54.2%, 37.1%, and 24.8%, respectively (39). This is supported by a longitudinal study of mental and physical health in Norway, using data from a population-based twin registry (40). Binge eating was associated with overweight, obesity, symptoms of anxiety and depression, reduced life satisfaction, and higher rates of psychiatric treatment (40). There were also higher rates of insomnia and early menarche among binge eating women (40). About 25% of women with BED have irregular menses (21). In addition, individuals with BED have higher levels of relationship distress, and impaired social functioning compared to healthy individuals (41). Compared to obese individuals without BED, obese individuals with BED report poorer psychological functioning, that is higher depression rates, lower self-esteem, and higher rates of emotional eating and shape and weight concerns (36). BED is also associated with several medical comorbidities, including sleep disturbances, type 2 diabetes, metabolic syndrome and gastrointestinal distress (36). The association with metabolic syndrome is independent of BMI (42).

1.1.8 Comorbidity and metabolic disturbances

Many patients with EDs meet lifetime criteria for other DSM-5 disorders (5). Such comorbidity is reported by 84.4% and 79.0% of respondents with BN and BED, respectively (7). Compared to healthy individuals, women with BN and BED report higher rates of

psychiatric comorbidities, such as depression, anxiety, and psychosocial stress, compared to healthy individuals (43-45).

BN and BED are associated with various medical complications (7). Lower fasting levels and stunted release of the satiety hormone cholecystokinin (CCK), disturbances in taste receptors and reduced taste sensitivity is found in BN patient (46). As a result, they may continue eating even after a substance has become less pleasant (46), or consume greater amounts of food than control, despite similar levels of satiety and hunger (47). Other metabolic disturbances found among BN patients is larger gastric capacity (48), increased release of insulin and hypoglycemia after binge eating and vomiting (49). Together, this might increase appetite, maintain problematic eating patterns, and reinforce repeated cycles of binge eating and compensatory behaviors (46). Some studies suggest that BED patients may have metabolic responses comparable to obese individuals, given the high prevalence of obesity among this patient group (3, 7).

BN and BED patients have increased risk of developing celiac disease (3), and several studies find that both BN and BED are associated with type-2 diabetes, musculoskeletal pain, hypertension and ulcers (3, 7, 41, 44, 50).

1.1.9 Weight

Typically, BN has been associated with mean weight within the normal range, whereas BED has been related to obesity and overweight (7, 51). However, recent reviews found an association between both BN and recurrent binge eating, and overweight and obesity (9, 52). Both patient groups seem to have significant higher BMI than healthy subjects (3, 7). Around 40% and 80% of individuals with BN and BED are obese, reported in community and clinical samples, respectively (7, 9, 53). Lower BMIs among BN patients is associated with a history of AN (54). See Table 1 for classification of overweight and obesity.

Table 1 – Classification of overweight and obesity by BMI*

Classification		BMI, kg/m^2
Underweight		< 18.5
Normal		18.5 – 24.9
Overweight		25.0 – 29.9
Obese		> 30.0
	Class I	30.0 – 34.9
	Class II	35.0 – 39.9
	Class III	> 40.0

*Adapted from Flegal et al. (55); BMI: Body Mass Index.

Rising rates of overweight is reported in BN samples, and normal weighted BN and BED patients tend to gain weight as time passes (7, 56). Weight gain seem to be greater among individuals who are overweight at the onset of the ED (57). Community surveys have found increased rates of combined obesity and ED behaviors (58, 59). These findings highlight the need for the development of therapies that address both the ED pathology and excessive weight, especially when they co-occur (52).

1.1.10 Weight regulation

Binge eating is suggested as a precursor to developing obesity (60). However, studies have found reduced energy requirements for weight maintenance among BN patients compared to matched control subjects without BN (61-63). Decreased energy expenditure is suggested a metabolic adaptation to extreme dietary restriction and semi-starvation in BN patients, and bingeing and compensatory behaviors may serve to elevate this lowered metabolism (64). Interestingly, an association between active bingeing and purging and low resting metabolic rate (RMR) is reported in a BN sample (65). However, abstinence from this bulimic behavior decreased the metabolism even further (65). Thus, BN patients may experience weight gain as normal eating patterns are introduced and bulimic behavior is reduced (62, 65). As this patient

group is very focused on body weight and shape, this might be an obstacle to change during treatment (65). Existing research on BED samples have not found enough evidence for changes in metabolism in this patient group (64). There seem to be no differences in metabolic rate between BED patients and control subjects (66, 67).

1.1.11 Seeking treatment

In a sample of lifetime BN and BED patients, the majority received treatment for emotional problems (7). However, only 47.4% and 38.3% of BN and BED cases, respectively, ever received treatment specifically for their ED (7). Between 80-94% of BN and BED subjects never seek professional help, or delay it for about 4-5 years (68, 69). Obstacles to seek treatment may be shame, denial, refusal for change, or lack of motivation (16). As a result, up to 90% of those with an ED may go untreated (70). Early detection and treatment of ED patients may be of high importance, given that dieting is a risk factor for BN, and a longer duration of bulimic behaviors is associated with more severe BN (71-73).

1.2 Treatment

1.2.1 Cognitive behavior therapy

Cognitive Behavior Therapy (CBT) is the leading evidence-based treatment for BN in adults (25), and is recommended by Norwegian and international guidelines for BN and BED patients (5, 74, 75). Currently, no other treatment is examined more than CBT considering treatment of binge eating (21). CBT is associated with clinically significant improvements during treatment as well as good maintenance in the longer term (46, 76).

Evidence suggest that CBT is as effective as, or more effective than, various forms of psychological treatments, and interpersonal psychotherapy (IPT) and guided self-help are the current leading second-line treatments (22, 74, 77, 78). IPT has proven comparable eventual effects as CBT, typically after 8-12 months, despite slower initial effects (79).

Dr. Christopher Fairburn described the CBT theory for EDs in 1981, and developed a treatment manual published in 1993 (80). The primarily intention was to treat BN (CBT-BN), but the treatment has been reformulated to a 'transdiagnostic' model to fit all types of binge eating (81). A more complex form of CBT is reserved for patients with marked additional psychopathology (25, 81).

BN and BED patients tend to restrict their eating, and adhere to their own extreme, and highly specific, dietary rules. Subjectively breaking these rules is interpreted as a lack of self-control, with consequently episodes of uncontrolled overeating. The almost inevitable slips encourage a dietary pattern with greater dietary restraint and thus an increased risk of binge eating and maintenance of the core psychopathology (22, 25). Binge eating as a distraction and a way of coping with different mood states, and wrong beliefs according to the effectiveness of compensatory behaviors in terms of weight management, may maintain the binge eating behavior (25).

CBT is designed to overcome dysfunctional eating; that is consuming too few calories, skipping meals, and eating only rigidly restricted types of food (22). Treatment is directed at reducing dietary restraint, by introducing forbidden foods and increase the overall amount of food, and establish more appropriate eating patterns (46). CBT also addresses the patients´

inability to cope with situations that trigger binge eating and compensatory behaviors, and aims for reduced concerns about body weight and shape (22, 46).

BED patients show significantly less dietary restraint than BN patients, and tend to overeat even between binge episodes (46). For this patient group, reducing calorie intake has positive effects in reducing binge eating (46, 82). In contrast to CBT-BN, dietary restriction is essential in treatment manuals for BED, given the importance of weight loss or prevention of weight gain (46).

The CBT theory is designed not to focus too much on calories and composition of food (46). Patients are discouraged from counting calories or fat grams, or precisely measuring or weighing food, and the therapists are instructed to use approximate terms (80). The purpose is to allow the patients a certain margin of error in choosing their food intake (80).

1.2.2 Effects of CBT

CBT is associated with significant improvements in all specific features of BN; that is binge eating, purging, dietary restraint and abnormal attitudes about body shape and weight, and reduced ED psychopathology (46, 76). Both group and individual administration of CBT have shown decreases in binge eating, vomiting, misuse of laxatives, extreme exercise, drive for thinness and body dissatisfaction (83). Among completers of the CBT-BN, between 40-50% cease binge eating and purging (22). Group-based CBT reduces both the frequency and severity of binge eating behavior, and improves body image and self-esteem among obese women who binge eat (84). CBT has also been shown to be effective in terms of reducing bingeing, purging and dietary restraint in BED samples (79, 85). In contrast to cases of BN, these effects do not seem superior to those of alternative treatments such as IPT and traditional behavioral weight loss management (77, 86). CBT alone do not appear to produce weight loss in ED patients and overweight individuals (25, 87)., but some studies indicate effect on body weight when CBT is combined with medication (76, 88) or behavioral weight loss treatments (BWLT) (85, 89).

Among treatment completers, 40%-50% cease binge eating and purging behavior during CBT (22), whereas up to 50% of participants report no effect at all (90). According to the Norwegian, national guidelines for EDs, 494/1000 and 349/1000 of BN and BED patients, respectively, do not achieve any remission from CBT (5). ED patients are a heterogenous

group, and individuals may therefore need different approaches to treatment. For a subgroup, CBT may be unnecessarily intensive or not sufficient, and less intensive programs or self-help models can successfully be provided (91). Opposite, a complex treatment approach, with a combination of CBT with other treatments is needed for some patients, mainly those with additional psychopathology (81). A combination of CBT with other treatments approaches is likely necessary for some, if not most, patients (81).

1.2.3 Outcome predictors

Factors found to predict less likelihood of abstinence following CBT treatment, are high baseline frequency of binge eating and longer duration of illness (83), higher baseline psychopathology, greater weight and shape concerns, and extreme dietary restraint (92).

On the contrary, a moderate dietary restraint is associated with better treatment outcome (92). Also found to be favorable is a lower baseline frequency of binge eating and purging, and reduction of these behaviors during the initial weeks (79). A rapid response, defined as ≥ 65%-70% reduction in binge eating by the end of the fourth session, is associated with higher rates of abstinence immediately after weight loss treatment (92). Similar, Fairburn found the best predictor of favorable outcome in treating BN patients to be early change in the frequency of purging (93). As a result, Fairburn recommends two weekly sessions in the initial four weeks of treatment in his CBT manual (22). In addition, a young age at disease onset is found to predict favorable improvements (87).

1.2.4 Nutrition therapy in treatment of EDs

Nutrition therapy, or dietary counseling, is defined as "a process by which a health professional with special training in nutrition helps people make healthy food choices and form healthy eating habits" (94). Nutritional knowledge for ED patients may be provided by various health care professional, but registered dietitians are considered the best qualified and trained providers (95). Factors that affect food intake, such as lifestyle, interpersonal relationships, body image, self-esteem and level of physical activity, have to be considered by the providers during treatment (96).

Like CBT programs, nutrition therapy aims to establish a consistent eating pattern and a normal eating behavior, and introduce participants to forbidden foods (46, 75, 96). Calorie

counting, weighing of food portions, and the use of exchange list are features of nutrition therapy that have proven effect in treatment of obese BED patients (46).

Nutrition therapy is found to be effective in terms of producing rapid improvement in general eating behavior and reducing binge eating frequency (97). BN patients have demonstrated greater reductions in BMI, and frequency of binge eating and compensatory behaviors during a 6-session Healthy Weight Program (HWP) compared to controls (98). Unlike CBT, this intervention encouraged calorie restriction and weight management, using drive for thinness as a motivator (98). Healthy weight loss treatments and dietary restriction do not seem to maintain the EDs in BN and BED patients, and studies demonstrate that nutritional therapy can be successfully combined with CBT for these patients (82, 98-100).

1.2.5 Physical activity in treatment of EDs

Regular PA is associated with reduced obesity and osteoporosis, improvements in ED factors of self-esteem, body image, anxiety, depression and negative mood, and enhanced interpersonal relations (101). However, PA is not established as a treatment for ED (101, 102). Reviews reveal limited literature on PA in BN and BED (103, 104). Reasons may be that health care professionals believe that increased calorie expenditure is an obstacle for weight recovery, or that excessive exercise is associated with increased ED symptomatology (101, 102, 105).

Estimated prevalence of excessive exercisers among female ED patients are 39%, and as much as 43.5% among those with lifetime diagnosis of BN (106). Excessive exercise can be defined as more than 3 h/day, exercising despite injuries or illnesses, and/or distress when unable to exercise (106). Reduction of exercise dependence score is associated with lower ED severity and improved health-related quality of life (105, 107, 108). PA can be an alternative strategy to regulate negative affects among excessive exercisers with longstanding ED (107).

However, PA has many beneficial effects. BN and BED patients engaged in regular PA has reported less ED pathology, body dissatisfaction, binge frequency and drive for thinness compared to patients in CBT only (100, 103, 104, 109, 110). Moreover, regular exercise may reduce negative mood, a trigger for bingeing and purging, and improve well-being and self-image (100, 111). Also, a greater sense of achievement, perceived body attractiveness, and

improved physical health related quality of life is associated with reduced ED psychopathology (101, 112).

Therefore, ED patients could benefit from the promotion (when low or not existing) or moderation (when excessive) of exercise (101). In addition, being given the opportunity to exercise increases overall compliance during treatment, including adherence to meal plans (104).

PA programs may have a positive effect on weigh reduction, and modest weight loss is reported independent of the effect of caloric restriction (113). Inclusion of exercise components to weight loss interventions are proven to be more effective for long-term weight loss in overweight subjects than dietary instructions alone (114). Engagement in PA can reduce emotional eating and increase flexible dietary control, and thus lead to weight reduction (115, 116). Acute bouts of exercise augment appetite regulation in both normal weighted and obese individuals, and do not cause increased energy consumption (117, 118).

1.2.6 Combined treatments

CBT may be superior to weight loss treatments regarding binge eating reduction (119, 120). However, CBT is not designed to assess weight reduction (52, 86, 92). Weight loss treatments reduce binge eating behaviors and body weight, but weight gain after treatment is common (119). Second, traditional weight loss treatments do not address the ED psychopathology (121). Therefore, overweight and obese ED patients could benefit from combined CBT and nutritional education (46, 54). Better results on achieving abstinence and completion of the study is reported with combined cognitive therapy and nutritional therapy for BN, compared to CBT alone (97).

Adding PA to CBT has shown efficacy for enhancing weight loss, or proved to be the only treatment reducing BMI (92, 103, 110). Moreover, exercisers report greater reductions in binge eating frequency and higher rates of abstinence during treatment (110). PA as an adjunct to CBT may also improve overall health, well-being and sustainability of outcomes (92, 112). PA has been found as a positive addition to CBT and nutritional treatment, by reason of decreased negative mood, improved ED and an effective weight loss among BED patients (122).

1.3 Eating behavior

1.3.1 Dysfunctional eating behavior

Eating behavior is a result of behavioral, cognitive and affective components (123). Cognitive processes may suppress physiological feelings of hunger and satiety, and such chronic self-control may result in disrupted eating patterns (123). In the American Psychiatric Association (APA) guidelines, normalization of nutrition and eating habits is highlighted as a central goal in the treatment of BN patients (75). Dysfunctional eating patterns can be separated into three factors; cognitive restraint of eating (CR), uncontrolled eating (UE), and emotional eating (EE) (124).

1.3.2 Cognitive restraint

Restrained eaters control their food intake in order to control their weight and body shape (125). The restraint theory suggest that effective self-regulation can prevent weight-gain in western 'obesogenic' environments, but dietary restraint may reduce sensitivity to internal cues for satiety (126). Among successful restrained eaters, certain disinhibiting events may interfere with self-control and cause overeating, and a binge-like eating pattern (123, 126). Consumption of subjectively forbidden foods and negative emotional states are examples of such disinhibiting events (123).

Higher scores on the CR scale are related to significant higher body weight in normal weight subjects, but are associated with lower BMI values in overweight individuals (126, 127). High restraint may serve as a marker for overeating tendencies among normal weight subjects, while lead to lower BMI values in obese individuals through reduced food intake (126). However, studies find an inverted u-shape relationship within in the overweight range, meaning that individuals scoring high or low on the restraint scale have the lowest BMI values (127, 128).

1.3.3 Uncontrolled eating behavior

UE reflects the tendency to lose control over food intake (125, 129). UE is a collective label for disinhibition and hunger, but many former as well as recent studies still use the term disinhibition (129-133).

Disinhibition is associated with higher BMI and obesity, and is found to be the best predictor of food consumption (130, 131, 134). Individuals with disinhibited eating are more likely to make unhealthful food choices, characterized by greater wanting for high-fat foods, and higher consumption of high-fat, high-salt and sweet foods, and processed meat (132, 133).

Research on the relationship between disinhibition and BMI during weight loss treatment report two main findings. First, decreased weight during treatment is related to decreased disinhibition (130, 135). Second, higher initial disinhibition scores tend to predict weight regain after treatment (130, 136).

1.3.4 Emotional eating behavior

EE is a behavioral response to emotionally charged feelings or situations, negative as well as positive (137). However, in obese individuals, episodic overeating is primarily a result of negative affective emotions (137, 138). Such negative emotions include, but is not limited to, depression, anxiety, loneliness, shame, anger and stress (137-139). Negative emotional states may affect UE behavior, as well as disrupt the cognitive control over food intake (125). Several theories suggest that binge eating is a strategy to avoid or regulate negative emotions, or occurs because of unsuccessful restricted eating (140). EE indicates unhealthy eating behaviors and attitudes, and is associated with unsuccessful attempts to reduce weight (141, 142).

1.3.5 Interactions between CR, UE and EE

Restraint interact with disinhibition, and may contribute to limited weight gain in periods of uncontrolled eating behavior (127, 143). The combination of low restraint and high disinhibition is associated with the highest BMI, high restraint and high disinhibition with lower BMI, while low restraint and low disinhibition is associated with the lowest BMI (130, 143).

Whereas UE is strongly related to greater body size and weight, disinhibited eating may also function as a moderator of the relationship between restrained eating and BMI (128). Dietrich et al. found that the inverted U-shaped relationship between CR and BMI remained the same at low levels of disinhibition. At high levels of disinhibition on the other hand, there was a positive relationship (127). The model indicates that with higher levels of disinhibition, subjects in the normal weight range successfully compensate with increased restriction, while overweight individuals fail to adapt their dietary restraint, and attempts to restrict food intake decrease (127).

Studies indicate a modulative relationship between the three eating behavioral domains (130). The highest BMI values is found among uncontrolled and emotional (but not restrained) eaters (mean BMI 29.6 kg/m^2), followed by subjects scoring high on all three scales (mean BMI 28.9 kg/m^2). Being neither an uncontrolled, emotional or restrained eater is associated with having the lowest BMI (mean BMI 25.6 kg/m^2) (128). These findings support the need of an overall knowledge about the combination of eating behavior scores in treatment of obesity (130).

1.3.6 Eating behavior among BN and BED patients

The knowledge about eating behavior and eating patterns among BN and BED patients is relatively limited, except for overeating and binge eating (144). The following will present findings from existing literature.

Whereas BN subjects tend to have higher scores on the restraint scale, individuals with BED have lower levels (145). In the case of BN patients, high CR may reflect their restrictive eating behavior between binge episodes (145). As extreme dietary restraint is related to severity of eating pathology and other EDs, CBT aims to reduce the restrained eating for this patient group (130, 146). Contrary, increased dietary restraint during treatment is a predictor of binge abstinence and weight control among BED patients, and low CR score is proposed as a main contributor to weight gain (145, 147, 148). Despite this, the CBT approaches for BED, is nearly the same as for BN, with reducing CR as the main target (80).

Higher disinhibition scores are related to BED, but have also been associated with BN (130, 149). UE score appears to be the primary factor associated with greater body size and weight

(130, 143). Reducing UE score has the potential to produce weight loss, improve ED symptomatology, and reduce binge eating in ED patients (130, 147, 148).

It has been suggested that BN and BED patients are unable to regulate emotion states effectively, and uses binge eating as an alternative strategy to cope with negative feelings (150-152). Thus, binge eating may serve as a temporary relief from emotional distress generated from high self-expectations and -standards (153, 154). Negative emotions are reported as the main reason for binge eating behavior by the majority of BN and BED patients, and self-rated negative mood preceding binge events is significantly greater compared to levels before regular eating situations (155, 156). Correspondingly, the belief that food can relieve negative emotions is correlated to binge eating frequency (157).

Interestingly, BED patients report higher levels of both pleasure and relaxation, and less physical discomfort during binge events than BN patients, and BN patients have reported their highest levels of negative emotions after bingeing (158). Contrary, feelings such as relief and excitement is reported after purging episodes, supporting that BN patients use purging as a strategy to reduce concerns about weight gain and negative affect following binge eating (159).

1.4 Eating pattern among patients with BN and BED

1.4.1 Energy intake

According to dietary intake data obtained by patients with BN and BED, these groups consume more energy than healthy control subjects (160-162). About 1000 kcal greater EIs than obese controls are reported among BED patients (66, 163). In comparison, BN patient consumed over twice as much (4446 kcal) during a 24-h period than controls (1845 kcal) (161).

Some studies report an average EI 800-1000 kcal higher on binge days compared to binge free days, based on self-reported food diaries for 7-28 days (66, 164, 165). For BN patients, an average intake of three times as much food as controls is reported during binge episodes (160). The frequency and size of meals seem to be normal in binge eating BN patients. However, 37% of the BN women's meals is found to be greater than 1000 kcal (161).

Some studies suggest that non-purging BN and BED patients eat less food than non-bingeing individuals on binge-free days (160, 164). Periods of highly restrictive eating behavior may function as compensation for overconsumption, to prevent weight gain (164). The theory was confirmed by Rossiter et al., as the average daily EI among non-purging BN subjects was 1500 kcal on binge-free days, compared to 2000 kcal among healthy controls (164, 166). These numbers are in line with an updated dietary survey among healthy, Norwegian women, where the reported, average daily EI was 1912 kcal (167).

1.4.2 Macronutrient composition

There are no currently recommendation for macronutrient distribution specific to the ED population (96). Surveys of dietary habits and food choices among BN and BED patients are important for knowledge and practical implications in the treatment of these EDs (99, 163, 164).

Binge eaters seem to choose more nutritious foods on binge-free days than during binge episodes (99, 164). Data from self-monitored food records in a female BN sample revealed higher intakes of protein and dietary fiber on non-binge days than during binge episodes (164). Studies show that during days of binge eating, BN and BED patients consume higher levels of fat and lower levels of protein than healthy subjects (99, 163, 164, 168). Yanovski et al. found no differences in macronutrient composition when comparing BED patients and healthy controls during normal meals (99). During binge meals, however, the BED women consumed a greater percentage energy from fat (38.9% vs 33.5%) and a lesser percentage energy from protein (11.4% vs 15.4%) than non-bingers (99). Similar findings were reported in a BED sample prior to a 6-month weight reduction program (163). The mean energy intake and the percentage of energy from fat on binge days was significantly higher than binge free days (3350 kcal vs 2695 kcal, and 39.9% vs 37.3%, respectively) (163).

During a 7-day period, BN and BED patients seem to have lower percentage intake of protein than overweight, healthy controls, with no differences in percentage intake of fat and carbohydrate (169). These findings differ from the hypothesis that bulimic patients compensate binge eating with periods of extreme restrictive eating, and that binge eaters consume greater proportions of fat and carbohydrate (46). Latner and Wilson conclude that there is no supporting evidence that BN patients have a higher-than-normal craving of

carbohydrates and carbohydrate-rich foods, and that these foods trigger and generates binge events (46).

1.4.3 Micronutrient deficiencies in EDs

Inadequate dietary intake of various nutrients may be a consequence of unbalanced eating patterns (170). Research show that patients suffering from mental disorders are often deficient in essential vitamins, minerals, and omega-3 fatty acids, and supplements may reduce symptoms for some patients (171). Among ED patients, avoidance of dietary fat or restrictive selection of food items can lead to nutrient deficiencies (170). Other ED patients may consume excessive amounts of certain products or have a very repeating eating pattern, resulting in high intakes of certain micronutrients (170). ED severity is associated with risk of micronutrient deficiency (172). In general, all ED patients have their own, unique dietary practices, making it difficult to draw a uniform instruction for nutrient deficiencies in EDs (170).

Purging behaviors such as self-induced vomiting and laxative abuse can cause dangerous micronutrient deficiencies such as hypokalemia, hypomagnesemia and hypophosphatemia (170). Hyponatremia may be a consequence of limited intake of sodium, or misuse of laxatives and diuretics (170). The average dietary intake of iron among 18-39-year old, Norwegian women is found to be 9.4-11 mg/d in a national survey, which is below the recommendation (167). Women in childbearing age have increased needs for iron due to menstrual bleeding (173). Meats have a high content of iron, whereas the majority of iron in the Nordic diet comes from cereal products (173). Setnick have reported that most ED patients have an insufficient intake of calcium (170). However, this will most probably not be reflected in serum calcium levels (170). As for age-matched, healthy women in the general, Norwegian population, female ED patients may consume inadequate amounts of folate and vitamin D (167). High prevalence of vitamin D deficiency and insufficiency are detected in ED samples (174). Modan-Moses found that only 16.7% of ED subjects had optimal serum levels of vitamin D, whereas 7.8% and 22.2% had vitamin D deficiency (<15 ng/ml) and insufficiency (15-20 ng/ml), respectively (174).

1.4.4 Food choice

Descriptions of dietary habits among ED patients often differentiate between “high-energy food” rich in carbohydrates and fats, and “low-energy foods”, low in carbohydrates and fats. The former is supposed preferred by BN and BED subjects (169). In addition, ED patients tend to have greater levels of uncontrolled eating behavior, which is associated with less healthful food choices (130, 175). Higher scores on the UE, and EE, scales correlate with less healthful food choices, such as food with high content of calories, fat, sugar, and salt, processed meat, ice cream and butter (132, 133, 138). In addition, disinhibition is found to correlate with decreased consumption of fruits and high-fiber bread (176).

Both when instructed to binge eat and during normal meals, BED subjects consume greater amounts of high-fat foods, such as cake, ice cream, potato chips, and butter, compared to control (99). It is demonstrated that fat consumption among binge eaters is negatively correlated with intake of fruits and vegetables (177).

Protein is found to be the most satiating macronutrient (178). After two 14-day periods with repeated-measures design, consuming either high-protein or high-carbohydrate supplementation, BN and BED patients reported greater feelings of satiety and reduced temptation to binge eat following the protein period (178). There is a possibility that a macronutrient composition with increased intake of protein during binge-free periods, may protect against disinhibited eating behavior and overconsumption (46). Dietary fat is often avoided by ED patients (96), but it is an essential part of a balanced diet. Although fat consumption is positively related to binge eating symptoms among women (177), inclusion of fat in the diet may reduce impulses to binge eat and prevent overeating (96).

1.4.5 Meal frequency

A comparable number of meals per day is reported between BN subjects and healthy controls (161). Contradictory, some studies find differences in meal frequency between BN and BED patients, and healthy controls (144). For example, BN subjects have reported significant fewer meals compared to BED and control, whereas BED subjects have reported a higher frequency of snacks per day (144). In the same study sample, more frequently meal consumption was associated with less binge eating, and breakfast frequency was inversely related to BMI in the BED group (144). Among BED subjects, those eating three meals per day reported lower

body weight and fewer binges than participants who did not regularly eat three meals per day (179). Individuals with the highest meal frequency have reported higher intakes of carbohydrates, folic acid, vitamin C, calcium, magnesium, iron, and dietary fiber, and lower intakes of dietary fat, cholesterol and sodium than less frequent eaters (180).

A regular eating pattern, defined as three meals and two-three planned snacks per day, is a central component of CBT. Adherence to such a regular eating pattern is associated with reduced binge eating behavior following a self-help program of CBT (181). Similarly, increased evening meal consumption was negatively correlated with binge eating frequency and purging behaviors in BN patient after a CBT program (182). Surprisingly, decreased mid-morning snack and lunch consumption predicted decreased purging behavior at 4-month follow-up, indicating variable influence on bingeing and purging by different meals (182).

1.4.6 The Norwegian dietary guidelines

Norwegian guidelines on diet, nutrition and physical activity are food-based dietary guidelines, and the key message is to "have a varied diet with plenty of vegetables, fruit and berries, wholegrain products and fish, and limited amounts of processed meat, red meat, salt and sugar". More specifically, the recommendations are to eat at least five portions of vegetables, fruits and berries each day, to eat wholegrain products each day, having fish for dinner two to three times a week, and consume a limited amount of processed meat, red meat and beverages with high sugar content. This dietary pattern has proven to protect against different lifestyle diseases, osteoporosis, dental caries and to maintain a healthy body weight (183). Appendices 2 and 3 present the Norwegian quantitative dietary recommendations, and selected guidelines for macro and micro nutrients, respectively.

The guidelines for PA is based on both Nordic and international guidelines, with focus on health promotion and disease prevention (184). For the adult population, the recommendation is to reduce sedentary behavior, and engage in at least 150 minutes of moderate-intensity, or 75 minutes of vigorous-intensity, or an equivalent combination of moderate- and vigorous-intensity activity per week. To achieve additional health benefits, the amounts should be doubled (184).

1.5 Aim and hypothesis

The aim of the study was to compare the effect of two different treatment programs; CBT and Physical Exercise and Dietary Therapy (PED-t), in women with BN and BED. Treatment effect was examined on

1) Eating behavior; binge eating, cognitive restraint, uncontrolled eating behavior, and emotional eating behavior.

2) Eating pattern; nutrient intake and food selection.

It was hypothesized that $H_{1\text{-}0}$: there is no difference in change of eating behavior (binge eating, cognitive restraint, uncontrolled and emotional eating) between the treatment groups CBT and PED-t, $H_{1\text{-}1}$: there is a significant different change in eating behavior between treatment groups, $H_{2\text{-}0}$: there is no difference in change of nutrient intake and food selection between the treatment groups CBT and PED-t, and $H_{2\text{-}1}$: there is a significant different change of nutrient intake and food selection between treatment groups.

2 Methods

2.1 Study design

This master project is a part of the PED-t trial at The Norwegian School of Sport Sciences lead by Ph.D. candidate Therese Fostervold Mathisen. The master student contributed to the PED-t trial by taking blood samples, and conduct 24-h recalls from a selection of participants. The randomized, controlled prospective study included women with BN and BED diagnosis, allocated to one of two treatment groups; CBT or PED-t. Both interventions had a structure of 20 group sessions during a period of 16 weeks, in groups of 5-8 participants. Participants who fulfilled inclusion criteria but were waiting for availability in one of the two treatments, served as a waiting list control condition. They answered self-reported questionnaires about eating behavior periodically during the 16-week treatment period. An overview of the PED-t trial and this master project are presented in Figure 1 and 2, respectively.

2.2 Participants

Of 419 eligible females recruited to the PED-t trial, 164 patients were included in this study. Participants were women aged 18-40 years with a body mass index (BMI) in the range of 17.5-35.0 kg/m^2, a DSM-5 diagnosis of BN or BED with duration of at least 3 months, and with mild to severe symptoms (minimum one episode of compensatory behavior or binge eating per week, respectively (185)). Inclusion and exclusion criteria are summarized in Table 2.

2.3 Outcomes

Demographic and clinical variables comprised age, body weight, body height, BMI, disease onset, disease duration, and history of anorexia nervosa (AN). The primary outcome measures were 1) group difference in changes in eating behavior as measured by the BES and TFEQ-R21, validated tools for characterizing eating behaviors (124, 129) and identify binge eating (186) in obese individuals, and 2) group difference in changes in nutrient intake and food selection as measured by the 24-h recall.

Table 2 – Inclusion and exclusion criteria of the PED-t intervention trial

Inclusion	Women between 18 and 40 years of age
	BMI 17.5-35 kg/m^2
	DSM-5 bulimia nervosa, mild and moderate severity, meaning 1-3 and 4-7 episodes of binge eating per week, respectively. Duration ≥ 3 months
	DSM-5 binge eating disorder, mild and moderate severity, meaning 1-3 and 4-7 episodes of binge eating per week, respectively. Duration ≥ 3 months
	Residential address ≤ 1.5 hour away from Oslo
Exclusion	Not available during the study period
	BMI > 35 kg/m^2
	Pregnancy or planned pregnancy the first coming year
	Athletes at national or international level
	Completed therapy in form of CBT for eating disorders the last two years
	Severe axis I and/or II mental disorder. Needing treatment/in treatment for severe depression, personal disorder, suicide issues, substance use, obsessive-compulsive disorder, or anxiety
Stop procedure during study period	Acute worsening of the eating disorder. Decision will be made in consultation with the participant's doctor/medical Director
	Weight loss ≥ 2 kg during one week for participants with an BMI < 19 kg/m^2 at inclusion

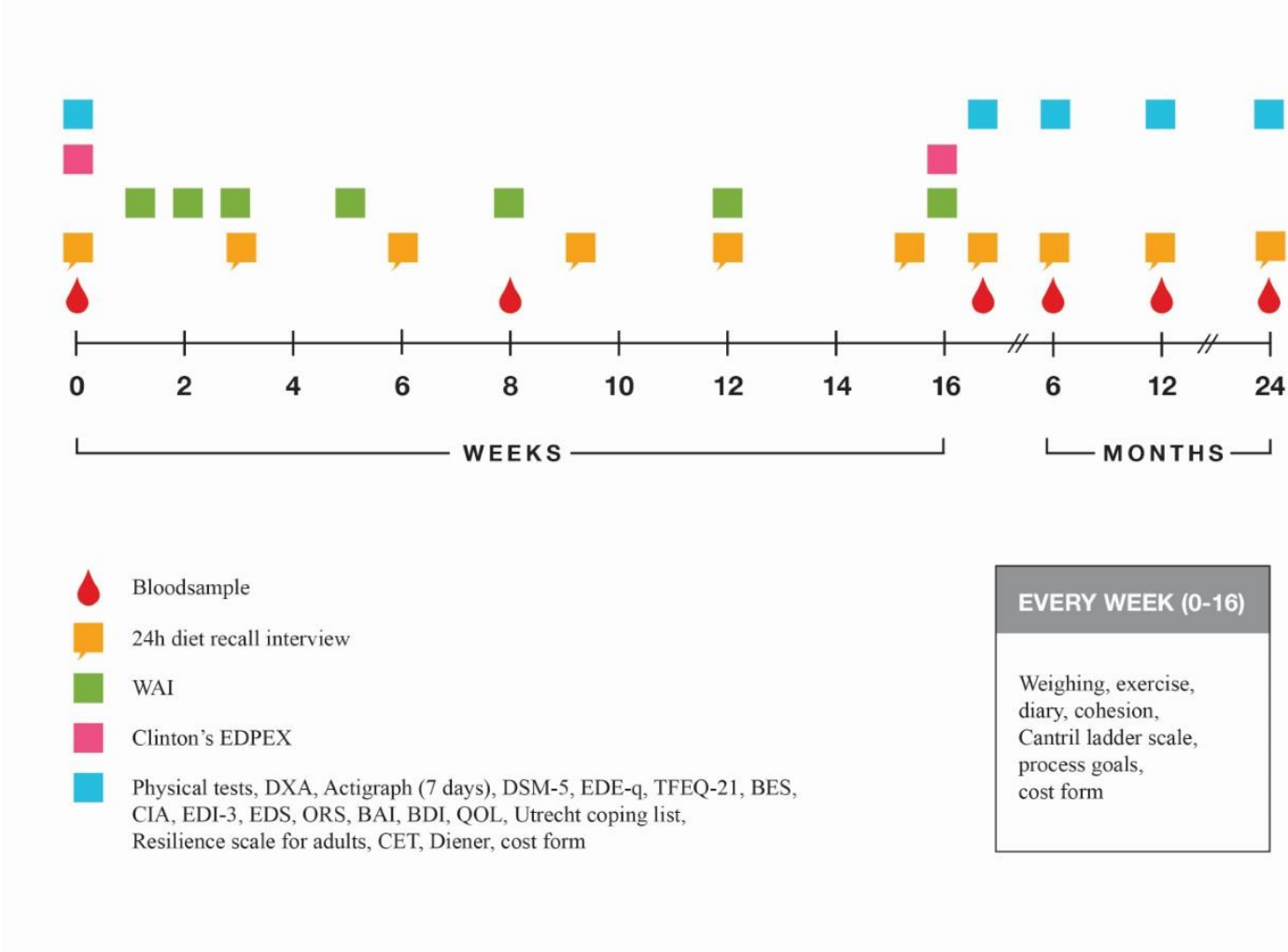

Figure 1 – Overview of the PED-t trial

WAI: Working Alliance Inventory; EDPEX: Eating Disorder Patients' Expectations and Experiences of treatment; DXA: Dual-energy X-ray Absorptiometry; DSM-5: Diagnostic and Statistical Manual of Mental Disorders, fifth edition; EDE-q: Eating Disorder Examination Questionnaire; TFEQ-21: Three Factor Eating Questionnaire-21; BES: Binge Eating Scale; CIA: Clinical Impairment Assessment; EDI-3: Eating Disorder Inventory-3; EDS: Eating Disturbance Scale; ORS: Outcome Rating Scale; BAI: Back Anxiety Inventory; BDI: Beck Depression Inventory; QOL; CET: Compulsive Exercise Test.

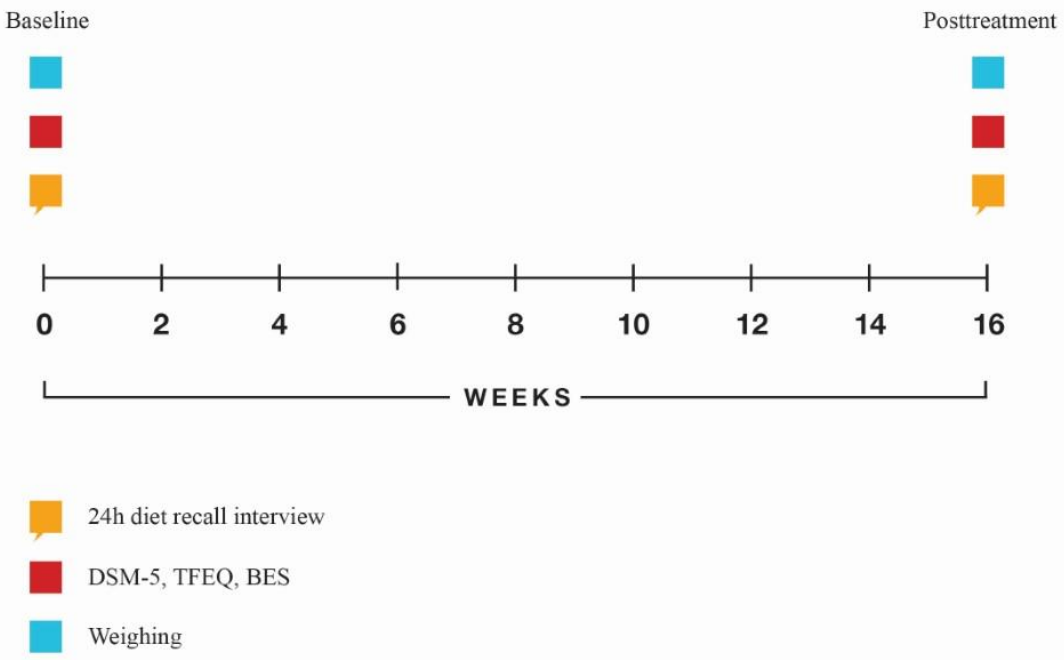

Figure 2 – Overview of the master project

DSM-5: Diagnostic and Statistical Manual of Mental Disorders, fifth edition; EDE-q: Eating Disorder Examination Questionnaire; TFEQ: Three Factor Eating Questionnaire; BES: Binge Eating Scale.

2.4 Recruitment

Participants were recruited by general practitioners (GP) and psychiatric centers in Oslo, social media, websites, newspapers, magazines and posters at the University of Oslo and University Colleges in Oslo and Akershus. Interested participants contacted study employees for more information about the study; more specifically about purpose, recruitment and randomization, treatment, voluntary consent and the opportunity to withdraw at any time during the treatment period. In parallel, the study employees considered eligibility according to criteria. Participants underwent a telephonically screening interview with Eating Disorder Examination Questionnaire (EDE-q) and the Mini-International Neuropsychiatric Interview (MINI). Final inclusion was based on three written and signed declarations, by the women themselves, and a signed letter from the women's GP confirming their suitability.

2.5 Randomization

Baseline measures were completed before randomization. Randomization was completed through a block design generated via http://www.randomizer.org. A randomization list with block size 8 was generated. Scientists and test personnel were blinded before the randomization. Therapists and participants were not blinded to treatment allocation. Participants were informed about group allocation after completion of the baseline measurements.

2.6 Interventions

Both CBT and PED-t groups underwent 16 weeks of treatment, consisting of 20 sessions. The treatments were led by physical trainers and dietitians, all holding a master degree in sport sciences. They did also follow each participant throughout the study, meaning keeping records over participation, homework and submissions. After the intervention period, participants were followed at 6, 12 and 24 months' post treatment.

2.6.1 CBT

The treatment manual was based on the transdiagnostic model of CBT for ED-patients, modified to a group format (25, 187). As well as the generic core ED-pathology, the treatment aimed to address additional mood intolerance, clinical perfectionism, core low self-esteem,

and interpersonal difficulties. The treatment sessions were run by psychologists with experience with CBT and EDs. All CBT sessions, lasting for about 90 minutes and conducted at The Norwegian School of Sport Sciences, were divided into 4 stages. The first 4 weeks addressed the ED and attempted to engage and educate the participants, and to ensure early behavior change. During the next 2 sessions, the groups evaluated the progress so far, and identified any barriers to change. Stage 3 was the largest part of the CBT treatment, and occupied 10 sessions. Focus was to modify the core psychopathology of ED, that is over-evaluation of eating, shape and weight, extreme dieting, binge eating and purging. The emphasis of the final stage of treatment, sessions 17-20, was maintenance of progress and relapse prevention. The concept of relapse intervention was to give the patients different strategies to avoid falling back into an unhealthy eating pattern and further binge eating. The therapist will explain relapse as a three-stage model, where the group members can deal with the situation, and not as an all-or-nothing view (80). Table 3 provides an overview of the contents of all sessions in the CBT treatment arm.

Table 3 – Overview of the content of the 16-week CBT treatment

Stage	Therapy session	Targets	Main content
1	1-4	Engagement, preparation and early behavior change	Educate about the nature of CBT and how the therapist and the participants work together Engage the participants in the treatment Develop a case formulation for each participant Strategies to take control over the behavioral symptoms of BN and BED

2	5-6	Monitoring and evaluating progress and barriers to change	A detailed review of progress so far, and to identify barriers to change
3	7-16	Modifying the core pathology of ED	Reduce the over-evaluation of weight and shape Address extreme dieting, binge eating and purging
4	17-20	Consolidating change and relapse prevention	Secure that progress is maintained after treatment end

2.6.2 PED-t

The PED-t intervention is a treatment method developed at The Norwegian School of Sport Sciences, designed by the research group engaged in the trial, particularly to treat BN and BED. The manual was based on recent dietary recommendations (184) and guidelines for guided physical exercise (173, 188-191), and aimed to achieve and stabilize a normal body weight and a healthy body image. Focus was to (re)establish healthy routines, educate about harmful effects of unhealthy behavior and to change focus from body appearance to the body's functionality. Knowledge about basic and sports related nutritional needs was given (31). The sessions consisted of 45-60 minutes of physical exercise (PE) and 45-60 minutes of dietary therapy, conducted at The Norwegian School of Sport Sciences. All participants received this combined treatment, and therefore the effects cannot be separated for physical and dietary therapy.

Physical exercise

The PE program consisted of resistance training combined with aerobic interval training to increase maximal strength and aerobic capacity. With a frequency of three weekly sessions, two sessions were resistance exercise of which one was supervised. The third session was unsupervised interval running. The PE module is supervised by personnel educated in exercise science at the Norwegian School of Sport Sciences. A detailed overview of the

exercise module of the PED-t treatment arm is described in the study protocol (31). This master project does not have results directly related to physical fitness.

Dietary therapy

Table 4 provides an overview of the contents of all sessions in the dietary module of the PED-t treatment. The dietary module translated Norwegian guidelines on diet (184) into practice. These guidelines are based on the 'Food-based dietary guidelines for public health promotion and prevention of chronic diseases – Methodology and scientific evidence', published by Norwegian Nutrition Council in 2011 (192) and Nordic Nutrition Recommendations 2012 (NNR 2012) published by Nordic Council of Ministers in 2014 (173). Each session was a combination of knowledge dissemination from a dietician, and a participant controlled part based on experiences from last session and topics they wanted to or needed to discuss. Focus was to (re)establish healthy dietary routines. The first module of the dietary therapy (sessions 1-5) targeted dietary routines and structure, and aimed to educate participants about a healthy meal frequency and portion size, to handle specific eating situations, and enlighten them as to exercise theory. The next module (sessions 6-17) covered nutritional knowledge about energy needs, nutrients and sports nutrition (193), as well as practical skills for daily routines, nutritional labels and food shopping. The final module (sessions 18-20) was reserved reflections, repetition and summary, and formulation of an individual plan regarding exercise, diet and daily routines. Two main targets of the nutritional guidance were to achieve complete meals and secure dietary quality. Based on a priori, professional knowledge, focus was directed away from fats to prevent fear of energy dense foods. Rather than specific amounts of foods and calories, the program communicated methods for creating a healthy, balanced diet. Keywords were 4 daily main meals, the need for snacks, to separate main meals from snacks, that two hands can represent a normal portion size, and how to use the plate model. In addition, there were some detailed nutritional information about the need for carbohydrates and proteins, fat quality, calcium and vitamin D, iron and vitamin C. In addition, participants got homework and submissions throughout the period, i.e. registration of the meals consumed between each weekly session, and work on individual tasks.

Table 4 – Overview of the content of the dietary module of the 16-week PED-t treatment arm

Session	Theme	Main content
Dietary routines and structure		
1	Meal frequency	Overall goal of four meals a day during the intervention
2	Portion size	Achieve sufficient portion sizes for main meals
3	Eating situation	Where do you eat, and what do you do while eating
4	Exercise theory part I	Training quality and restitution
5	Lookback and repetition	
Nutritional knowledge and practical skills		
6	Energy	Estimate calories, total energy needs and resting metabolism
7	Daily routines	What do you prioritize, which stress level are you at
8	Carbohydrates	Talking about the topic and looking at participants' meal pictures
9	Diet and exercise	Food and energy before, during and after physical exercise
10	Fat	Talking about the topic and looking at participants' meal pictures
11	Proteins	Talking about the topic and looking at participants' meal pictures
12	Packaging	Reading and understanding

13	Practical session: grocery shopping	Participants are given practical tasks to buy food on impulse
14	Exercise theory part II	
15	Iron and vitamin C	Talking and looking at participants' own meal pictures
16	Calcium and vitamin D	
17	Mental focus and summary	How to handle adversity and to be aware of your own view on existence Summary of sessions so far
Summary of future plans		
18	Individual summary	Reflections, repetition and summary
19	Summary	Experiences and dividends
20	Presentation	Presentation of their own mastery plan (exercise, diet, daily routines)

2.7 Measurements

Eating behavior was measured on group level by The Binge Eating Scale (BES) and the 21-item version of The Three Factor Eating Questionnaire (TFEQ-R21). Eating pattern was measured on group level by 24-h dietary recalls (24-h recall). These measurements and procedures are presented below. Weight monitoring was not included in study aims. However, weight change was considered important for discussion of treatment effects, and is presented as final results.

2.7.1 Eating behavior

The Binge Eating Scale

The Binge Eating Scale (BES) is a 16-item self-reported questionnaire developed by Gormally et.al. to identify binge eaters and assess binge eating severity among obese persons (186, 194). The questionnaire describes a binge episode in terms of both behavioral (e.g., lack of impulse control, or eating continuously without regular meals) and emotional expressions (e.g., feeling ashamed about their weight, or guilty when eating too much) (194). BES is proved to be a valid instrument to identify BED in obese women seeking weight loss treatment (186). However, BES is not a diagnostic tool.

Examples of items and statements in the BES questionnaire are:

1

I eat the same around friends and family as I do when I am alone

Sometimes I do not eat what I want around others because I am aware of my problems with food

I often eat little around other people because I feel embarrassed

I'm so ashamed of overeating, I only eat at times when no one sees me. I eat in secret

2

I eat three meals a day and occasionally a snack

I eat three meals a day and I usually snack as well

I eat many meals, or skip meals regularly

There are times when I seem to eat continuously without regular meals

Each statement has a scoring weight from 0 (indicates no binge eating problems) to 3 (reflects severe binge eating problems), and the total score vary from 0-46 points (194). Marcus et al. created a cut-off for three different levels of binge eating severity: non-bingers scoring 17 and less, moderate bingers scoring between 18 and 26, and severe binge eaters scoring 27 and above (195).

Scale score was computed when the participant had answered at least one-half of that scale's items (196). See Appendix 4 for the BES.

The Three Factor Eating Questionnaire

The Three Factor Eating Questionnaire (TFEQ) is a self-reported questionnaire used to assess eating behavior in overweight and normal weight individuals (124). The original 51-item TFEQ, developed by Stunkard and Messick in 1985 (197), measured three dimensions of human eating behavior; cognitive restraint (CR), disinhibition and susceptibility to hunger (197). The CR scale measured restriction of food intake to control body weight and shape, or to promote weight loss. Disinhibition reflected episodes of loss of control over eating, while the hunger scale reflected subjective feelings of hunger and food cravings (129). A Swedish study on obese samples tested the construct validity of the TFEQ and aimed to create a more efficient questionnaire through item reduction (129). The CR scale demonstrated robust validity, while disinhibition and hunger were grouped into one factor, entitled uncontrolled eating (UE) (129). In addition, the study identified a scale on emotional eating (EE). EE concerns the probability to overeat in relation to negative mood states, such as when feeling anxious, depressed, or lonely (129, 198). The shortened and revised 18-item version (TFEQ-R18) has been replicated and validated in obese as well as normal weight subjects (129, 199).

The later refined version TFEQ-R21, with three more items added to the EE domain, has also shown good reliability and validity (124, 200).

In the present study, the TFEQ-R21 questionnaire was used. It is a 21-item instrument with 20 four-point Likert scale items and one eight-point numerical rating scale. Each of the items are given a score between 1 and 4, and raw scale scores can then be calculated. Higher scores in the respective scales indicate greater CR (6 items), or susceptibility to UE (9 items) or EE (6 items).

Example of a statement to assess CR are

I consciously hold back at meals in order not to weight gain

Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

UE is assessed by a statement such as

I am always hungry so it is hard for me to stop eating before I finish the food on my plate

Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

EE is assessed by a statement such as

When I feel lonely, I console myself by eating

Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

Scale score was computed when the participant had answered at least one-half of that subscale's item (196). Possible score ranges and preliminary cut-off values (not validated) for the TFEQ-R21 are presented in Table 5. See Appendix 5 for the TFEQ-R21.

The mean score for each of the TFEQ-R21 (CR, UE, and EE) scales, and the total score for the BED, were calculated. The data were then used to find the between- and within-group difference in scores between baseline and post treatment.

Table 5 – Score ranges and preliminary cut-off values for the TFEQ-R21

	Possible score range	Normal eating behavior	Extreme eating behavior
CR	6-24	6-17	18-24
UE	9-36	9-24	25-36
EE	6-24	6-17	18-24

CR: cognitive restraint; UE: uncontrolled eating behavior; EE: emotional eating behavior.

2.7.2 Eating pattern

24-hour dietary recall

The 24-h recall is an open-ended survey, proved to be a valid method for subjective estimates of food consumed over the previous 24 hours (201). It typically requires 20-30 minutes to collect detailed information about foods, ingredients, brand names and preparation methods for a single day (201). Unannounced 24-h recalls are proved to be preferable to food records in terms of collecting dietary data information (202). The amount of food is estimated using standard measuring containers or photographs (201). To reduce bias associated with the 24-h dietary recall method, it is recommended to use support materials, such as photographs of foods and serving sizes (203). Image-assisted 24-h dietary recalls enhance self-report and provide valid estimates of energy intake among adults (204).

Procedure 24-hour dietary recall

Two 24-h recall interviews were conducted on randomly selected days at baseline (week 0) and post treatment (week 17-18) to determine daily intake of energy and selected nutrients. Each recall lasted for 15-30 minutes. Prior to the survey, none of the patients knew that yesterday's dietary intake should be examined. Trained interviewers, blinded to treatment assignment, used guidelines to collect detailed information over the telephone from midnight to midnight the previous 24 hours. Patients had been sent a photographic booklet of standardized portion sizes, validated for portion size estimation at group level (205). The interviewers had a check list developed from a standardization of interview methods (206). The main points of the 24-h recall were; information about the interview, review of

yesterday's meals, time and type of meal, detailed information about amount and type of food, beverages and supplements, and controlling for possible omitted foods. Patients were also asked about binging, defined subjectively, vomiting and sleeping time the previous night. These data are not analyzed in this master project.

Analysis of eating pattern

The nutrient intake was evaluated against the Norwegian dietary guidelines, with scientific basis in the Nordic Nutrition Recommendations 2012 (NNR 2012) (173). The 24-h recalls were analyzed by using the online diet tool "Kostholdsplanleggeren" (207), developed by the Norwegian Directorate of Health and the Norwegian Food Safety Authority. Basis for the calculations are the Food Composition Table (208), and a report on measurements, weights and portion sizes (209). The analyzes displayed the total intake of energy (kJ and kcal), and macro- and micronutrients for each participant.

To evaluate food selection, foods were categorized in nine different categories. The components were selected to reflect the content of The Norwegian dietary guidelines (184), but also based on previous analysis and a priori information about food intake for this patient group (210, 211). Similar food groups and indexes are proven to rank participants according to the level of adherence to the Nordic Nutrition Recommendations (212). The selected categories were 1) Fruits, berries and vegetables, 2) Milk, yoghurt and cheese, 3) Carbohydrate staple foods, 4) Whole grain, 5) Fish and fish products, 6) Lean meat, 7) Fat meat, 8) Plant derived fat sources and 9) Sweets, cakes and snacks. The participants' total intake (grams, g) in each category were then calculated.

A daily intake of 100 g juices could be included in the groups of fruits, berries and vegetables. Vegetables did not include potatoes or legumes, since these are not included in the Norwegian recommendation of five à day. Carbohydrate staple foods included bread, potatoes, rice, pasta and porridge. Whole grain included products with at least 50% of whole grain. Like earlier studies on different eating patterns (212-214), fish and fish products were covered in one category. Data from these populations indicate too low intakes to distinguish between lean and fat fish. Studies on ED in general (169), and BED patients specifically (99), show no significant difference in intakes of fish between these patients and controls. Meat was separated based on the amount of fat; more or less than 10 %, and included both chicken, red and processed meat. This was selected to reflect the content of the PED-t intervention. Plant

derived fat sources included oils, margarines, avocado, unsalted nuts and seeds. Based on the dietary advice to reduce salt intake, salted nuts and seeds were included in the category of sweets and snacks. This category also covered chocolate, chips, cakes, pastries, ice cream, buns and waffles.

2.7.3 Validation of self-reported dietary intakes

A calculation of resting metabolic rate (RMR) was used to examine the probability of underestimation of caloric consumption in this study, and thereby validating the self-reported dietary intakes.

RMR is a component of the body's total energy expenditure (TEE). REE refers to the energy requirement when the body is awake, but in rest, and accounts for 60%-70% of TEE (64). TEE and RMR can be measured by using the doubly labeled water (DLW) technique and by indirect calorimetry, respectively (64, 215). The Harris-Benedict equation is an alternative for calculation of RMR when indirect calorimetry is not available (216). Mifflin et al. published a revised version of Harris-Benedict equation in 1990 (217). According to the Mifflin-St. Jeor equation, RMR for women is: (10 x weight in kilograms) + (6.25 x height in centimeters) – (5 x age in years) – 161. The Mifflin-St. Jeor equation is predictive for modern lifestyles, estimating RMR within 10% of measured values in normal weight and obese samples (218). When multiplied by a factor for daily physical activity level (PAL), the RMR is used to estimate TEE. According to NNR 2012, PAL values between 1.3-1.5 is defined as seated work with no option of moving around, and little or no leisure activity (173). Values between 1.6-1.7 is defined as seated work with some requirement to move around, and with some leisure activity. An average PAL value for adults in Nordic countries is assumed to be around 1.6 (173).

In this study, RMR was calculated by using the Mifflin-St. Jeor Equation to estimate daily energy requirements for the study participants (217). Next, PAL values were estimated by dividing reported EI by RMR. Mean values were compared between the groups.

2.8 Statistics

Sample size calculation was done in the original PED-t trial protocol (31), and is not presented in this master thesis.

The software IBM SPSS version 24 was used to conduct all statistical analysis. Continuous variables are described using means and standard deviations (SD), categorical data as counts and percentages. Parametric statistical methods were chosen when assumptions of normality and linearity were met. Group differences in change in eating behavior and eating pattern, and weight change were examined with independent-samples t-tests or one-way between-group ANOVA. The significance of within-group changes in eating behavior, eating pattern and weight were also tested, using paired-samples t-tests. Differences during treatment are presented as estimated means with 95% confidence intervals (CI). Data is presented without correction for multiple comparisons. *P* values <0.05 were considered statistically significant, and all tests were two-tailed. Effect sizes are based on Cohen's guidelines (219). According to him, 0.01 is a small effect, 0.06 is a medium effect, and 0.14 is a large effect (219).

3 Results

First, numbers and patient characteristics are presented. Following, between- and within-group differences in eating behavior and eating pattern are given. Weight changes, and estimated RMR and PAL values are presented as final results.

3.1 Randomization and patient characteristics

Four hundred and nineteen women were recruited. After taking into consideration the inclusion criteria and EDE-Q, 232 persons were excluded, leaving 187 eligible participants in the study. Twenty-three BN or BED patients participated as a waiting list control group. Data from 23 persons were categorized as missing values. A total of 164 participants were selected and randomized to either CBT or PED-t. In this master project, pretest and posttest 24-h recalls were reported by 116 participants (CBT: 54; PED-t: 62), and TFEQ and BES by 97 participants (CBT: 43; PED-t: 54). Eleven patients in the control group reported TFEQ and BES at both assessments. A flow chart is illustrated in Figure 3.

Fourteen participants (11%) of those who submitted pretests in this master project, dropped out during the intervention period. Reported reasons for dropout were moving overseas, pregnancy, being too occupied with school, receiving another treatment offer, familiar conditions, no motivation, and recovery. There was no difference in dropout rates between the two treatment arms, were 5/14 (36%) dropped out of CBT, and 9/14 (64%) dropped out of PED-t ($p=0.22$). Non-completers did not differ from completers in baseline characteristics, BES and TFEQ score, or any 24-h recall variable and food item at baseline (p-values > 0.05).

Baseline demographic and clinical characteristics of the randomized patients are presented in Table 6. There were no differences between the two intervention groups in mean baseline values. The control group had a significant longer history of ED than both CBT and PED-t.

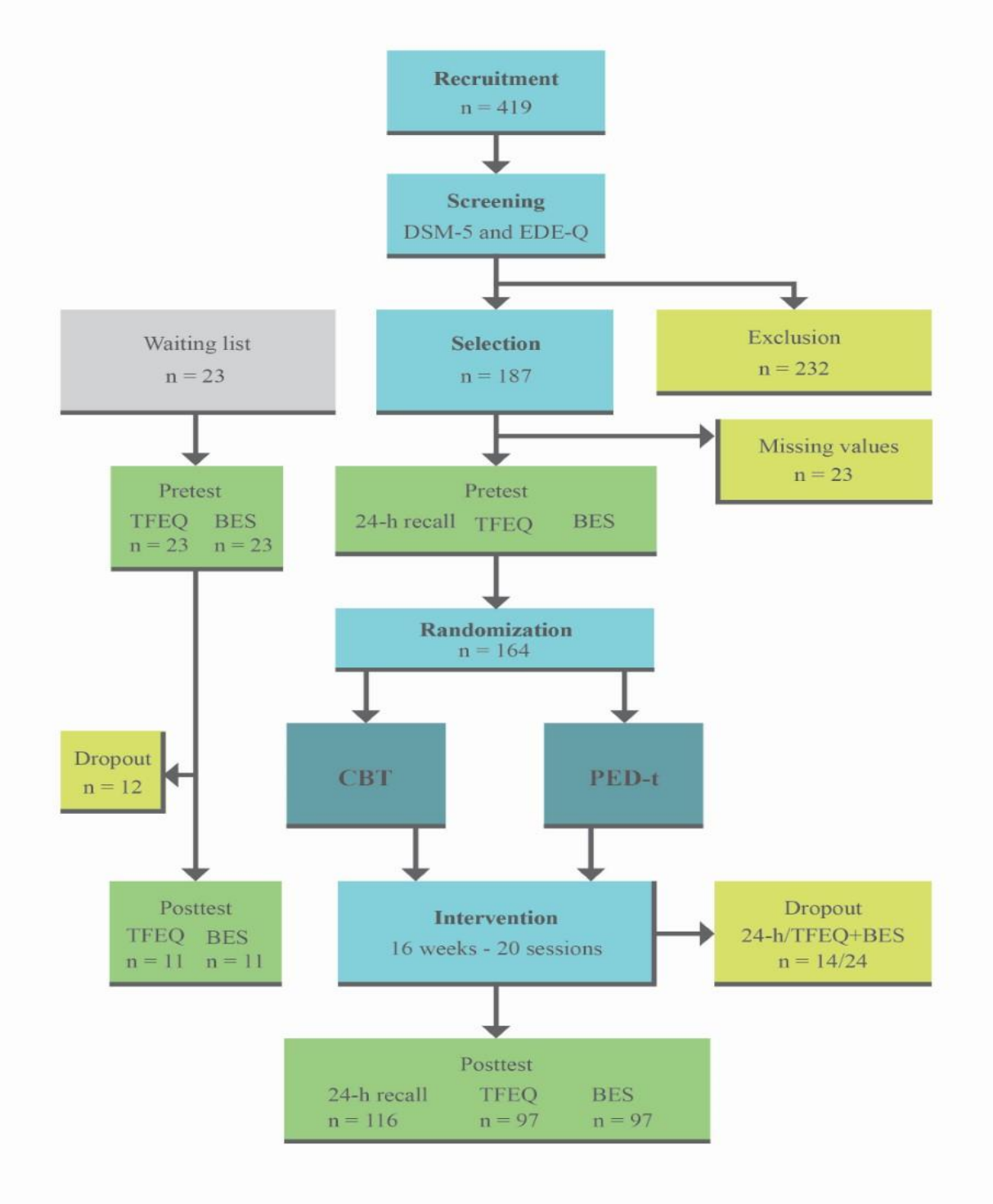

Figure 3 – Flow chart for the master project

DSM-5: Diagnostic and Statistical Manual of Mental Disorders, fifth edition; EDE-q: Eating Disorder Examination Questionnaire; 24-h recall: 24-hour recall; TFEQ-R21: the 21-item Three Factor Eating Questionnaire; BES: Binge Eating Scale; CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy.

Table 6 – Demographic and clinical characteristics of the randomized patients

	CBT n = 63	**PED-t** n = 67	**Control** n = 22	**p**
		n (%)		
BN	41 (65)	42 (63)		
BED	22 (35)	25 (37)		
		Mean (SD) Range		
BMI, kg/m^2	25.5 (5.4) 17.7 – 44.7	25.6 (5.3) 17.3 – 38.9		0.89
Age, years	28 (5) 17 – 39	29 (6) 28 – 41		0.47
Age at onset, years	16 (3) 11 – 24	16 (4) 6 – 32	15 (7) 6 – 31	0.49
Duration of ED, years	11 (6) 1 – 24	12 (7) 0 – 28	17 (9) 1 – 30	CBT vs Control: < 0.01[a] PED-t vs Control: 0.02[b] CBT vs PED-t: 0.54
		n (%)		
History of AN				
Yes	10 (19)	12 (18)	1 (5)	
No	37 (68)	42 (63)	18 (90)	No significant
Do not know	7 (13)	13 (19)	1 (5)	differences

CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; BN: Bulimia Nervosa; BED: Binge Eating Disorder; ED: Eating Disorder; AN: Anorexia Nervosa; SD: Standard Deviation; [a]Mean difference -6.3 years (1.8); [b]Mean difference -5.0 years (1.8).

3.2 Change in eating behavior

There were no differences between the three groups in any eating behavior variable at baseline, and all groups reported dysfunctional eating behavior. Analyses revealed no differences in BES, CR, UE and EE changes between CBT and PED-t groups. However, compared to controls, CBT improved scores for BES and EE, and PED-t improved scores for BES, UE and EE. Both intervention groups had significant reductions in total BES, UE and EE scores during treatment, with mean changes of -9.0, -4.3, and -2.5 in CBT, and -10.4, -5.9, and -3.7 in PED-t, respectively. Mean scores, changes, and group differences in BES and TFEQ-R21 subscales are presented in Table 7 and Figure 4. Differences in BES, UE and EE had medium effect sizes, whereas difference in CR had a small effect size, measured with Eta squared (220).

Although not included in study aims, analysis was also conducted for treatment subgroups (BN and BED). Changes in eating behavior for treatment subgroups are illustrated in Figure 5. Baseline and post treatment scores, and changes in eating behavior are presented in Appendix 6.

Table 7 – Mean scores, changes, and group differences in eating behavior during treatment

	CBT n=43	PED-t n=54	Control n=11	CBT vs PED-t		CBT vs Control		PED-t vs Control	
Variable		Mean (SD)		Mean difference (95% CI)	p	Mean difference (95% CI)	p	Mean difference (95% CI)	p
BES									
Pre	42.6 (5.9)	43.4 (6.8)	41.9 (8.4)	-0.8 (3.5, 1.9)	0.54	0.7 (-3.8, 5.1)	0.76	1.5 (-2.8, 5.9)	0.49
Post	33.6 (7.4)	33.0 (8.3)	38.9 (10.4)	0.6 (-2.7, 3.9)	0.72	-5.3 (-10.7, 0.2)	0.06	-5.9 (-11.2, -0.5)	0.03*
Change	-9.0 (5.6)[#]	-10.4 (8.6)[#]	-3.00 (7.2)	1.4 (-1.6, 4.4)	0.35	-6.0 (-10.9, 1.0)	0.02*	-7.4 (-12.2, -2.5)	<0.01*
TFEQ									
CR									
Pre	17.6 (2.9)	16.6 (3.2)	17.3 (2.8)	1.1 (-0.2, 2.3)	0.09	0.40 (-1.6, 2.3)	0.70	-0.66 (-2.6, 1.3)	0.51
Post	17.0 (3.3)	15.2 (3.0)	16.0 (4.0)	1.9 (0.5, 3.2)	<0.01*	1.0 (-1.1, 3.2)	0.35	-0.8 (-2.9, 1.3)	0.45
Change	-0.6 (3.0)	-1.4 (3.1)[#]	-1.3 (3.5)	0.8 (-0.5, 2.1)	0.22	0.6 (-1.5, 2.7)	0.55	-0.2 (-2.2, 1.9)	0.90
UE									
Pre	27.3 (4.9)	28.1 (4.2)	28.3 (6.2)	-0.8 (-2.7, 1.1)	0.42	-1.1 (-4.2, 2.1)	0.50	-0.3 (-3.4, 2.8)	0.84
Post	23.0 (4.7)	22.2 (5.9)	26.5 (6.0)	0.8 (-1.4, 3.0)	0.48	-3.6 (-7.2, 0.1)	0.06	-4.3 (-7.9, -0.8)	0.02*
Change	-4.3 (4.7)[#]	-5.9 (5.1)[#]	-1.8 (4.9)	1.6 (-0.4, 3.5)	0.12	-2.5 (-5.8, 0.8)	0.14	-4.0 (-7.3, -0.8)	0.01*
EE									
Pre	18.3 (4.1)	18.6 (3.7)	16.6 (4.5)	-0.3 (-1.9, 1.3)	0.70	1.7 (-1.0, 4.3)	0.21	2.0 (-0.6, 4.6)	0.13
Post	15.8 (3.6)	14.9 (4.4)	17.0 (4.2)	1.0 (-0.7, 2.6)	0.25	-1.2 (-3.8, 1.6)	0.68	-2.1 (-4.8, 0.6)	0.12
Change	-2.5 (3.6)[#]	-3.7 (4.1)[#]	0.4 (3.7)	1.3 (-0.3, 2.9)	0.12	-2.8 (-5.5, -0.2)	<0.01*	-4.1 (-6.7, -1.5)	<0.01*

CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; BES: Binge Eating Scale; TFEQ: Three Factor Eating Questionnaire; CR: Cognitive restraint; UE: Uncontrolled eating; EE: Emotional eating; SD: Standard Deviation; 95% CI: 95% confidence interval; [#]Significant change from baseline; *Significant different change between groups.

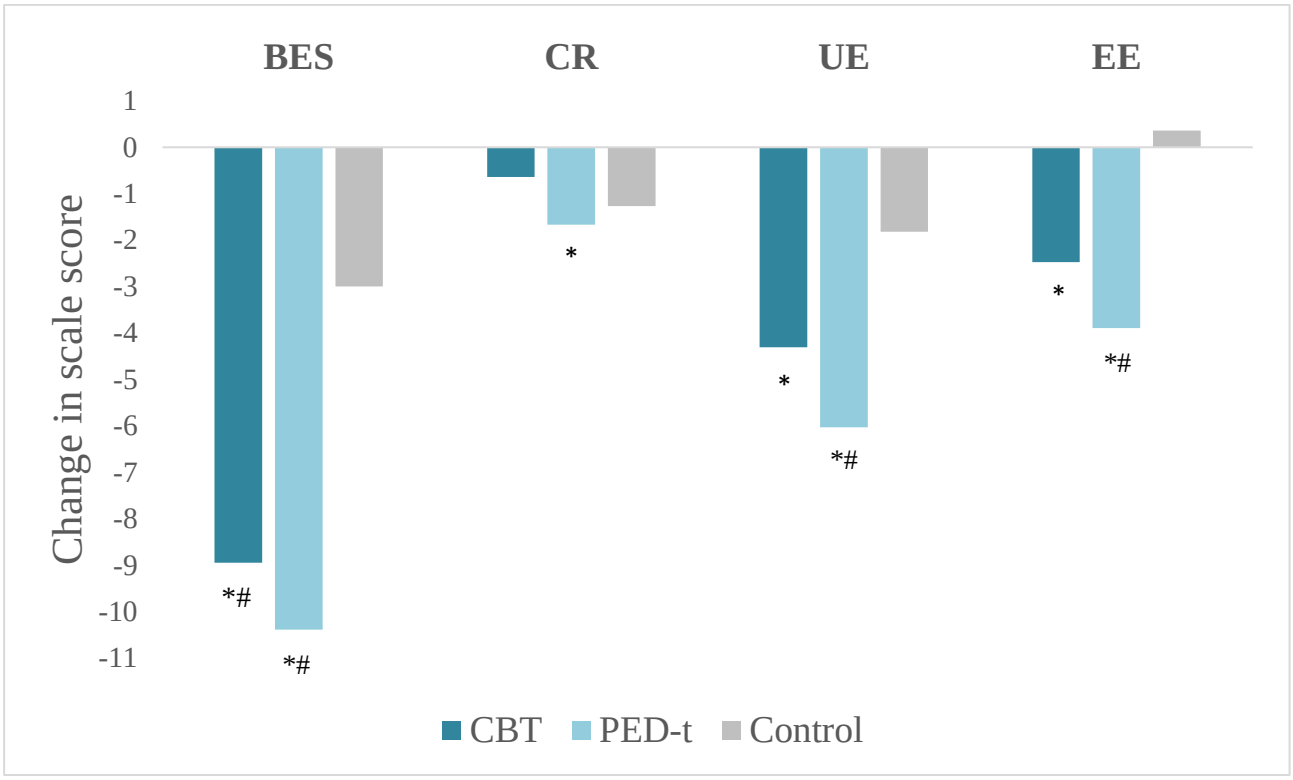

Figure 4 – Change in eating behavior during treatment

BES: Binge Eating Scale; CR: Cognitive restraint; UE: Uncontrolled eating; EE: Emotional eating; CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; *Significant change from baseline; #Significant different change compared to control.

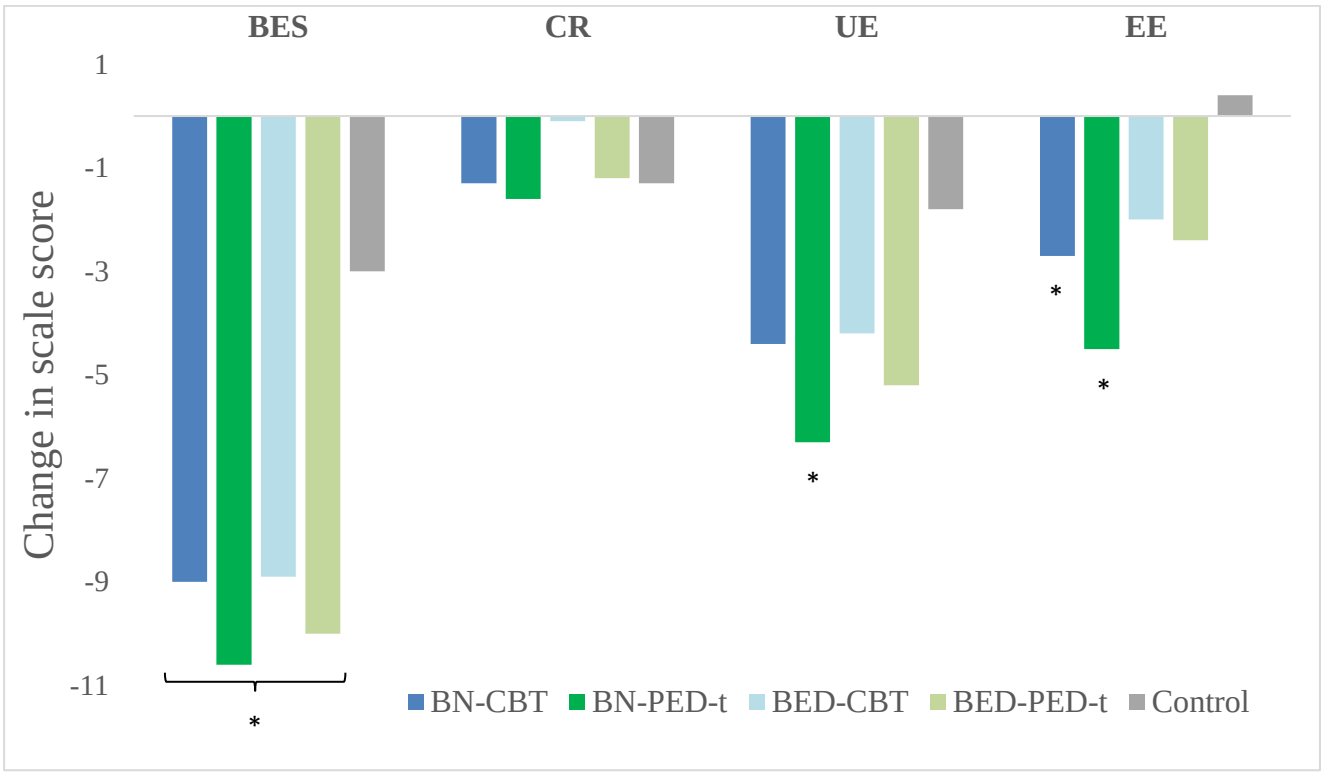

Figure 5 – Change in eating behavior for treatment subgroups

BES: Binge eating scale; CR: Cognitive restraint; UE: Uncontrolled eating; EE: Emotional eating; BN: Bulimia Nervosa; BED: Binge Eating Disorder; CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; *Significant different change compared to control.

3.3 Change in dietary eating pattern

There were no differences in change of nutrient intake and dietary eating pattern between CBT and PED-t (Table 8). Except a greater fat intake among PED-t patients ($p = 0.05$), there were no differences in baseline intakes between the intervention groups. Despite consumption of fat and protein in the higher range, and of carbohydrates in the lower range, both groups reported macro nutrient intakes within the recommended intakes of E%, and dietary fiber above the recommended intake, at both assessment points.

An increased consumption of whole grain was found in the PED-t group only, and the reported post treatment intake was significantly higher than in CBT ($p = 0.04$) (Figure 6). The CBT group decreased the consumption of fat meat during treatment. Both CBT and PED-t reported a significant reduction in consumption of fruits, berries and vegetables, with no difference between the groups.

Differences between the interventions were detected when separated in subgroups. BN-PED-t reported a larger reduction in fat intake compared to BN-CBT (-28 g vs 7.5 g, $p = 0.05$) (Figure 7). A greater increase in dietary fiber was found in BED-PED-t compared to BN-PED-t and BED-CBT. Also, BED-PED-t reported a greater increase in consumption of whole grain sources than BN-CBT (98 g vs 21 g, $p = 0.05$). Decreased intake of fruits, berries and vegetables was found in BN patients only. Details on change in dietary eating pattern for treatment subgroups are presented in Appendix 8.

Neither the CBT or the PED-t group met the recommended daily allowance (RDA) of vitamin D, retinol, folate, iron, and iodine at baseline and post treatment. A detailed overview of mean baseline and post treatment intakes, and changes in nutrient intake during treatment, are presented in Appendix 7.

Table 8 – Nutrient intake and dietary eating pattern during treatment

	CBT n=54			PED-t n=62				
	Mean (SD)	95% CI	E%	Mean (SD)	95% CI	E%	Mean difference (95% CI)	p
Kilocalories								
Energy								
Pre	2122.7 (754.9)	1916.7, 2328.8		2608.0 (1979.7)	2105.2, 3110.7			0.08
Post	2104.6 (1246,2)	1764.4, 2444.8		2397.6 (1452.8)	2028.6, 2766.5			0.25
Change	-18.1 (1301.9)			-210.4 (1587.4)			192.2 (-346.7, 731.2)	0.48
Grams								
Fat								
Pre	86.1 (39.9)	75.2, 97.0	37	110.7 (87.6)	88.5, 132.9	38		0.05
Post	87.5 (60.3)	71.0, 103.9	37	95.6 (61.2)	80.0, 111.1	36		0.47
Change	1.4 (64.6)			-15.1 (76.6)			16.5 (-9.8, 42.8)	0.22
Carbohydrate								
Pre	247.1 (107.5)	217.7, 276.4	47	295.4 (237.9)	235.0, 355.8	45		0.15
Post	244.7 (162.2)	200.4, 288.9	47	277.0 (182.7)	230.6, 323.4	46		0.32
Change	-2.4 (174.1)			-18.4 (199.1)			16.0 (-53.3, 85.2)	0.65
Dietary fiber								
Pre	27.8 (14.4)	23.9, 31.8	3	28.8 (21.6)	23.3, 34.3	2		0.77
Post	28.3 (17.5)	23.5, 33.1	3	28.9 (19.1)	24.0, 33.7	2		0.87
Change	0.5 (12.8)			0.1 (17.0)			0.4 (-5.2, 6.0)	0.88
Protein								
Pre	97.8 (35.5)	88.1, 107.4	18	114.1 (92.9)	90.5, 137.8	18		0.20
Post	92.3 (37.8)	81.9, 102.6	18	116.1 (72.5)	97.7, 134.5	19		0.03*
Change	-5.5 (44.6)			2.0 (72.3)			-7.5 (-30.0, 15.0)	0.51
Fruits, berries and vegetables								
Pre	422.1 (293.2)	342.1, 502.1		352.2 (309.9)	273.5, 430.9			0.82
Post	235.4 (298.5)	153.9, 316.8		207.8 (239.0)	147.1, 268.4			0.58

Change	-186.7 (365.7)*[a]		-144.5 (387.3)*[b]		-42.3 (-181.5, 96.9)	0.55
Milk and dairy products						
Pre	326.6 (291.8)	246.9, 406.2	437.9 (610.8)	282.8, 593.0		
Post	369.0 (259.9)	298.0, 439.9	413.2 (344.4)	325.8, 500.7		
Change	42.4 (278.2)		-24.7 (552.8)		67.1 (-91.3, 225.5)	
Carbohydrate staple foods						
Pre	236.1 (150.6)	195.0, 277.2	256.4 (212.7)	202.4, 310.4		0.56
Post	232.3 (154.5)	190.1, 274.5	289.4 (137.7)	254.4, 324.3		0.04*
Change	-3.8 (207.7)		33.0 (187.6)		-36.8 (-109.5, 35.9)	0.32
Whole grain sources						
Pre	98.5 (99.0)	71.5, 125.5	98.0 (121.2)	67.2, 128.7		0.47
Post	112.0 (94.8)	86.2, 137.9	157.9 (133.6)	123.9, 191.8		0.04*
Change	13.5 (132.9)		59.9 (138.8)*[c]		-46.4 (-96.5, 3.8)	0.07
Fish and fish products						
Pre	40.4 (73.4)	20.4, 60.5	69.8 (118.0)	39.8, 99.7		0.11
Post	51.0 (68.4)	32.3, 69.7	67.6 (114.2)	38.6, 96.6		0.34
Change	10.6 (95.5)		-2.2 (123.8)		12.8 (-28.4, 53.9)	0.54
Lean meat						
Pre	52.5 (80.1)	30.7, 74.4	59.2 (94.4)	35.2, 83.2		0.69
Post	58.7 (77.8)	37.5, 80.0	86.8 (95.3)	62.6, 111.0		0.08
Change	6.2 (104.4)		27.7 (122.2)		-21.5 (-63.6, 20.7)	0.32
Fat meat						
Pre	62.4 (101.1)	34.8, 90.0	56.0 (118.8)	25.9, 86.2		0.76
Post	33.4 (68.0)	14.8, 51.9	58.7 (97.7)	33.9, 83.5		0.10
Change	-29.0 (101.3)*[d]		2.7 (151.6)		-31.7 (-79.9, 16.4)	0.20
Plant based fat sources						
Pre	41.6 (74.3)	21.3, 61.9	23.7 (38.0)	14.1, 33.4		0.12
Post	43.2 (60.0)	26.9, 59.5	31.4 (41.6)	20.8, 41.9		0.23

Change	1.6 (89.2)		7.7 (58.3)		-6.1 (-33.4, 21.4)	0.66
Chocolate and snacks						
Pre	121.0 (179.5)	72.0, 170.0	157.4 (291.7)	83.3, 231.5		0.43
Post	81.4 (155.9)	38.8, 124.0	119.4 (307.6)	41.3, 197.5		0.41
Change	-39.6 (231.1)		-38.0 (299.6)		-1.6 (-101.1, 97.9)	0.97

CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; SD: Standard Deviation; 95% CI: 95% confidence interval;

E%: percentage of total energy intake; *Significant change from baseline; [a]$p < 0.01$; [b]$p < 0.01$; [c]$p < 0.01$; [d]$p = 0.04$.

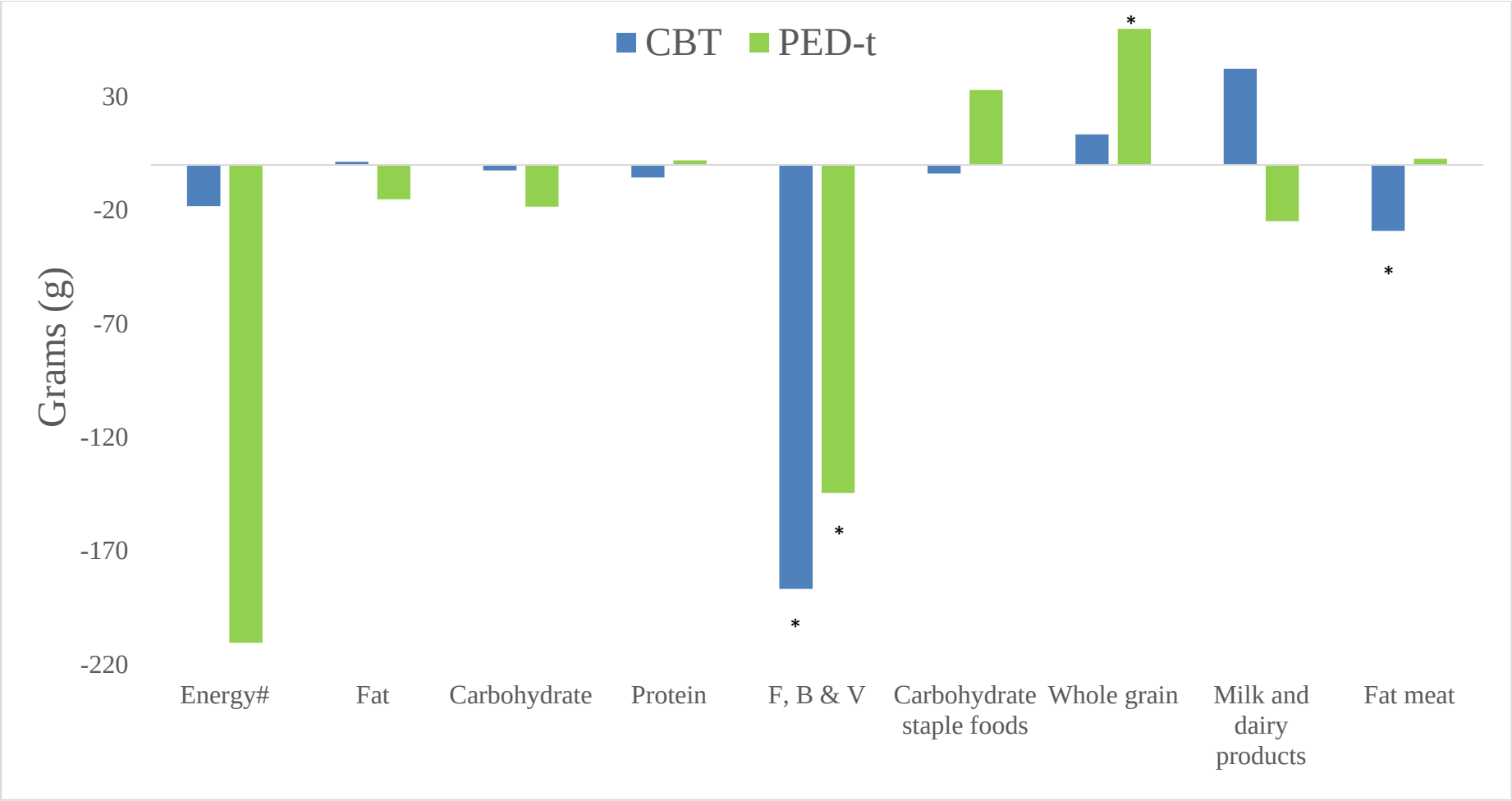

Figure 6 – Change in nutrient intake and food selection[a] during treatment

CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; [#]Energy as kilocalories; F, B & V: fruits, berries and vegetables; *Significant change from baseline to post treatment; [a]Only selected food categories are presented.

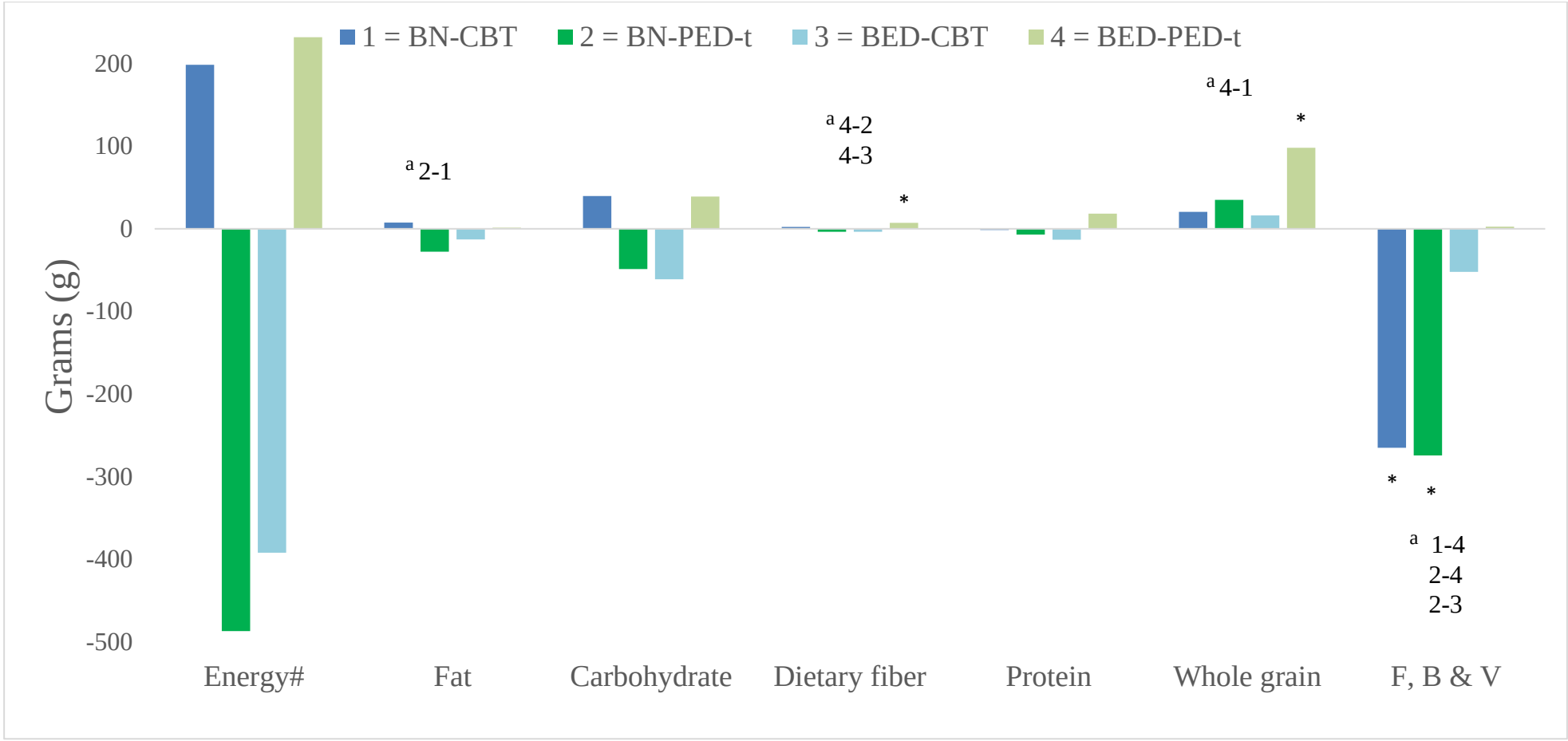

Figure 7 – Change in nutrient intake and food selection[b] for treatment subgroups

BN: Bulimia Nervosa; BED: Binge Eating Disorder; CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; [#]Energy as kilocalories; F, B & V: fruits, berries and vegetables; *Significant different from baseline to post treatment; [a]Significant differences between treatment subgroups; [b]Only selected food categories are presented.

3.4 Weight change

There were no differences in mean baseline weight between the CBT and PED-t groups, or between BN and BED patients across the intervention groups (Table 9).

There was a significant difference in weight change during treatment between the intervention groups (p=0.01). A significant weight change was seen in the PED-t group only. Mean weight gain was 1.3 kg (p < 0.01). The CBT group had a weight reduction of -0.3 kg (p=0.50). The difference in weight change had a small effect size (d = 0.19).

Table 9 – Weight change during treatment

		CBT n=49	**PED-t** n=58	**CBT vs PED-t**	**p**
		Mean (95% CI) Range			
Weight, kg					
Baseline	BN + BED	70.7 (67.0, 74.4) 50.0 – 102.8	72.2 (67.9, 76.7) 43.4 – 120.1		0.59
	BN	66.9 (63.5, 70.3) 50.0 – 86.1	65.2 (61.0, 69.4) 43.3 – 105.1		0.54
	BED	78.6 (70.6, 86.6) 50.8 – 102.8	83.9 (76.6, 91.1) 61.7 – 120.1		0.32
Post treatment	BN + BED	70.4 (66.4, 74.4) 48.6 – 106.5	73.5 (69.1, 78.0) 43.2 – 124.7		0.30
	BN	66.4 (62.5, 70.2) 48.6 – 91.8	67.1 (62.6, 71.7) 43.2 – 114.6		0.80
	BED	78.7 (70.0, 87.4) 52.2 – 106.5	84.1 (76.5, 91.6) 62.3 – 124.7		0.34
		Mean (SD)		**Mean (95% CI)**	
Weight change	BN + BED	-0.3 (3.4)	1.3 (3.11)[#]	-1.6 (-2.8, -0.3)	0.01*
	BN	-0.5 (3.5)	1.9 (3.1)[#]	-2.4 (-4.0, -0.8)	< 0.01*
	BED	0.1 (3.3)	0.2 (2.9)	-0.1 (-2.2, 1.9)	0.91

CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; BN: Bulimia Nervosa; BED: Binge Eating Disorder; 95% CI: 95% confidence interval; SD: Standard Deviation; [#]Significant change from baseline to post treatment; *Significant difference between treatment groups.

When separated into subgroups based on EDs, analysis revealed significant weight gain in the BN-PED-t group (1.9 kg, p < 0.01), a weight change statistically different than in the BN-CBT (p < 0.01). There was no difference between BED patients in the CBT and PED-t groups.

3.5 Estimated RMR and PAL values

Due to weight gain during treatment, mean RMR increased in PED-t (Table 10). Calculated PAL value for PED-t decreased from 1.8 at baseline to 1.6 at posttest. PAL values were higher compared to CBT, with 1.5 at both assessments.

Table 10 – Estimated RMR and PAL values at baseline and post treatment for each intervention group

	Baseline			Post treatment		
	RMR, kcal	EI, kcal	PAL	RMR, kcal	EI, kcal	PAL
CBT	1460	2123	1.5	1456	2105	1.5
PED-t	1468	2608	1.8	1480	2398	1.6

RMR: resting metabolic rate; EI: energy intake; kcal: kilocalories; PAL: physical activity level; CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy.

4 Discussion

4.1 Summary of main findings

The main finding was that the interventions produced comparable changes of eating behavior. Both CBT and PED-t reduced the mean score on BES, and the UE and EE scale scores (Table 7). Compared to control, greater improvements on BES and EE was found in CBT, whereas greater improvements on BES, UE and EE was reported in PED-t.

Second, no difference in change of eating pattern was found between CBT and PED-t (Table 8). Higher post treatment intakes of protein, dietary fiber, and carbohydrate staple foods were reported in PED-t compared to CBT. Both groups reported reduced consumption of fruits, berries and vegetables during treatment. PED-t increased the intake of whole grain, whereas CBT reduced the intake of fat meat.

BN patients in PED-t reported a larger reduction in fat intake than BN patients in CBT. BED-PED-t increased the intake of dietary fiber compared to BN-PED-t and BED-CBT, and reported a greater increase in intake of whole grain sources than BN-CBT. A reduction of fruits, berries and vegetables consumption was found in BN patients only.

Next, methodological considerations about this master study are discussed. A discussion on results; change in eating behavior, nutrient intake and dietary eating pattern is then presented. Finally, findings on weight change during treatment are considered.

4.2 Methodological considerations

4.2.1 Study selection

One strength of this study is the inclusion of comparable BN and BED patients not receiving any treatment, making up a control group. However, the control group consisted of only two BED patients. Regarding nutrient intake and eating pattern, a comparison between the interventions and control was not possible, as dietary data was not collected for the control patients. Moreover, the control group had a significant longer history of ED at baseline compared to CBT and PED-t. Longer duration of bulimic behaviors is associated with more severe BN, and is found to predict worse outcome in BN (72, 73). However, studies on the

natural course of BN report similar or higher rates of remission and relapse as studies on CBT for BN (221, 222). The control group did not differ from CBT or PED-t in any of the baseline 24-h recall, BES or TFEQ-R21 measurements, indicating comparable levels of ED pathology between the groups at baseline.

This study may be limited by a small statistical power. Power analysis was not assessed for the hypothesis of this master project. Sample size calculation was computed based on an overall effect in the PED-t trial, and a sample size of 144 (72 + 72) was required. In this master study, a total of 164 patients were randomized to the intervention programs, whereas 111 patients completed the post treatment tests. Using a G*Power software, a power of 0.21 was determined for changes in eating behavior during treatment (223). Such a small power increases the risk of making type II errors, that is concluding there is no effect when there is one.

This study is strengthened by the outpatient sample of BN and BED patients, as studies including clinical samples may not be generalizable to the general ED population. The outpatients setting may capture real-life changes, not directed of an unnatural environment or a laboratory setting. However, this study is based on a sample of female BN and BED patients aged 18-40, with mean age in late 20s, BMI values of 25.5-25.6 kg/m^2, recruited via social media. Generalization to younger or older subjects, men, and patients with other EDs should be made with caution. Moreover, this master project did not consider level of ED psychopathology, and results can only be generalized to BN and BED patients with dysfunctional eating behavior.

4.2.2 The intervention programs

One main strength of this study is comparison of a preliminary treatment manual (PED-t) to the evidence-based CBT, and to a waitlist control group. Hence, effects can be attributed to the specific programs. Second, participants were randomized into groups, and thus selected by chance. The randomization took place after pretest assessments, and participants and scientists were therefore blinded to the intervention at baseline. However, therapists and participants were not blinded to treatment allocation. Participants used an individual ID-number throughout the intervention. Thus, interviewers and scientists were blinded to group affiliation during the 24-h recalls, and during analysis of eating pattern and eating behavior.

Also, as there was no contact between the treatment groups during the intervention, risk of "contamination" bias was minimized.

One limitation is the lack of a group of healthy control subjects not receiving any treatment, which could have corrected for natural time changes, or any changes simply by being engaging in a study.

The duration of the intervention could be discussed. The original 20-session intensity of the CBT program is proven to be suitable for most adult ED patients (25, 81). However, a more intensive treatment manual has proven to increase completion rates compared to a long-term version of CBT (121, 224). In this master project, the PED-t sessions had a longer duration compared to CBT, but no difference in dropout rates was detected.

4.2.3 Collection and analysis of eating behavior

This study is strengthened by the combination of the TFEQ-R21 and the 16-item BES, measuring several dimensions of dysfunctional eating behavior. TFEQ-R21 and BES are two of the questionnaires with the smallest item number (225). Hence, the combination did probably not cause a great respondent burden. However, the TFEQ-R21 limited the opportunity to compare results to other intervention studies, as many former studies are based on the original 51-item TFEQ, or other questionnaires such as the 33-item Dutch Eating Behavior Questionnaire (DEBQ) (226) and the 28-item Eating Disorder Examination Questionnaire (EDE-Q) (227). DEBQ and EDE-Q are proven to be reliable and valid measures of dysfunctional eating behaviors in ED subjects, but measures other aspects of eating behavior than the TFEQ-R21 (225, 226, 228-230). The variance between different instruments was illustrated by Safer et al., who found that BN patients reduced their CR score during CBT treatment measured by EDE-Q, but found no effect on the TFEQ CR scale (231).

The CR scale of the TFEQ was separated into subscales by Westenhofer et al. (232). RC and FC represent an all-or-nothing or a graduated approach, respectively, to eating, dieting, and weight management (148). The differentiation between RC and FC could have been be more relevant to this master project if the study participants were defined as extreme restrained eaters. Also, the TFEQ-R21 does not differentiate between rigid (RC) and flexible (FC) subscales, and this master project only discussed the total score of CR.

4.2.4 Collection and analysis of dietary data

In this study, analysis on eating pattern was based on 24-h recalls conducted by telephone interviews, which is considered a more accurate method for monitoring nutritional intake compared to food records (202). 24-h recalls are subjective, retrospective and open-ended questionnaires administered by trained interviewers, suitable for large-scale studies (201). They provide detailed information with a small respondent burden compared to prospective food records, were the participants are instructed to record time and duration for each eating episode, and ingredients, brand name, quantity, and preparation method for all foods consumed (165, 169).

Self-reported dietary recalls have shown to be biased toward underreporting of food intake rather than overreporting, especially in overweight and obese study subjects (204, 233). Weight, poor body image, weight concerns, dieting and dietary restraint have all been linked to underestimation of dietary intake (233). Other suggested explanations for underreporting are lack of motivation or perceived burden of food reporting (233). To reduce the systematic error of underestimation in this study, participants were given a photographic booklet of standardized portion sizes. Image-assisted dietary assessments are proven to enhance self-report and provide valid estimates of energy intake (204). The booklet increased the participant's ability to estimate quantities, and remember amounts and types of foods eaten the previous 24-hour period (234).

Study participants often increase their knowledge about food composition and portion estimation during a dietary intervention, and self-report accuracy is usually improved during treatment (235). Also, subjects may omit food items they see as "unhealthy", to satisfy the interviewer (233). Furthermore, the intervention group may engage in a positive relationship with the program and the professionals involved, and thus creating a more accurate report of dietary intake (236). In this study, both CBT and PED-t received active treatments. Thus, variances in overreporting on healthy behaviors or omission of "unhealthy" foods were probably avoided, as reflected in decreased reported intakes of fruits, berries and vegetables in both intervention groups.

This master project may be limited of the single 24-h recall at baseline and post treatment. Three 24-h recalls taken at each assessment point are reported to be optimal for estimating EI compared to DLW-derived energy expenditure (237). In addition, only one recall may not

reflect the true binge eating frequency, as studies have reported between 3-11 weekly binges in BN and BED patients (97, 238, 239).

This study has some strengths compared to studies implemented in laboratory settings. In laboratory settings, participants are instructed to 'eat as much as you would during a normal meal', or 'eat as much as you can'. These settings do not necessarily represent the patients' own dietary habits (169). Second, participants are often instructed not to eat a definite period prior to the test meals, to ensure certain levels of hunger (99). At high levels of hunger, subjects may eat more than they would in normal conditions.

A minor limitation of this study is that it did not correct for intake of dietary supplements. In the latest dietary survey in Norway (Norkost 3, 2010-2011), dietary supplements were taken by 58% of the women (167). When supplements were included, the average intake of vitamin A, D, E and C, and iron and folate increased (167). It is possible that inclusion of dietary supplements in this master project could have corrected for some of the reported micro nutrient intakes below RDA.

4.2.5 Validation of self-reported dietary intakes

The following section discusses aspects of the self-reported dietary data, in attempt to clarify their validity. Similar EIs as reported throughout this study, are found in other samples of BN and BED patients (169). Studies reporting higher EIs might have included subjects with higher BMI values and thereby higher energy needs (161, 163, 239). Also, this master study may have captured periods of binge free days, were caloric consumption is lower than in "worse" periods (164, 165). Moreover, compensating periods could skew the mean down (160). Studies have reported significant weight losses following treatments comparable to this master project (240, 241). However, these studies included samples with higher initial BMI values, suggesting a greater initial effect on weight loss during treatment compared to the participants in this master project. Together, these considerations support that dietary intakes reported by CBT and PED-t are valid.

This master project is limited by lack of data on RMR, hormone levels, PA level, change in physical fitness, binge eating frequency, and compensatory behaviors, parameters that could give additional information when validating dietary data and interpreting the results.

Preferably, EI should be compared to the participant's RMR. The RMR is related to lean body mass, but is also regulated by hormones (215). After periods of caloric restriction and/or weight reduction, biological responses work to maintain body weight at a defined "ideal" level, and RMR is reduced (242). Hence, less energy is needed for weight maintenance, and it is more difficult to sustain weight loss (242). Normalization of the eating pattern and less dietary restraint, combined with a lower RMR compared to healthy individuals, could possibly explain the observed weight gain in BN-PED-t (64). Moreover, weight gain in PED-t despite no change in reported EI could be due to reduced frequency of compensatory behaviors.

Studies have found that exercise may serve as an appetite regulator, through changed body composition, changes in hormone levels, and in turn through the modulation of hunger (243). An acute bout of moderate or high-intensity exercise may decrease levels of hormones known to stimulate appetite, while increase those known to suppress food intake (118, 244). Interestingly, PED-t tended to reduce the mean EI during treatment. Thus, this study is in line with former research suggesting that exercise do not increase levels of hunger and energy intake (118, 243).

Although exercise was not a component of the CBT intervention, the CBT group had a low estimated PAL value at baseline and post treatment, and it might seem that EI was underreported. On the other hand, it is reported that only 20% of the Norwegian, adult population meet the recommendation of 30 minutes of daily PA (167). Thus, the great majority have a sedentary lifestyle, corresponding to a low PAL value. As might be expected, the PED-t group's estimated PAL values were higher than for CBT. Surprisingly, the PAL decreased from baseline to post treatment. If the actual level of PA increased during the PED-t program, the EI in PED-t at post treatment was underreported compared to baseline. It might be suggested that despite greater nutritional knowledge, the PED-t group reported lower EI at post assessment because they considered it as "expected" by the researchers.

4.2.6 Randomization and attrition rates

Previous CBT intervention trials have reported great variations in attrition, with dropout rates ranging from 5% to 41% (83). Comparable to dropout rates found in this study, Fairburn et al. reported that in general, 80-85% of BN patients complete CBT treatment (22). Vist et al. reported that an average of 27.3% BN patients drop out of CBT treatment (245). Notable, dropout rates found in this master project is based on participants reporting data at baseline, but not at post treatment. Higher dropout rates may be found in the entire PED-t trial.

4.3 Improvements in eating behavior

According to Marcus' cut-off value in the BES, all participants were defined as severe binge eaters at baseline (195). Despite reductions during treatment, the intervention groups were 6 points above the cut-off value for severe binge eating at post treatment. The values were not affected by outliers, and analysis revealed a comparable distribution of the lowest and highest quartiles in CBT and PED-t. There were no differences in baseline BES score, or change during treatment, between BN and BED patients. It may be suggested that the improvements in binge eating behavior will continue after the intervention period, and that further reductions will be detected at follow-up assessments in the PED-t trial (100, 110).

Moderate and severe binge eating behavior, defined as BES score 18-27, and >27, respectively, has been associated with the tendency to set unrealistically strict diets (194). Moreover, moderate and severe binge eaters have reported low efficacy expectations to sustain a diet (194). Thus, it may be suggested that the patients have acquired a healthier approach to dieting throughout this study, as reflected in both treatment programs.

CBT and PED-t were classified as having uncontrolled and emotional eating behavior at baseline, but ended up within the normal range for UE and EE during treatment. The control group had mean scores just beneath the cut-off values for uncontrolled and emotional eating behavior at post assessment. These findings indicate that both interventions improve dysfunctional eating behavior related to binge eating, uncontrolled and emotional eating behavior.

The significant reductions in UE score could possibly be reflected in the numerical, but non-significant reductions in EI reported by both groups. As higher UE scores are related to higher

body weight and unhealthful food choices, this could mean important effects for general health and risk for lifestyle diseases (130, 132, 133). When group-based weight loss treatment was combined with individual CBT for BED, Downe et al. found that decreased disinhibition score was associated with binge abstinence (147). This finding supports the coinciding reductions in BES and UE found in this master study.

Studies have reported associations between disinhibition and increased food intake in response to exercise. Keim et al. studied the self-selected food intake in reduced-obese women for 14 days, while the women performed exercise 5 days a week. Those who consumed excess energy during the intervention scored significantly higher on the disinhibition scale (246). Similarly, Visona and George reported that overweighed women with high disinhibition increased their food intake 12 hours after exercise, compared to a condition without exercising (247). As the PED-t individuals in this study sample were within the normal range of UE behavior at post treatment, increased post exercise EI is not expected. This is also supported by the greater numerical reduction in EI in PED-t, although non-significant from CBT.

With significant reductions on the EE scale, CBT and PED-t ended up within the normal range of emotional eating behavior during treatment. Negative emotions are reported as the main reason for binge eating behavior by the majority of BN and BED patients (155, 156). Also, it is proposed that BN patients use purging as a strategy to reduce concerns about weight gain and negative affect following binge eating, as negative emotions are replaced with feelings such as relief and excitement after purging (159). Taken together, these findings support that decreased EE during this intervention could generate improvements in binge eating, reflected in the coinciding reductions in BES score.

Treatment effects were also examined for the 4 subgroups; BN-CBT, BED-CBT, BN-PED-t, and BED-PED-t. Significant reductions in mean UE and EE scores were reported by all groups. Interestingly, a significant different change in UE compared to control was found in BN-PED-t only. Moreover, a significant greater reduction in EE compared to control was found in BN patients only. It may seem that BN patients, and BN-PED-t particularly, have responded best to treatment considering improvements in UE and EE behavior.

4.3.1 Changes in CR score

There were no differences in mean CR change between the intervention groups, or control. The mean CR scores were within the normal range for all participants at both assessment points. Therefore, a reduction in CR would possibly have no, or a minor, clinical impact for these patients.

A significant reduction in total CR score was reported in the PED-t group, and the post score in PED-t differed from the CBT group. This difference could reflect the distinction between the two treatment manuals. The dietary module of the PED-t treatment addresses meal frequency, sufficient portion sizes, and total energy needs, which could alter the patients' perceptions of amounts. Contrary, CBT addresses the ED pathology rather than dietary routines, with focus on over-evaluation of eating, shape and weight, extreme dieting, binge eating and purging. It might be suggested that reduced CR in PED-t reflects reduced fear of "forbidden" foods.

Treatment effect on dietary restraint in BN and BED patients show great diversity. Increased levels of dietary restraint in BED patients and overweight subjects following CBT and/or weight reduction programs are reported in several other studies (136, 147, 163, 240, 241). Contrary, Safer et al. found significant decreases in restraint among BN patients who completed 18 weeks of CBT (231). Significant effect on restraint during treatment could be due to higher initial BMI values compared to the CBT and PED-t groups in this master project (136, 147, 163, 241). Moreover, changes in restraint could be reported for abstainers only, defined as subjects being abstinent from binge eating the previous 28 days (147).

BN-CBT had significant higher CR scores than BED-PED-t at both assessment points (see Appendix 6). This is supported in a study by Wilfley et al., where BED patients reported lower scores for CR compared to BN (36).

Studies have reported associations between increased CR score and binge abstinence in BED patients receiving CBT and weight control treatment (145, 147). In this study, BED-CBT patients received the same treatment as BN-CBT, although they could be more favorable of treatments aiming to increase dietary restraint (145, 147).

4.4 Changes in nutrient intake

No differences between CBT and PED-t was detected for change in nutrient intake and food selection. This could possibly be explained by the similar targets of the two intervention programs; establish regular eating patterns, reduce dietary restraint and increase the flexibility in selection of foods (46). Through education about caloric and nutrient content, energy needs, and composition of meals and snacks, PED-t aimed to establish healthy eating habits (46, 97). Similar, the CBT addressed an unhealthy eating pattern, to reduce binge eating frequency and compensatory behaviors. Another explanation for the lack of more significant treatment effects on nutrient composition could possibly be the level of nutritional knowledge in this sample. It is suggested that ED patients possess adequate nutrition knowledge, and some may be ambivalent and resistant to treatment due to maintaining processes during a prolonged ED (248-251).

Significant changes in nutrient intake were reported by Masheb et al. in BED patients following a 6-month combined treatment of CBT and general nutrition, or CBT and a low-energy density diet (239). Using 24-h recalls, the participants reported significant reductions in fat intake (39 E% to 30 E%), and increased intakes of protein (16 E% to 19 E%), carbohydrates (46 E% to 52 E%) and dietary fiber (239). Two distinctions from this master project should be noticed. First, the overall sample had an initial BMI of more than 39 kg/m^2, compared to BMI values of 25.5 kg/m^2 and 25.6 kg/m^2 in CBT and PED-t, respectively. Second, despite comparable number of treatment sessions, the intervention period lasted for six months.

No significant change in EI was detected during CBT and PED-t. Moreover, there were no differences in EIs between the intervention groups. This is comparable to findings reported by Segura-Garcia et al. (169). However, when separated in subgroups, the main daily EI at baseline was significantly higher in BN-PED-t than both BED subgroups (Appendix 8), supported by previous comparisons of EI in BN and BED (161, 252).

Some within-group changes during treatment were detected. CBT patients reported reduced intake of fat meat, whereas PED-t patients increased the intake of whole grain products. Interestingly, both groups decreased the consumption of fruits, berries and vegetables. Moreover, higher post treatment intakes of protein, dietary fiber, and carbohydrate staple

foods were reported in PED-t compared to CBT. Results based on subgroups are used for further discussion.

The greater protein intake in PED-t at post treatment was significant for BN patients only, which had a higher consumption than both CBT subgroups. As protein is found to be the most satiating macronutrient, this may have a long-term effect on EI (178). Moreover, an increased consumption of protein could improve the satiety deficit found in BN and BED, and decrease the risk of binge eating (178).

Interestingly, the change in fat intake was significant different between BN-PED-t (-28 g) and BN-CBT (8 g). For BN-PED-t, this reduction is in line with the findings reported by Masheb et al. (82). It might seem that BN patients in PED-t had a positive effect towards more protein, and less fat, and thus a higher dietary quality conforming to the Norwegian dietary guidelines (173, 184).

Increased consumption of dietary fiber and whole grain was found in BED-PED-t. The increase in whole grain products equals approximately two slices of bread. No change was detected for carbohydrates, but it would be interesting to examine the nutrient separated into simple and complex carbohydrates. During PED-t, patients learned to use the plate model, and the need for carbohydrates was discussed. Binge episodes are often high in sweet foods and simple carbohydrates (99, 164). Therefore, it might be suggested that during PED-t, the carbohydrate consumption is shifted towards reduced intake of simple, but a greater intake of complex carbohydrates. No significant change in carbohydrate intake might be explained by binge episodes high in carbohydrates, skewing the mean intake upwards and concealing any possible reduction of simple carbohydrates (164).

Higher disinhibition score is related to decreased consumption of high-fiber bread (176). The reported increase of dietary fiber and whole grain products in BED-PED-t could possibly be related to the observed reduction in UE. It would be interesting to see if the reported dietary changes in PED-t are reflected in improved BES and UE scores, as higher ratings have demonstrated associations with less healthful food choices (132, 210). However, correlation analysis was not included in study aims, and were therefore not conducted.

When examining the reductions in intake of fruits, berries and vegetables, these were significant only for BN subjects. BN-CBT and BN-PED-t reported mean reductions equal 2-3

daily portions. It is hard to justify this reduction, but one possible explanation is an increased focus on nutritious, energy-dense foods, either to reduce the risk of binge eating or to secure energy needs when exercising (BN-PED-t). Masheb et al. reported an opposite effect during the combined CBT treatment, as fruit consumption increased by one serving a day during treatment (239). However, this was a sample of BED patients. It has been demonstrated that fat consumption among binge eaters is negatively correlated with intake of fruits and vegetables (177). However, this could not explain the lowered intake among BN patients in this study, as reported fat intake did not increase during treatment, and there was a significant difference between BN patients in CBT and PED-t at post treatment. It is possible that the baseline consumption of fruits, berries and vegetables was relatively high, and that a reduction is a sign of improved eating pattern as they might be replacing low-energy foods with more nutritious foods. With an assumption of compensating days of low-energy foods following binge eating days, the decreased consumption of fruits, berries and vegetables can reflect improvements in bulimic behaviors.

4.4.1 Nutrient intake relative to recommended intakes

Both groups had intakes of macronutrients and dietary fiber within the recommended ranges, although in the higher range of E% from fat and protein, and in the lower range from carbohydrates. Comparable energy distribution on binge days is reported by using 24-h recalls in a BED sample, although with slightly higher E% from fat and carbohydrates, and lower E% from protein (253).

Neither the CBT or the PED-t group met the recommended daily allowance (RDA) of vitamin D, retinol, folate, iron, and iodine at baseline and post treatment. In this study, approximately 20% of the participants used supplements throughout the study, with no differences between the groups (data not shown). The most frequently used supplements were omega-3, cod liver oil, vitamin D, multivitamin, iron and B12. The use of vitamin D is a positive insight, as it might reduce the risk of low BMD and osteoporosis found in BN patients (33, 34), and correct the low intakes found in young females and ED samples (167, 174).

4.5 Weight change

PED-t individuals had a mean weight gain of 1.3 kg during treatment. When separated in subgroups, this was significant for BN-PED-t only (1.9 kg). It might be suggested that engagement in PA has reduced the frequency of compensatory behaviors, as regular PA is associated with improved self-esteem, body image, negative mood, and ED pathology (101). It should be noted that one BN patient in PED-t had a weight gain of 10 kg, skewing the mean upwards.

Reduced BMI is reported in BED patients following a CBT and nutritional education program (239). These participants reported eating approximately 400 kcal less at post treatment (6 month) compared to baseline, enough to lose about 0.45 kg per week (239). Given that this master project conducted the post weight assessment at week 17-18, we do not know if the numerical decrease in EI found in PED-t (-210 kcal) could promote weight loss in the longer term.

Weight gain in PED-t could possibly be explained by increased EI in response to exercise (254). However, randomized cross-over studies report no compensatory increase in EI after bouts of moderate and intensive exercise (118, 255). We suggest that PED-t individuals over time will get a greater lean body mass due to exercise, known to increase the body's TEE (256, 257). A possible weight reduction or loss in fat mass could therefore be detected in the longer term (258, 259). It is important to notice, that even at low levels of weight losses, significant improvements in waist circumference, clinical and psychological aspects may occur (260).

Some research supports the lack of weight reduction in this master project. First, obese patients suffering from BED do not seem to lose weight with a purely CBT program (122). When combined with exercise, BED patients have reported significantly better results for weight reduction at 1-year follow-up compared to CBT alone (110). Second, although both CBT and PED-t promote healthy eating strategies, improvements in binge eating frequency do not necessarily reduce body weight (21). Reductions in bulimic and compensatory behaviors in BN patients will counterbalance the effect on weight, whereas individuals with BED will still have a problem with a generally high EI (21).

5 Conclusions

In sum, this study sought to examine and compare the efficacy of CBT and PED-t as treatments for BN and BED. The effect was examined on

1) Eating behavior; binge eating, cognitive restraint, uncontrolled eating behavior, and emotional eating behavior.

2) Eating pattern; nutrient intake and food selection.

This trial established support for CBT and PED-t in treatment of BN and BED patients, with no difference in treatment effect between the interventions. Both groups reported significant improvements in dysfunctional eating behavior. Indications of greater treatment effect in some parameters following PED-t were detected. Compared to CBT, BN-PED-t tended to report better improvements in eating behavior, and BN-PED-t and BED-PED-t appeared to have greater improvements in different dietary variables.

Interpretations should be made with the reservation that this master study had a low statistical power, as the power analysis was computed based on an overall effect in the PED-t trial. A power of 0.21 is too small to draw conclusions about change in eating behavior and eating pattern.

To our knowledge, this is the first study to examine change in eating behavior and eating pattern following CBT or physical exercise and dietary therapy among women with BN- and BED diagnosis.

Future research should investigate whether PED-t provide similar or better improvements in ED pathology than CBT for BN and BED, in terms of changed eating behavior and nutrient intake, and whether these effects are sustained in the longer term. Moreover, changes in nutrient intake should be compared to a control group of BN and BED patients not receiving any treatment. It would also be beneficial to examine changes in eating behavior and nutrient intake in relation to RMR, PA level and body composition.

References

1. American Psychiatric Association (APA). DSMTF. Diagnostic and statistical manual of mental disorders : DSM-5. Arlington, VA: American Psychiatric Association; 2013.
2. Culbert KM, Racine SE, Klump KL. Research Review: What we have learned about the causes of eating disorders - a synthesis of sociocultural, psychological, and biological research. J Child Psychol Psychiatry. 2015;56(11):1141-64.
3. Keski-Rahkonen A, Mustelin L. Epidemiology of eating disorders in Europe: prevalence, incidence, comorbidity, course, consequences, and risk factors. Curr Opin Psychiatry. 2016;29(6):340-5.
4. Smink FR, van Hoeken D, Hoek HW. Epidemiology, course, and outcome of eating disorders. Curr Opin Psychiatry. 2013;26(6):543-8.
5. Helsedirektoratet. Nasjonal faglig retningslinje for tidlig oppdagelse, utredning og behandling av spiseforstyrrelser. Statens Helsetilsyn; 2017. p. 103.
6. Cooper Z, Fairburn CG. Management of bulimia nervosa and other binge eating problems. Advances in Psychiatric Treatment. 2009;15(2):129-36.
7. Kessler RC, Berglund PA, Chiu WT, Deitz AC, Hudson JI, Shahly V, et al. The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. Biol Psychiatry. 2013;73(9):904-14.
8. Stice E, Marti CN, Rohde P. Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. J Abnorm Psychol. 2013;122(2):445-57.
9. Hay P, Girosi F, Mond J. Prevalence and sociodemographic correlates of DSM-5 eating disorders in the Australian population. J Eat Disord. 2015;3:19.
10. Gotestam KG, Agras WS. General population-based epidemiological study of eating disorders in Norway. Int J Eat Disord. 1995;18(2):119-26.
11. Rosenvinge JH, Gotestam, K. G. Eating disorders - how should treatment be organized? Tidsskr Nor Laegeforen. 2002;122(3):285-8.
12. Bulik CM, Kleiman SC, Yilmaz Z. Genetic epidemiology of eating disorders. Curr Opin Psychiatry. 2016;29(6):383-8.
13. Fairburn CG, Shafran R, Cooper Z. A cognitive behavioural theory of anorexia nervosa. Behav Res Ther. 1999;37(1):1-13.
14. Shisslak CM, Crago M, McKnight KM, Estes LS, Gray N, Parnaby OG. Potential risk factors associated with weight control behaviors in elementary and middle school girls. J Psychosom Res. 1998;44(3-4):301-13.
15. Stice E, Marti CN, Durant S. Risk factors for onset of eating disorders: evidence of multiple risk pathways from an 8-year prospective study. Behav Res Ther. 2011;49(10):622-7.
16. Skarderud F, Rosenvinge JH, Gotestam KG. Eating disorders - an overview. Tidsskr Nor Laegeforen. 2004;124(15):1938-42.
17. Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. Behaviour research and therapy. 2003;41(5):509-28.
18. Rosenvinge JH, Pettersen G. Epidemiology of eating disorders part II: an update with a special reference to the DSM-5. Advances in Eating Disorders. 2015;3(2):198-220.
19. World Health Organization (WHO). Growing up in unequal: gender and sosioeconomic differences in young people's health and well-being. Jo Inchley et.al, editor. Copenhagen, Denmark: World Health Organization; 2016.
20. Slof-Op 't Landt MC, van Furth, E. F., van Beijsterveldt, C. E., Bartels, M., Willemsen, G., de Geus, E. J., Ligthart, L., Boomsma, D. I. Prevalence of dieting and fear of

weight gain across ages: a community sample from adolescents to the elderly. Int J Public Health. 2017.
21. Fairburn CG. Få bukt med overspising. 1 ed. Oslo: Gyldendal Akademisk; 2014.
22. Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. Behav Res Ther. 2003;41.
23. Fairburn CG, Harrison PJ. Eating disorders. Lancet. 2003;361(9355):407-16.
24. Milos G, Spindler A, Schnyder U, Fairburn CG. Instability of eating disorder diagnoses: prospective study. Br J Psychiatry. 2005;187:573-8.
25. Murphy R, Straebler S, Cooper Z, Fairburn CG. Cognitive behavioral therapy for eating disorders. Psychiatr Clin North Am. 2010;33(3):611-27.
26. Garner D, Fairburn C. Relationship between anorexia nervosa and bulimia nervosa: Diagnostic implications1988. 56-79 p.
27. Fairburn CG, Cooper PJ. The clinical features of bulimia nervosa. Br J Psychiatry. 1984;144:238-46.
28. Eddy KT, Dorer DJ, Franko DL, Tahilani K, Thompson-Brenner H, Herzog DB. Should bulimia nervosa be subtyped by history of anorexia nervosa? A longitudinal validation. Int J Eat Disord. 2007;40 Suppl:S67-71.
29. Mehler PS. Clinical practice. Bulimia nervosa. N Engl J Med. 2003;349(9):875-81.
30. Greenfeld D, Mickley D, Quinlan DM, Roloff P. Hypokalemia in outpatients with eating disorders. Am J Psychiatry. 1995;152(1):60-3.
31. Mathisen TF, Rosenvinge JH, Pettersen G, Friborg O, Vrabel K, Bratland-Sanda S, et al. The PED-t trial protocol: The effect of physical exercise –and dietary therapy compared with cognitive behavior therapy in treatment of bulimia nervosa and binge eating disorder. BMC Psychiatry. 2017;17(1):180.
32. Robinson L, Micali N, Misra M. Eating disorders and bone metabolism in women. Curr Opin Pediatr. 2017;29(4):488-96.
33. Mountjoy M, Sundgot-Borgen J, Burke L, Carter S, Constantini N, Lebrun C, et al. The IOC consensus statement: beyond the Female Athlete Triad--Relative Energy Deficiency in Sport (RED-S). Br J Sports Med. 2014;48(7):491-7.
34. Melin A, Tornberg AB, Skouby S, Moller SS, Sundgot-Borgen J, Faber J, et al. Energy availability and the female athlete triad in elite endurance athletes. Scand J Med Sci Sports. 2015;25(5):610-22.
35. Crow SJ, Thuras P, Keel PK, Mitchell JE. Long-term menstrual and reproductive function in patients with bulimia nervosa. Am J Psychiatry. 2002;159(6):1048-50.
36. Wilfley DE, Citrome L, Herman BK. Characteristics of binge eating disorder in relation to diagnostic criteria. Neuropsychiatr Dis Treat. 2016;12:2213-23.
37. Goldschmidt AB, Hilbert A, Manwaring JL, Wilfley DE, Pike KM, Fairburn CG, et al. The significance of overvaluation of shape and weight in binge eating disorder. Behav Res Ther. 2010;48(3):187-93.
38. Rieger E, Wilfley DE, Stein RI, Marino V, Crow SJ. A comparison of quality of life in obese individuals with and without binge eating disorder. Int J Eat Disord. 2005;37(3):234-40.
39. Grilo CM, White MA, Masheb RM. DSM-IV psychiatric disorder comorbidity and its correlates in binge eating disorder. Int J Eat Disord. 2009;42(3):228-34.
40. Reichborn-Kjennerud T, Bulik CM, Sullivan PF, Tambs K, Harris JR. Psychiatric and medical symptoms in binge eating in the absence of compensatory behaviors. Obes Res. 2004;12.
41. Brownley KA, Berkman ND, Peat CM, Lohr KN, Cullen KE, Bann CM, et al. Binge-Eating Disorder in Adults: A Systematic Review and Meta-analysis. Ann Intern Med. 2016;165(6):409-20.

42. Hudson JI, Lalonde JK, Coit CE, Tsuang MT, McElroy SL, Crow SJ, et al. Longitudinal study of the diagnosis of components of the metabolic syndrome in individuals with binge-eating disorder. Am J Clin Nutr. 2010;91(6):1568-73.
43. Johnson JG, Spitzer RL, Williams JB. Health problems, impairment and illnesses associated with bulimia nervosa and binge eating disorder among primary care and obstetric gynaecology patients. Psychol Med. 2001;31(8):1455-66.
44. Crow S, Kendall D, Praus B, Thuras P. Binge eating and other psychopathology in patients with type II diabetes mellitus. Int J Eat Disord. 2001;30(2):222-6.
45. Mustelin L, Raevuori A, Hoek HW, Kaprio J, Keski-Rahkonen A. Incidence and weight trajectories of binge eating disorder among young women in the community. Int J Eat Disord. 2015;48(8):1106-12.
46. Latner JD, Wilson GT. Cognitive-behavioral therapy and nutritional counseling in the treatment of bulimia nervosa and binge eating. Eat Behav. 2000;1(1):3-21.
47. Hadigan CM, Walsh BT, Devlin MJ, LaChaussee JL, Kissileff HR. Behavioral assessment of satiety in bulimia nervosa. Appetite. 1992;18(3):233-41.
48. Geliebter A, Melton PM, McCray RS, Gallagher DR, Gage D, Hashim SA. Gastric capacity, gastric emptying, and test-meal intake in normal and bulimic women. Am J Clin Nutr. 1992;56(4):656-61.
49. Johnson WG, Jarrell MP, Chupurdia KM, Williamson DA. Repeated binge/purge cycles in bulimia nervosa: role of glucose and insulin. Int J Eat Disord. 1994;15(4):331-41.
50. Raevuori A, Suokas J, Haukka J, Gissler M, Linna M, Grainger M, et al. Highly increased risk of type 2 diabetes in patients with binge eating disorder and bulimia nervosa. Int J Eat Disord. 2015;48(6):555-62.
51. Hudson JI, Hiripi E, Pope HG, Jr., Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychiatry. 2007;61(3):348-58.
52. Palavras MA, Hay P, Filho CA, Claudino A. The Efficacy of Psychological Therapies in Reducing Weight and Binge Eating in People with Bulimia Nervosa and Binge Eating Disorder Who Are Overweight or Obese-A Critical Synthesis and Meta-Analyses. Nutrients. 2017;9(3).
53. Villarejo C, Fernandez-Aranda F, Jimenez-Murcia S, Penas-Lledo E, Granero R, Penelo E, et al. Lifetime obesity in patients with eating disorders: increasing prevalence, clinical and personality correlates. Eur Eat Disord Rev. 2012;20(3):250-4.
54. Bulik CM, Marcus MD, Zerwas S, Levine MD, La Via M. The changing "weightscape" of bulimia nervosa. Am J Psychiatry. 2012;169(10):1031-6.
55. Flegal KM, Kit BK, Orpana H, Graubard BI. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. JAMA. 2013;309(1):71-82.
56. Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. The natural course of bulimia nervosa and binge eating disorder in young women. Arch Gen Psychiatry. 2000;57(7):659-65.
57. Reas DL, Grilo CM. Timing and sequence of the onset of overweight, dieting, and binge eating in overweight patients with binge eating disorder. Int J Eat Disord. 2007;40(2):165-70.
58. Darby A, Hay P, Mond J, Quirk F, Buttner P, Kennedy L. The rising prevalence of comorbid obesity and eating disorder behaviors from 1995 to 2005. Int J Eat Disord. 2009;42(2):104-8.
59. Zachrisson HD, Vedul-Kjelsas E, Gotestam KG, Mykletun A. Time trends in obesity and eating disorders. Int J Eat Disord. 2008;41(8):673-80.

60. Mussell MP, Mitchell JE, Weller CL, Raymond NC, Crow SJ, Crosby RD. Onset of binge eating, dieting, obesity, and mood disorders among subjects seeking treatment for binge eating disorder. Int J Eat Disord. 1995;17(4):395-401.
61. Gwirtsman HE, Kaye WH, Obarzanek E, George DT, Jimerson DC, Ebert MH. Decreased caloric intake in normal-weight patients with bulimia: comparison with female volunteers. Am J Clin Nutr. 1989;49(1):86-92.
62. Obarzanek E, Lesem MD, Goldstein DS, Jimerson DC. Reduced resting metabolic rate in patients with bulimia nervosa. Arch Gen Psychiatry. 1991;48(5):456-62.
63. Castellini G, Castellani W, Lelli L, Sauro CL, Dini C, Lazzeretti L, et al. Association between resting energy expenditure, psychopathology and HPA-axis in eating disorders. World J Clin Cases. 2014;2(7):257-64.
64. de Zwaan M, Aslam Z, Mitchell JE. Research on energy expenditure in individuals with eating disorders: a review. Int J Eat Disord. 2002;32(2):127-34.
65. Altemus M, Hetherington MM, Flood M, Licinio J, Nelson ML, Bernat AS, et al. Decrease in resting metabolic rate during abstinence from bulimic behavior. Am J Psychiatry. 1991;148(8):1071-2.
66. Raymond NC, Peterson RE, Bartholome LT, Raatz SK, Jensen MD, Levine JA. Comparisons of energy intake and energy expenditure in overweight and obese women with and without binge eating disorder. Obesity (Silver Spring). 2012;20(4):765-72.
67. Alger S, Seagle H, Ravussin E. Food intake and energy expenditure in obese female bingers and non-bingers. Int J Obes Relat Metab Disord. 1995;19(1):11-6.
68. Rosenvinge JH, Klusmeier AK. Treatment for eating disorders from a patient satisfaction perspective: a Norwegian replication of a British study. European Eating Disorders Review. 2000;8(4):293-300.
69. Hoek HW, van Hoeken D. Review of the prevalence and incidence of eating disorders. The International journal of eating disorders. 2003;34(4):383-96.
70. Schumann SA. Suspect an eating disorder? Suggest CBT. J Fam Pract. 2009;58(5):265-8.
71. Fairburn CG, Welch SL, Doll HA, Davies BA, O'Connor ME. Risk factors for bulimia nervosa. A community-based case-control study. Arch Gen Psychiatry. 1997;54(6):509-17.
72. Witcher DB, Williamson DA. Duration of bulimia nervosa and symptom progression: a retrospective analysis of treatment-seeking bulimics. J Subst Abuse. 1992;4(3):255-61.
73. Keel PK, Mitchell JE. Outcome in bulimia nervosa. Am J Psychiatry. 1997;154(3):313-21.
74. Wilson GT, Shafran R. Eating disorders guidelines from NICE. Lancet. 2005;365(9453):79-81.
75. Yager J DM, Halmi K, Herzog D, Mitchell J, Powers P, et al. . Practice Guideline for the Treatment of Patients With Eating Disorders. In: Association AP, editor. 3rd ed. Arlington, VA2012.
76. Berkman ND, Bulik CM, Brownley KA, Lohr KN, Sedway JA, Rooks A, et al. Management of eating disorders. Evid Rep Technol Assess (Full Rep). 2006(135):1-166.
77. Wilfley DE, Welch RR, Stein RI, Spurrell EB, Cohen LR, Saelens BE, et al. A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. Arch Gen Psychiatry. 2002;59(8):713-21.
78. Champion L, Power MJ. Interpersonal Psychotherapy for Eating Disorders. Clin Psychol Psychother. 2012;19(2):150-8.
79. Agras WS, Walsh T, Fairburn CG, Wilson GT, Kraemer HC. A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. Arch Gen Psychiatry. 2000;57(5):459-66.

80. Fairburn CG, Marcus, M.D., & Wilson, G.T. . Cognitive-behavioral therapy for binge eating and bulimia nervosa: A comprehensive treatment manual. In: Fairburn CG, & G.T. Wilson (Eds), editor. Binge eating: nature, assessment and treatment. New York: Guilford Press; 1993. p. 361-404.
81. Fairburn CG, Cooper Z, Doll HA, O'Connor ME, Bohn K, Hawker DM, et al. Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: a two-site trial with 60-week follow-up. Am J Psychiatr. 2009;166.
82. Masheb RM, Grilo CM, Rolls BJ. A randomized controlled trial for obesity and binge eating disorder: low-energy-density dietary counseling and cognitive-behavioral therapy. Behav Res Ther. 2011;49(12):821-9.
83. Shapiro JR, Berkman ND, Brownley KA, Sedway JA, Lohr KN, Bulik CM. Bulimia nervosa treatment: a systematic review of randomized controlled trials. Int J Eat Disord. 2007;40(4):321-36.
84. Shelley-Ummenhofer J, MacMillan PD. Cognitive-behavioural treatment for women who binge eat. Can J Diet Pract Res. 2007;68(3):139-42.
85. Wilson GT, Wilfley DE, Agras WS, Bryson SW. Psychological treatments of binge eating disorder. Arch Gen Psychiatry. 2010;67.
86. McElroy SL, Guerdjikova AI, Mori N, Munoz MR, Keck PE. Overview of the treatment of binge eating disorder. CNS Spectr. 2015;20(6):546-56.
87. Larranaga A, Fluiters E, Docet MF, Fernandez Sastre JL, Garcia-Mayor RV. Comparative study of cognitive-behavioral psychotherapy and nutritional support in patients with different types of eating disorders. Med Clin (Barc). 2014;143(5):196-200.
88. Agras WS, Telch CF, Arnow B, Eldredge K, Wilfley DE, Raeburn SD, et al. Weight loss, cognitive-behavioral, and desipramine treatments in binge eating disorder. An additive design. Behav Ther. 1994;25(2):225-38.
89. Grilo CM, Masheb RM, Wilson GT, Gueorguieva R, White MA. Cognitive-behavioral therapy, behavioral weight loss, and sequential treatment for obese patients with binge-eating disorder: a randomized controlled trial. J Consult Clin Psychol. 2011;79(5):675-85.
90. Wilson GT, Grilo CM, Vitousek KM. Psychological treatment of eating disorders. Am Psychol. 2007;62(3):199-216.
91. Hay PP, Bacaltchuk J, Stefano S, Kashyap P. Psychological treatments for bulimia nervosa and binging. Cochrane Database Syst Rev. 2009(4):Cd000562.
92. Iacovino JM, Gredysa DM, Altman M, Wilfley DE. Psychological treatments for binge eating disorder. Curr Psychiatry Rep. 2012;14(4):432-46.
93. Fairburn CG, Agras WS, Walsh BT, Wilson GT, Stice E. Prediction of outcome in bulimia nervosa by early change in treatment. Am J Psychiatry. 2004;161(12):2322-4.
94. U.S. National Institutes of Health. NCI Dictionary of Cancer Terms 2017 [Available from: https://www.cancer.gov/publications/dictionaries/cancer-terms?cdrid=589397.
95. Ozier AD, Henry BW. Position of the American Dietetic Association: nutrition intervention in the treatment of eating disorders. J Am Diet Assoc. 2011;111(8):1236-41.
96. Reiter CS, Graves L. Nutrition therapy for eating disorders. Nutr Clin Pract. 2010;25(2):122-36.
97. Hsu LK, Rand W, Sullivan S, Liu DW, Mulliken B, McDonagh B, et al. Cognitive therapy, nutritional therapy and their combination in the treatment of bulimia nervosa. Psychol Med. 2001;31(5):871-9.
98. Burton E, Stice E. Evaluation of a healthy-weight treatment program for bulimia nervosa: a preliminary randomized trial. Behav Res Ther. 2006;44(12):1727-38.
99. Yanovski SZ, Leet M, Yanovski JA, Flood M, Gold PW, Kissileff HR, et al. Food selection and intake of obese women with binge-eating disorder. Am J Clin Nutr. 1992;56(6):975-80.

100. Sundgot-Borgen J, Rosenvinge JH, Bahr R, Schneider LS. The effect of exercise, cognitive therapy, and nutritional counseling in treating bulimia nervosa. Med Sci Sports Exerc. 2002;34(2):190-5.
101. Hausenblas HA, Cook BJ, Chittester NI. Can exercise treat eating disorders? Exerc Sport Sci Rev. 2008;36(1):43-7.
102. Cook B, Hausenblas H, Crosby RD, Cao L, Wonderlich SA. Exercise dependence as a mediator of the exercise and eating disorders relationship: a pilot study. Eat Behav. 2015;16:9-12.
103. Vancampfort D, Vanderlinden J, De Hert M, Adamkova M, Skjaerven LH, Catalan-Matamoros D, et al. A systematic review on physical therapy interventions for patients with binge eating disorder. Disabil Rehabil. 2013;35(26):2191-6.
104. Vancampfort D, Vanderlinden J, De Hert M, Soundy A, Adamkova M, Skjaerven LH, et al. A systematic review of physical therapy interventions for patients with anorexia and bulemia nervosa. Disabil Rehabil. 2014;36(8):628-34.
105. Bratland-Sanda S, Martinsen EW, Rosenvinge JH, Ro O, Hoffart A, Sundgot-Borgen J. Exercise dependence score in patients with longstanding eating disorders and controls: the importance of affect regulation and physical activity intensity. Eur Eat Disord Rev. 2011;19(3):249-55.
106. Shroff H, Reba L, Thornton LM, Tozzi F, Klump KL, Berrettini WH, et al. Features associated with excessive exercise in women with eating disorders. Int J Eat Disord. 2006;39(6):454-61.
107. Bratland-Sanda S, Sundgot-Borgen J, Ro O, Rosenvinge JH, Hoffart A, Martinsen EW. Physical activity and exercise dependence during inpatient treatment of longstanding eating disorders: an exploratory study of excessive and non-excessive exercisers. Int J Eat Disord. 2010;43(3):266-73.
108. Cook B, Engel S, Crosby R, Hausenblas H, Wonderlich S, Mitchell J. Pathological motivations for exercise and eating disorder specific health-related quality of life. Int J Eat Disord. 2014;47(3):268-72.
109. Levine MD, Marcus MD, Moulton P. Exercise in the treatment of binge eating disorder. Int J Eat Disord. 1996;19(2):171-7.
110. Pendleton VR, Goodrick GK, Poston WS, Reeves RS, Foreyt JP. Exercise augments the effects of cognitive-behavioral therapy in the treatment of binge eating. Int J Eat Disord. 2002;31(2):172-84.
111. Alpers GW, Tuschen-Caffier B. Negative feelings and the desire to eat in bulimia nervosa. Eat Behav. 2001;2(4):339-52.
112. Vancampfort D, Probst M, Adriaens A, Pieters G, De Hert M, Stubbs B, et al. Changes in physical activity, physical fitness, self-perception and quality of life following a 6-month physical activity counseling and cognitive behavioral therapy program in outpatients with binge eating disorder. Psychiatry Res. 2014;219(2):361-6.
113. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. Obes Res. 1998;6 Suppl 2:51s-209s.
114. Miller WC, Koceja DM, Hamilton EJ. A meta-analysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. Int J Obes Relat Metab Disord. 1997;21(10):941-7.
115. Annesi JJ, Vaughn LL. Relationship of Exercise Volume with Change in Depression and Its Association with Self-Efficacy to Control Emotional Eating in Severely Obese Women. Adv Prev Med. 2011;2011.

116. Andrade AM, Coutinho SR, Silva MN, Mata J, Vieira PN, Minderico CS, et al. The effect of physical activity on weight loss is mediated by eating self-regulation. Patient Educ Couns. 2010;79(3):320-6.
117. Martins C, Robertson MD, Morgan LM. Effects of exercise and restrained eating behaviour on appetite control. Proc Nutr Soc. 2008;67(1):28-41.
118. Martins C, Stensvold D, Finlayson G, Holst J, Wisloff U, Kulseng B, et al. Effect of moderate- and high-intensity acute exercise on appetite in obese individuals. Med Sci Sports Exerc. 2015;47(1):40-8.
119. Wilson GT. Treatment of binge eating disorder. Psychiatr Clin North Am. 2011;34(4):773-83.
120. Palavras MA, Hay P, Touyz S, Sainsbury A, da Luz F, Swinbourne J, et al. Comparing cognitive behavioural therapy for eating disorders integrated with behavioural weight loss therapy to cognitive behavioural therapy-enhanced alone in overweight or obese people with bulimia nervosa or binge eating disorder: study protocol for a randomised controlled trial. Trials. 2015;16:578.
121. da Luz FQ, Swinbourne J, Sainsbury A, Touyz S, Palavras M, Claudino A, et al. HAPIFED: a Healthy APproach to weIght management and Food in Eating Disorders: a case series and manual development. J Eat Disord. 2017;5:29.
122. Fossati M, Amati F, Painot D, Reiner M, Haenni C, Golay A. Cognitive-behavioral therapy with simultaneous nutritional and physical activity education in obese patients with binge eating disorder. Eat Weight Disord. 2004;9(2):134-8.
123. Bond MJ, McDowell AJ, Wilkinson JY. The measurement of dietary restraint, disinhibition and hunger: an examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). Int J Obes Relat Metab Disord. 2001;25(6):900-6.
124. Cappelleri JC, Bushmakin AG, Gerber RA, Leidy NK, Sexton CC, Lowe MR, et al. Psychometric analysis of the Three-Factor Eating Questionnaire-R21: results from a large diverse sample of obese and non-obese participants. Int J Obes (Lond). 2009;33(6):611-20.
125. Gade H, Rosenvinge JH, Hjelmesaeth J, Friborg O. Psychological correlates to dysfunctional eating patterns among morbidly obese patients accepted for bariatric surgery. Obes Facts. 2014;7(2):111-9.
126. Johnson F, Pratt M, Wardle J. Dietary restraint and self-regulation in eating behavior. Int J Obes (Lond). 2012;36(5):665-74.
127. Dietrich A, Federbusch M, Grellmann C, Villringer A, Horstmann A. Body weight status, eating behavior, sensitivity to reward/punishment, and gender: relationships and interdependencies. Front Psychol. 2014;5:1073.
128. Loffler A, Luck T, Then FS, Sikorski C, Kovacs P, Bottcher Y, et al. Eating Behaviour in the General Population: An Analysis of the Factor Structure of the German Version of the Three-Factor-Eating-Questionnaire (TFEQ) and Its Association with the Body Mass Index. PLoS One. 2015;10(7):e0133977.
129. Karlsson J, Persson LO, Sjostrom L, Sullivan M. Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. Int J Obes Relat Metab Disord. 2000;24(12):1715-25.
130. Bryant EJ, King NA, Blundell JE. Disinhibition: its effects on appetite and weight regulation. Obes Rev. 2008;9(5):409-19.
131. Westenhoefer J, Broeckmann P, Munch AK, Pudel V. Cognitive control of eating behaviour and the disinhibition effect. Appetite. 1994;23(1):27-41.
132. Lahteenmaki L, Tuorila H. Three-factor eating questionnaire and the use and liking of sweet and fat among dieters. Physiol Behav. 1995;57(1):81-8.

133. Contento IR, Zybert P, Williams SS. Relationship of cognitive restraint of eating and disinhibition to the quality of food choices of Latina women and their young children. Prev Med. 2005;40(3):326-36.
134. Van Strien T, Cleven A, Schippers G. Restraint, tendency toward overeating and ice cream consumption. Int J Eat Disord. 2000;28(3):333-8.
135. Cuntz U, Leibbrand R, Ehrig C, Shaw R, Fichter MM. Predictors of post-treatment weight reduction after in-patient behavioral therapy. Int J Obes Relat Metab Disord. 2001;25 Suppl 1:S99-s101.
136. Karlsson J, Hallgren P, Kral J, Lindroos AK, Sjostrom L, Sullivan M. Predictors and effects of long-term dieting on mental well-being and weight loss in obese women. Appetite. 1994;23(1):15-26.
137. Geliebter A, Aversa A. Emotional eating in overweight, normal weight, and underweight individuals. Eat Behav. 2003;3(4):341-7.
138. Ganley RM. Emotion and eating in obesity: A review of the literature. Int J Eat Disord. 1989;8:343-61.
139. Zeeck A, Stelzer N, Linster HW, Joos A, Hartmann A. Emotion and eating in binge eating disorder and obesity. Eur Eat Disord Rev. 2011;19(5):426-37.
140. Nicholls W, Devonport TJ, Blake M. The association between emotions and eating behaviour in an obese population with binge eating disorder. Obes Rev. 2016;17(1):30-42.
141. Waller G, Osman S. Emotional eating and eating psychopathology among non-eating-disordered women. Int J Eat Disord. 1998;23(4):419-24.
142. Grilo CM, Shiffman S., Wing R. R. . Relapse crises and coping among dieters. J Consult Clin Psychol. 1989;4(Aug;57):488-95.
143. Dykes J, Brunner EJ, Martikainen PT, Wardle J. Socioeconomic gradient in body size and obesity among women: the role of dietary restraint, disinhibition and hunger in the Whitehall II study. Int J Obes Relat Metab Disord. 2004;28(2):262-8.
144. Masheb RM, Grilo CM, White MA. An examination of eating patterns in community women with bulimia nervosa and binge eating disorder. Int J Eat Disord. 2011;44(7):618-24.
145. Blomquist KK, Grilo CM. Predictive significance of changes in dietary restraint in obese patients with binge eating disorder during treatment. Int J Eat Disord. 2011;44(6):515-23.
146. Grilo CM, Crosby RD, Masheb RM, White MA, Peterson CB, Wonderlich SA, et al. Overvaluation of shape and weight in binge eating disorder, bulimia nervosa, and sub-threshold bulimia nervosa. Behav Res Ther. 2009;47(8):692-6.
147. Downe KA, Goldfein JA, Devlin MJ. Restraint, hunger, and disinhibition following treatment for binge-eating disorder. Int J Eat Disord. 2009;42(6):498-504.
148. Masheb RM, Grilo CM. On the relation of flexible and rigid control of eating to body mass index and overeating in patients with binge eating disorder. Int J Eat Disord. 2002;31(1):82-91.
149. Ardovini C, Caputo G, Todisco P, Dalle Grave R. Binge eating and restraint model: psychometric analysis in binge eating disorder and normal weight bulimia. European Eating Disorders Review. 1999;7(4):293-9.
150. Danner UN, Evers C, Stok FM, van Elburg AA, de Ridder DT. A double burden: emotional eating and lack of cognitive reappraisal in eating disordered women. Eur Eat Disord Rev. 2012;20(6):490-5.
151. Kittel R, Brauhardt A, Hilbert A. Cognitive and emotional functioning in binge-eating disorder: A systematic review. Int J Eat Disord. 2015;48(6):535-54.
152. Leehr EJ, Krohmer K, Schag K, Dresler T, Zipfel S, Giel KE. Emotion regulation model in binge eating disorder and obesity--a systematic review. Neurosci Biobehav Rev. 2015;49:125-34.

153. Deaver C, G Miltenberger R, Smyth J, Meidinger A, Crosby R. An Evaluation of Affect and Binge Eating2003. 578-99 p.
154. Heatherton TF, Baumeister RF. Binge eating as escape from self-awareness. Psychol Bull. 1991;110(1):86-108.
155. Haedt-Matt AA, Keel PK. Revisiting the affect regulation model of binge eating: a meta-analysis of studies using ecological momentary assessment. Psychol Bull. 2011;137(4):660-81.
156. Hilbert A, Tuschen-Caffier B. Maintenance of binge eating through negative mood: a naturalistic comparison of binge eating disorder and bulimia nervosa. Int J Eat Disord. 2007;40(6):521-30.
157. De Young KP, Zander M, Anderson DA. Beliefs about the emotional consequences of eating and binge eating frequency. Eat Behav. 2014;15(1):31-6.
158. Mitchell JE, Mussell MP, Peterson CB, Crow S, Wonderlich SA, Crosby RD, et al. Hedonics of binge eating in women with bulimia nervosa and binge eating disorder. Int J Eat Disord. 1999;26(2):165-70.
159. L. Cooper J, Professor LMPD, vice Chair T, L. Bigman O, I. Abramowitz S, Levin S, et al. Mood changes and affective disorder in the bulimic binge—Purge cycle1988. 469-74 p.
160. Walsh BT. Binge eating in bulimia nervosa. Binge eating: Nature, assessment, and treatment. New York, NY, US: Guilford Press; 1993. p. 37-49.
161. Weltzin TE, Hsu LK, Pollice C, Kaye WH. Feeding patterns in bulimia nervosa. Biol Psychiatry. 1991;30(11):1093-110.
162. Goldfein JA, Walsh BT, LaChaussee JL, Kissileff HR, Devlin MJ. Eating behavior in binge eating disorder. Int J Eat Disord. 1993;14(4):427-31.
163. Yanovski SZ, Sebring NG. Recorded food intake of obese women with binge eating disorder before and after weight loss. Int J Eat Disord. 1994;15(2):135-50.
164. Rossiter E, Agras, SW., Telch CF. The eating patterns of non-purging bulimic subjects1992. 111-20 p.
165. Timmerman GM. Caloric Intake Patterns of Nonpurge Binge-Eating Women. West J Nurs Res. 1998;20(1):103-18.
166. Brody J. Jane Brody's nutrition book New York: Bantam Books; 1987.
167. Helsedirektoratet. Norkost 3 En landsomfattende kostholdsundersøkelse blant menn og kvinner i Norge i alderen 18-70 år, 2010-11. 2012 06/2012.
168. Raymond NC, Bartholome LT, Lee SS, Peterson RE, Raatz SK. A comparison of energy intake and food selection during laboratory binge eating episodes in obese women with and without a binge eating disorder diagnosis. Int J Eat Disord. 2007;40(1):67-71.
169. Segura-García C, De Fazio P, Sinopoli F, De Masi R, Brambilla F. Food choice in disorders of eating behavior: Correlations with the psychopathological aspects of the diseases. Compr Psychiatry.55(5):1203-11.
170. Setnick J. Micronutrient deficiencies and supplementation in anorexia and bulimia nervosa: a review of literature. Nutr Clin Pract. 2010;25(2):137-42.
171. Lakhan SE, Vieira KF. Nutritional therapies for mental disorders. Nutr J. 2008;7:2.
172. Aparicio E, Canals J, Perez S, Arija V. Dietary intake and nutritional risk in Mediterranean adolescents in relation to the severity of the eating disorder. Public Health Nutr. 2015;18(8):1461-73.
173. Nordic Council of Ministers. Nordic Nutrition Recommendations 2012 - Integrating nutrition and physical activity. 5 ed. Ministers NCo, editor: Norden; 2014.
174. Modan-Moses D, Levy-Shraga Y, Pinhas-Hamiel O, Kochavi B, Enoch-Levy A, Vered I, et al. High prevalence of vitamin D deficiency and insufficiency in adolescent inpatients diagnosed with eating disorders. Int J Eat Disord. 2015;48(6):607-14.

175. Ardovini C, Caputo G, Todisco P, Dalle Grave R. Binge eating and restraint model: Psychometric analysis in binge eating disorder and normal weight bulimia1999. 293-9 p.
176. Borg P, Fogelholm M, Kukkonen-Harjula K. Food selection and eating behaviour during weight maintenance intervention and 2-y follow-up in obese men. Int J Obes Relat Metab Disord. 2004;28(12):1548-54.
177. Wilson PL, O'Connor DP, Kaplan CD, Bode S, Mama SK, Lee RE. Relationship of fruit, vegetable, and fat consumption to binge eating symptoms in African American and Hispanic or Latina women. Eat Behav. 2012;13(2):179-82.
178. Latner J. Macronutrient effects on satiety and binge eating in bulimia nervosa and binge eating disorder2003. 309-11 p.
179. Masheb RM, Grilo CM. Eating patterns and breakfast consumption in obese patients with binge eating disorder. Behav Res Ther. 2006;44(11):1545-53.
180. Kerver JM, Yang EJ, Obayashi S, Bianchi L, Song WO. Meal and snack patterns are associated with dietary intake of energy and nutrients in US adults. J Am Diet Assoc. 2006;106(1):46-53.
181. Zendegui EA, West JA, Zandberg LJ. Binge eating frequency and regular eating adherence: the role of eating pattern in cognitive behavioral guided self-help. Eat Behav. 2014;15(2):241-3.
182. Ellison JM, Simonich HK, Wonderlich SA, Crosby RD, Cao L, Mitchell JE, et al. Meal patterning in the treatment of bulimia nervosa. Eat Behav. 2016;20:39-42.
183. Helsedirektoratet. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer - Metodologi og vitenskapelig kunnskapsgrunnlag. Oslo: Nordic 2011 01.01.2011.
184. Helsedirektoratet. Anbefalinger om kosthold, ernæring og fysisk aktivitet. Oslo2014.
185. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-5), 5.th edn. American Psychiatric Pub; 2013.
186. Freitas SR, Lopes CS, Appolinario JC, Coutinho W. The assessment of binge eating disorder in obese women: a comparison of the binge eating scale with the structured clinical interview for the DSM-IV. Eat Behav. 2006;7.
187. Fairburn CG, Cooper Z, Doll HA, O'Connor ME, Bohn K, Hawker DM, et al. Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: a two-site trial with 60-week follow-up. The American journal of psychiatry. 2009;166(3):311-9.
188. Cook B, Wonderlich SA, Mitchell J, Thompson R, Sherman R, McCallum K. Exercise in eating disorders treatment: systematic review and proposal of guidelines. Med Sci Sports Exerc. 2016;48.
189. World Health Association (WHO). Global recommendations on physical activity for health. 2010.
190. Garber CE, Blissmer B, Deschenes MR, Franklin BA, Lamonte MJ, Lee IM, et al. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Medicine and science in sports and exercise. 2011;43.
191. Loveless MS, Ihm JM. Resistance exercise: how much is enough? Curr Sports Med Rep. 2015;14.
192. Helsedirektoratet. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer - Metodologi og vitenskapelig kunnskapsgrunnlag. Oslo; 2011.
193. IOC consensus statement on sports nutrition 2010. J Sports Sci. 2011;29 Suppl 1:S3-4.
194. Gormally J, Black S, Daston S, Rardin D. The assessment of binge eating severity among obese persons. Addict Behav. 1982;7(1):47-55.

195. Marcus MD, Wing RR, Hopkins J. Obese binge eaters: affect, cognitions, and response to behavioural weight control. J Consult Clin Psychol. 1988;56(3):433-9.
196. Cappelleri JC, Zou, K. H., Bushmakin, A. G., Alvir, J. M. J., Alemayehu, D., Symonds, T. Patient-Reported Outcomes: Measurement, Implementation and Interpretation. Series CHCB, editor2014.
197. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res. 1985;29(1):71-83.
198. de Lauzon B, Romon M, Deschamps V, Lafay L, Borys JM, Karlsson J, et al. The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in a general population. J Nutr. 2004;134(9):2372-80.
199. Hyland ME, Irvine, Sidney H., Thacker, Clive, Dann, Peter L., Dennis, Ian. Psychometric analysis of the Stunkard-Messick Eating Questionnaire (SMEQ) and Comparison with the Dutch Eating Behavior Questionnaire (DEBQ). Current Psychology. 1989;8(3):228-33.
200. Tholin S, Rasmussen F, Tynelius P, Karlsson J. Genetic and environmental influences on eating behavior: the Swedish Young Male Twins Study. Am J Clin Nutr. 2005;81(3):564-9.
201. Shim JS, Oh K, Kim HC. Dietary assessment methods in epidemiologic studies. Epidemiol Health. 2014;36:e2014009.
202. Buzzard IM, Faucett CL, Jeffery RW, McBane L, McGovern P, Baxter JS, et al. Monitoring dietary change in a low-fat diet intervention study: advantages of using 24-hour dietary recalls vs food records. J Am Diet Assoc. 1996;96(6):574-9.
203. Salvador Castell G, Serra-Majem L, Ribas-Barba L. What and how much do we eat? 24-hour dietary recall method. Nutr Hosp. 2015;31 Suppl 3:46-8.
204. Gemming L, Utter J, Ni Mhurchu C. Image-assisted dietary assessment: a systematic review of the evidence. J Acad Nutr Diet. 2015;115(1):64-77.
205. Lillegaard IT, Overby NC, Andersen LF. Can children and adolescents use photographs of food to estimate portion sizes? Eur J Clin Nutr. 2005;59(4):611-7.
206. Callmer E HU, Haraldsdóttir J, Bjørge Løken E, Seppänen R, Trygg K,. Standardisation of 24-hour recall. Vår föda. 1986;38 (Suppl 4):201-98.
207. Norwegian Directorate of Health and The Norwegian Food Safety Authority. Kostholdsplanleggeren: Norwegian Directorate of Health and the Norwegian Food Safety Authority; [Available from: https://www.kostholdsplanleggeren.no/.
208. Norwegian Food Safety Authority. Norwegian Food Composition Database 2016: The Norwegian Directorate of Health and the University of Oslo; 2016 [Available from: www.matvaretabellen.no.
209. The Norwegian Food Safety Authority and The Norwegian Directorate of Health. Mål, vekt og porsjonsstørrelser for matvarer. 2015.
210. Dalton M, Finlayson G. Psychobiological examination of liking and wanting for fat and sweet taste in trait binge eating females. Physiol Behav. 2014;136:128-34.
211. Schwingshackl L, Schwedhelm, C., Hoffmann, G., Lampousi, A. M., Knuppel, S., Iqbal, K., Bechthold, A., Schlesinger, S., Boeing, H. Food groups and risk of all-cause mortality: a systematic review and meta-analysis of prospective studies. Am J Clin Nutr. 2017.
212. Meinila J, Valkama A, Koivusalo SB, Stach-Lempinen B, Lindstrom J, Kautiainen H, et al. Healthy Food Intake Index (HFII) - Validity and reproducibility in a gestational-diabetes-risk population. BMC Public Health. 2016;16:680.
213. Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HA, Kuczynski KJ, et al. Update of the Healthy Eating Index: HEI-2010. J Acad Nutr Diet. 2013;113(4):569-80.

214. Olsen A, Egeberg R, Halkjaer J, Christensen J, Overvad K, Tjonneland A. Healthy aspects of the Nordic diet are related to lower total mortality. J Nutr. 2011;141(4):639-44.
215. Frayn KN. Energy Balance and Body Weight Regulation. In: Snell K, editor. Metabolic Regulation - A Human Perspective. 3rd ed: Wiley-Blackwell; 2010. p. 329-50.
216. Harris JA, Benedict FG. A Biometric Study of Human Basal Metabolism. Proc Natl Acad Sci U S A. 1918;4(12):370-3.
217. Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr. 1990;51(2):241-7.
218. Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. J Am Diet Assoc. 2005;105(5):775-89.
219. Cohen J. Statistical Power Analysis for the Behavioral Sciences. Hillsdale, NJ: Lawrence Erlbaum Associates; 1988.
220. Pallant J. SPSS Survival Manual - A step by step guide to data analysis using IBM SPSS. 6th ed: A&U Academic; 2016.
221. Sodersten P, Bergh C, Leon M, Brodin U, Zandian M. Cognitive behavior therapy for eating disorders versus normalization of eating behavior. Physiol Behav. 2017;174:178-90.
222. Herzog DB, Dorer DJ, Keel PK, Selwyn SE, Ekeblad ER, Flores AT, et al. Recovery and relapse in anorexia and bulimia nervosa: a 7.5-year follow-up study. J Am Acad Child Adolesc Psychiatry. 1999;38(7):829-37.
223. Faul F, Erdfelder, E., Lang, A.-G. & Buchner, A. . G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behav Res Methods. 2007;39:175-91.
224. Schlup B, Meyer AH, Munsch S. A non-randomized direct comparison of cognitive-behavioral short- and long-term treatment for binge eating disorder. Obes Facts. 2010;3(4):261-6.
225. Bohrer BK, Forbush KT, Hunt TK. Are common measures of dietary restraint and disinhibited eating reliable and valid in obese persons? Appetite. 2015;87:344-51.
226. van Strien T, Frijters JER, Bergers GPA, Defares PB. The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. Int J Eat Disord. 1986;5(2):295-315.
227. Fairburn CG, Beglin SJ. Assessment of eating disorders: interview or self-report questionnaire? Int J Eat Disord. 1994;16(4):363-70.
228. Luce KH, Crowther JH, Pole M. Eating Disorder Examination Questionnaire (EDE-Q): norms for undergraduate women. Int J Eat Disord. 2008;41(3):273-6.
229. Reas DL, Grilo CM, Masheb RM. Reliability of the Eating Disorder Examination-Questionnaire in patients with binge eating disorder. Behav Res Ther. 2006;44(1):43-51.
230. Rø Ø, Reas DL, Stedal K. Eating Disorder Examination Questionnaire (EDE-Q) in Norwegian Adults: Discrimination between Female Controls and Eating Disorder Patients. European Eating Disorders Review. 2015;23(5):408-12.
231. Safer DL, Agras WS, Lowe MR, Bryson S. Comparing two measures of eating restraint in bulimic women treated with cognitive-behavioral therapy. Int J Eat Disord. 2004;36(1):83-8.
232. Westenhoefer J. Dietary restraint and disinhibition: is restraint a homogeneous construct? Appetite. 1991;16(1):45-55.
233. Hill RJ, Davies PS. The validity of self-reported energy intake as determined using the doubly labelled water technique. Br J Nutr. 2001;85(4):415-30.

234. Subar AF, Freedman LS, Tooze JA, Kirkpatrick SI, Boushey C, Neuhouser ML, et al. Addressing Current Criticism Regarding the Value of Self-Report Dietary Data. J Nutr. 2015;145(12):2639-45.
235. Natarajan L, Pu M, Fan J, Levine RA, Patterson RE, Thomson CA, et al. Measurement error of dietary self-report in intervention trials. Am J Epidemiol. 2010;172(7):819-27.
236. Reeves RS, McPherson RS, Nichaman MZ, Harrist RB, Foreyt JP, Goodrick GK. Nutrient intake of obese female binge eaters. J Am Diet Assoc. 2001;101(2):209-15.
237. Ma Y, Olendzki BC, Pagoto SL, Hurley TG, Magner RP, Ockene IS, et al. Number of 24-hour diet recalls needed to estimate energy intake. Ann Epidemiol. 2009;19(8):553-9.
238. Hoopes SP, Reimherr FW, Hedges DW, Rosenthal NR, Kamin M, Karim R, et al. Treatment of bulimia nervosa with topiramate in a randomized, double-blind, placebo-controlled trial, part 1: improvement in binge and purge measures. J Clin Psychiatry. 2003;64(11):1335-41.
239. Masheb RM, Dorflinger LM, Rolls BJ, Mitchell DC, Grilo CM. Binge abstinence is associated with reduced energy intake after treatment in patients with binge eating disorder and obesity. Obesity (Silver Spring). 2016;24(12):2491-6.
240. Friederich HC, Schild S, Wild B, de Zwaan M, Quenter A, Herzog W, et al. Treatment outcome in people with subthreshold compared with full-syndrome binge eating disorder. Obesity (Silver Spring). 2007;15(2):283-7.
241. Kiernan M, King AC, Stefanick ML, Killen JD. Men gain additional psychological benefits by adding exercise to a weight-loss program. Obes Res. 2001;9(12):770-7.
242. Rosenbaum M, Leibel RL. Adaptive thermogenesis in humans. Int J Obes (Lond). 2010;34 Suppl 1:S47-55.
243. Blundell JE, Gibbons C, Caudwell P, Finlayson G, Hopkins M. Appetite control and energy balance: impact of exercise. Obes Rev. 2015;16 Suppl 1:67-76.
244. Schubert MM, Sabapathy S, Leveritt M, Desbrow B. Acute exercise and hormones related to appetite regulation: a meta-analysis. Sports Med. 2014;44(3):387-403.
245. Vist GE JS, Straumann GH, Ding KY, Reinar LM. Cognitive behaviour therapy compared to other psychotherapies for treatment of bulimia nervosa. . 2016.
246. Keim NL, Canty DJ, Barbieri TF, Wu MM. Effect of exercise and dietary restraint on energy intake of reduced-obese women. Appetite. 1996;26(1):55-70.
247. Visona C, George VA. Impact of dieting status and dietary restraint on postexercise energy intake in overweight women. Obes Res. 2002;10(12):1251-8.
248. Kaufer-Horwitz M, Villa, M., Pedraza, J., Dominguez-Garcia, J., Vazquez-Velazquez, V., Mendez, J. P., Garcia-Garcia, E. Knowledge of appropriate foods and beverages needed for weight loss and diet of patients in an Obesity Clinic. Eur J Clin Nutr. 2015;69(1):68-72.
249. Laessle RG, Scweiger, R., Daute-Herold, U., Schweiger, M., Fichter, M. M., & Pirke, K. M. Nutritional knowledge in patients with eating disorders. Int J Eat Disord. 1988;7(1):63-73.
250. Soh NL, Touyz SW, Dobbins TA, Surgenor LJ, Clarke S, Kohn MR, et al. Nutrition knowledge in young women with eating disorders in Australia and Singapore: a pilot study. Aust N Z J Psychiatry. 2009;43(12):1178-84.
251. National Institute for Clinical Excellence. Eating Disorders: Core Interventions in the Treatment and Management of Anorexia Nervosa, Bulimia Nervosa and Related Eating Disorders. 2004(35).
252. Guss JL, Kissilef HR, Walsh BT, Devlin MJ. Binge eating behavior in patients with eating disorders. Obes Res. 1994;2(4):355-63.
253. Raymond NC, Neumeyer B, Warren CS, Lee SS, Peterson CB. Energy Intake Patterns in Obese Women with Binge Eating Disorder. Obes Res. 2003;11(7):869-79.

254. Drenowatz C. Reciprocal Compensation to Changes in Dietary Intake and Energy Expenditure within the Concept of Energy Balance. Adv Nutr. 2015;6(5):592-9.
255. Douglas JA, King JA, McFarlane E, Baker L, Bradley C, Crouch N, et al. Appetite, appetite hormone and energy intake responses to two consecutive days of aerobic exercise in healthy young men. Appetite. 2015;92:57-65.
256. Hall KD, Sacks G, Chandramohan D, Chow CC, Wang YC, Gortmaker SL, et al. Quantification of the effect of energy imbalance on bodyweight. Lancet. 2011;378(9793):826-37.
257. Pourhassan M, Bosy-Westphal A, Schautz B, Braun W, Gluer CC, Muller MJ. Impact of body composition during weight change on resting energy expenditure and homeostasis model assessment index in overweight nonsmoking adults. Am J Clin Nutr. 2014;99(4):779-91.
258. Dashti S, Esfehani AJ, Leonard Joseph HJ. The preventive effect of physical activity on weight maintenance in overweight and obese women. Clin Ter. 2014;165(3):155-61.
259. Johns DJ, Hartmann-Boyce J, Jebb SA, Aveyard P. Diet or exercise interventions vs combined behavioral weight management programs: a systematic review and meta-analysis of direct comparisons. J Acad Nutr Diet. 2014;114(10):1557-68.
260. King NA, Horner K, Hills AP, Byrne NM, Wood RE, Bryant E, et al. Exercise, appetite and weight management: understanding the compensatory responses in eating behaviour and how they contribute to variability in exercise-induced weight loss. Br J Sports Med. 2012;46(5):315-22.

Appendices

Appendix 1 – Diagnostic criteria for featured eating disorders (1)

Anorexia Nervosa

Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. *Significantly low weight* is defined as a weight that is less than minimally normal or, for children and adolescents, less than minimally expected.

Intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight.

Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

F50.01 Restricting type: During the last 3 months, the individual has not engaged in recurrent episodes of binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas). The subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting, and/or excessive exercise.

F50.02 Binge-eating/purging type: During the last 3 months, the individual has engaged in recurrent episodes of binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

Severity: The minimum level of severity is based, for adult, on current body mass index (BMI) (see below) or, for children and adolescents, on BMI percentile.

Mild: BMI $\geq$ 17 kg/m^2

Moderate: BMI 16-16.99 kg/m^2

Severe: BMI 15-15.99 kg/m^2

Extreme: BMI < 15 kg/m^2

Bulimia Nervosa F50.2

Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

-Eating, in a discrete period of time (e.g. within any 2-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.

-A sense of lack of control over eating during the episode (e.g. a feeling that one cannot stop eating or control what or how much one is eating).

Recurrent inappropriate compensatory behaviors in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise.

The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for 3 months.

Self-evaluation is unduly influenced by body shape and weight.

The disturbance does not occur exclusively during episodes of anorexia nervosa.

Severity: The minimum level of severity is based on the frequency of inappropriate compensatory behaviors (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: An average of 1-3 episodes of inappropriate compensatory behaviors per week

Moderate: An average of 4-7 episodes of inappropriate compensatory behaviors per week

Severe: An average of 8-13 episodes of inappropriate compensatory behaviors per week

Extreme: An average of 14 or more episodes of inappropriate compensatory behaviors per week

Binge-Eating Disorder F50.8

Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

-Eating, in a discrete period of time (e.g. within any 2-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.

-A sense of lack of control over eating during the episode (e.g. a feeling that one cannot stop eating or control what or how much one is eating).

The binge-eating episodes are associated with three (or more) of the following:

Eating much more rapidly than normal. Eating until feeling uncomfortably full. Eating large amounts of food when not feeling physically hungry. Eating alone because of feeling embarrassed by how much one is eating. Feeling disgusted with oneself, depressed, or very guilty afterward. Marked distress regarding binge eating is present.

The binge eating occurs, on average, at least once a week for 3 months. The binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

Severity: The minimum level of severity is based on the frequency of episodes of binge eating (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: 1-3 binge-eating episodes per week

Moderate: 4-7 binge-eating episodes per week

Severe: 8-13 binge-eating episodes per week

Extreme: 14 or more binge-eating episodes per week

Appendix 2 – Excerpt of the quantitative dietary recommendations, compared to average intake of the Norwegian female population (183)

Food and nutrient	Quantitative recommendation	Average intake among Norwegian women (167)
Fruits, berries and vegetables, g/d	≥ 500	387
Wholegrain products, g/d	≥ 70	52
Fish, g/w	300-450	308
Fat fish, g/w	200	154
Red meat and processed meat, g/w	≤ 750	623
Added sugar, E%	≤ 10	7.4
Salt, g/d	≤ 6	6.3

Appendix 3 – Recommended intakes (RI) of macronutrients[1] and featured vitamins and minerals[2], and their potential health benefits (173, 184)

	RI (per day)	Potential health benefits
Macronutrients		
Total fat, E%	25-40	Weight reduction/-management
Saturated fatty acids, E%	≤ 10	Reduced LDL-cholesterol and LDL/HDL-ratio. Reduced risk of coronary heart disease (CHD).
Cis-monounsaturated fatty acids, E%	10-20	
Cis-polyunsaturated fatty acids, E%	5-10 ≥ 1 n-3 fatty acids	Provide enough fat-soluble vitamins and essential fatty acids.
Dietary fiber, g	25-35	Reduced risk of constipation, colorectal cancer, cardiovascular diseases and type-2 diabetes.
Added sugars, E%	≤ 10	Reduced risk of type-2 diabetes, reduced weight and dental caries. Maintain healthy body weight.
Protein, E%	10-20	Provide enough essential amino acids
Salt, g	≤ 6	Reduced risk of high blood pressure, stroke and CVD
Alcohol, E%	≤ 5	Prevent impaired nutritional quality of the diet
Vitamins and minerals		

Vitamin D, µg	10[3]	Reduced risk of ostemalacia. Together with Calcium: maintained bone mineral density (BMD) and reduced mortality.
Calcium, mg	800[5]	Together with vitamin D: maintained BMD, and reduced mortality.
Folate, µg	400/300[4]	Reduced risk of neural tube defects during pregnancy, and CVD. Protection against poor cognitive function and neurological disorders.
Vitamin C, mg	75	Reduced risk of scurvy, chronic diseases and mortality.
Iron, mg	15[6]	Reduced risk of iron deficiency anemia (IDA), reduced work capacity and impaired cell-mediated immunological defense.

[1]Adults and children from 2 years of age. [2]Females 18-30/31-60 years. [3]20 µg/d for people with little or no sun exposure. [4]400 µg/d for women of reproductive age. [5]900 mg/d for 18-20-year old's. [6]Some women require a larger iron supply than others due to menstrual iron losses, and more than the habitual diet can supply. 9 mg/d for post-menopausal women.

Appendix 4 – The Binge Eating Scale

Navn:	
Screeningnr:	
Random.nr:	
Dato:	

BES

VEILEDNING:
På de følgende sider er det grupper av nummererte uttalelser. Les alle uttalelser i hver gruppe og sett et kryss ved den uttalelse i hver gruppe som best beskriver dine følelser i forhold til de problemer du har med å kontrollere dine spisevaner.

1.

1.	☐	Jeg er ikke flau over vekten min eller størrelsen på kroppen min når jeg er sammen med andre.
2.	☐	Jeg tenker på hvordan andre ser meg, men det gjør meg normalt ikke skuffet over meg selv.
3.	☐	Jeg blir flau over mitt utseende og vekten min, og det gjør meg skuffet over meg selv.
4.	☐	Jeg er veldig flau over vekten min og jeg føler ofte dyp skam og avsky for meg selv. Jeg prøver å unngå kontakt med mennesker, fordi jeg er så flau.

2.

1.	☐	Jeg har ingen vanskeligheter med å spise behersket og sakte.
2.	☐	Selv om jeg later til å "sluke" maten, føler jeg meg ikke overmett fordi jeg har spist for mye.
3.	☐	Noen ganger har jeg en tendens til å spise fort, og da føler jeg meg ubehagelig mett etterpå.
4.	☐	Jeg har for vane å sluke maten, uten å tygge den ordentlig. Når jeg gjør det, føler jeg meg som regel ubehagelig overmett, fordi jeg har spist for mye.

3.

1.	☐	Jeg føler at jeg kan beherske min spisetrang, når jeg vil.
2.	☐	Jeg har en følelse av at jeg er dårligere til å beherske spisingen min enn gjennomsnittsmenneske.
3.	☐	Jeg føler meg helt hjelpeløs når det gjelder å beherske min spisetrang.
4.	☐	Fordi jeg føler meg så hjelpeløs når det gjelder å beherske spisingen min, er jeg blitt helt desperat for å prøve å få kontroll.

4.

1.	☐	Jeg har ikke for vane å spise, når jeg kjeder meg.
2.	☐	Iblant spiser jeg, når jeg kjeder meg, men ofte klarer jeg å "finne på" noe for å få tankene bort fra mat.
3.	☐	Jeg har for vane å "kjedespise" men det hender at jeg kan foreta meg noe for å få tankene vekk fra å spise.
4.	☐	Jeg har en innbitt vane med å "kjedespise". Ingenting synes å hjelpe meg til å bli kvitt denne vanen.

5.

1.	☐	Som regel er jeg fysisk sulten når jeg spiser noe.
2.	☐	Noen ganger spiser jeg noe helt impulsivt, selv om jeg egentlig ikke er sulten.
3.	☐	Jeg har den uvane å stadig spise mat som egentlig ikke smaker meg for å tilfredsstille en sultfølelse, enda jeg ikke trenger maten rent fysisk.
4.	☐	Selv om jeg ikke er fysisk sulten, får jeg en følelse av sult i munnen, som bare ser ut til å kunne tilfredsstilles hvis jeg spiser noe mat, f.eks. et stykke smørbrød som fyller munnen min. Noen ganger, når jeg spiser mat for å tilfredsstille munnsulten, spytter jeg ut maten for ikke å legge på meg.

6.

1.	☐	Jeg føler ikke skyld eller selvforakt etter at jeg har spist for mye.
2.	☐	Når jeg har spist for mye føler jeg iblant skyld eller selvforakt.
3.	☐	Nesten hele tiden føler jeg skyld eller selvforakt når jeg har spist for mye.

7.

1.	☐	Jeg mister ikke helt kontrollen med spisingen min under en slankekur, selv etter perioder hvor jeg har spist for mye.
2.	☐	Noen ganger når jeg spiser mye ”forbudt” mens jeg er på slankekur, føler jeg at nå har jeg ødelagt alt og så spiser jeg enda mer.
3.	☐	Jeg sier ofte til meg selv, når jeg har spist for mye under en slankekur: ”nå har jeg ødelagt det, så nå kan jeg like gjerne fortsette.” Når det hender, spiser jeg enda mer.
4.	☐	Jeg starter regelmessig på en streng slankekur, men jeg bryter kuren ved å begynne et ”etegilde”. Mitt liv ser ut til å være enten et ”etegilde” eller en sultekur.

8.

1.	☐	Jeg spiser sjelden så mye at jeg føler meg ubehagelig mett etterpå.
2.	☐	Noen ganger (kanskje en gang i måneden) spiser jeg så mye mat, at jeg ender opp med å føle meg ubehagelig overmett.
3.	☐	Jeg har regelmessige perioder hver måned, jeg konsumerer store mengder mat, enten til måltidene eller som mellommåltider.
4.	☐	Jeg spiser så mye mat at jeg stadig føler meg meget uvel etter å ha spist, og noen gang litt kvalm.

9.

1.	☐	Mitt kalori-inntak hverken stiger eller synker meget på en regelmessig basis.
2.	☐	Noen ganger etter at jeg har spist for mye, prøver jeg å redusere kalori-inntaket mitt til nesten ingenting for å kompensere for de ekstra kaloriene, jeg har spist.
3.	☐	Jeg spiser for mye om kvelden. Det virker som om det er naturlig for meg å ikke være sulten om morgenen, men å spise for mye om kvelden.
4.	☐	I mitt voksne liv har jeg hatt ukelange perioder hvor jeg nesten har sultet meg. Disse har etterfulgt perioder, hvor jeg har ”overspist”. Jeg synes å leve et liv enten i matorgier eller i sult.

10.

1.	☐	Jeg er normalt i stand til å slutte å spise, når jeg vil det. Jeg vet når ”nok er nok”.
2.	☐	En gang i blant får jeg en tvingende trang til å spise som jeg ikke synes å beherske.
3.	☐	Jeg får ofte en voldsom trang til å spise som jeg ikke synes å kunne beherske, men andre ganger har jeg min spisetrang under kontroll.
4.	☐	Jeg føler meg ute av stand til å beherske trangen til å spise. Jeg er redd for ikke å kunne stanse og spise frivillig.

11.

1.	☐	Jeg har ingen problemer med å slutte å spise, når jeg føler meg mett.
2.	☐	Jeg kan som regel slutte å spise, når jeg er mett, men iblant spiser jeg for mye, så jeg blir ubehagelig mett.
3.	☐	Jeg har vanskelig for å slutte å spise når jeg først har begynt. Vanligvis føler jeg meg ubehagelig overmett etter et måltid.
4.	☐	Fordi jeg ikke klarer å slutte å spise når jeg vil, må jeg noen ganger tvinge meg til å kaste opp for å lette på følelsen av å ha spist for mye.

12.

1.	☐	Det synes som om jeg spiser akkurat like mye når jeg er sammen med andre (familie, i selskaper) som når jeg er alene.
2.	☐	Noen ganger, når jeg er sammen med andre, spiser jeg ikke så mye som jeg har lyst til, fordi jeg er flau over spisingen min.
3.	☐	Ofte spiser jeg bare litt, når det er andre tilstede, fordi jeg er så veldig flau over spisingen min.
4.	☐	Jeg skammer meg sånn over den overdrevne spisingen min at jeg velger å ”ete” på tider, da jeg vet at ingen ser meg. Jeg føler meg som en ”skap-eter”.

13.

1.	☐	Jeg spiser tre måltider om dagen og tar bare iblant et mellommåltid.
2.	☐	Jeg spiser tre måltider om dagen, men jeg spiser normalt også litt mellom måltidene.
3.	☐	Når jeg småspiser for mye vender jeg meg til å hoppe over ordentlige måltider.
4.	☐	Det er hele perioder, hvor jeg later til å spise uavbrutt, uten noen planlagte måltider.

14.

1.	☐	Jeg tenker ikke mye på å prøve å beherske min uønskede spisetrang.
2.	☐	Jeg føler i det minste noe av tiden, at tankene kretser om å prøve å beherske min spisetrang.
3.	☐	Jeg føler at jeg ofte bruker mye tid på å tenke på hvor mye jeg spiste eller på å prøve å ikke spise mer.
4.	☐	Jeg synes at jeg mesteparten av mitt våkne liv er opptatt med tanke om å spise eller ikke spise. Jeg føler det som jeg stadig kjemper for ikke å spise.

15.

1.	☐	Jeg tenker ikke særlig på mat.
2.	☐	Jeg har sterke anfall av trang til mat, men de er kortvarige.
3.	☐	Det er dager, hvor det virker som om jeg ikke kan tenke på annet enn mat.
4.	☐	Det meste av min tid synes å være opptatt med tanker på mat. Jeg føler at jeg lever for å spise.

16.

1.	☐	Jeg vet normalt om jeg er fysisk sulten eller ikke. Jeg spiser en passende porsjon for å bli mett.
2.	☐	Det hender at jeg er usikker på om jeg er fysisk sulten eller ikke. Da er det vanskelig å vite hvor mye mat jeg skal spise for å bli mett.
3.	☐	Selv om jeg vet hvor mange kalorier jeg bør spise, har jeg ikke noen ide om hva som er ”normal” mengde mat for meg.

Appendix 5 - The Three Factor Eating Questionnaire R-21

Avsnittene nedenfor handler om matvaner og sultfølelse. Les hver påstand eller spørsmål og angi hvilket svar som passer best til deg.
Sett ett kryss i avkrysningsboksen til venstre for det svaret som passer best.

1. **Jeg tar med hensikt små porsjoner for å holde kroppsvekten nede.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

2. **Når jeg føler meg urolig, oppdager jeg ofte at jeg spiser.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

3. **Av og til når jeg begynner å spise, er det akkurat som om jeg ikke klarer å slutte.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

4. **Når jeg føler meg nedstemt, spiser jeg ofte for mye.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

5. **Jeg unngår visse typer mat fordi de er fetende for meg.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

6. **Når jeg er sammen med andre som spiser, får jeg selv ofte lyst på mat og begynner å spise.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

7. **Når jeg er anspent eller "oppgiret", føler jeg ofte trang til å spise.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

8. **Jeg får ofte så lyst på mat at magen føles som et stort hull som ikke kan fylles.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

9. **Jeg har alltid lyst på mat, så det er vanskelig for meg å slutte å spise før jeg har spist opp alt på tallerkenen.**
   - ☐ *Stemmer helt*
   - ☐ *Stemmer ganske bra*
   - ☐ *Stemmer ikke særlig bra*
   - ☐ *Stemmer ikke i det hele tatt*

10. **Når jeg føler meg ensom, trøster jeg meg selv med å spise.**
    - ☐ *Stemmer helt*
    - ☐ *Stemmer ganske bra*
    - ☐ *Stemmer ikke særlig bra*
    - ☐ *Stemmer ikke i det hele tatt*

11. **Jeg holder bevisst igjen ved måltidene for å ikke gå opp i vekt.**
    - ☐ *Stemmer helt*
    - ☐ *Stemmer ganske bra*
    - ☐ *Stemmer ikke særlig bra*
    - ☐ *Stemmer ikke i det hele tatt*

12. **Når jeg kjenner lukten av en biff som stekes eller ser en saftig kjøttbit, er det veldig vanskelig å la være å spise selv om jeg akkurat har avsluttet måltidet.**
    - ☐ *Stemmer helt*
    - ☐ *Stemmer ganske bra*
    - ☐ *Stemmer ikke særlig bra*
    - ☐ *Stemmer ikke i det hele tatt*

13. Jeg har alltid lyst på noe å spise, så jeg kan spise når som helst.

- ☐ *Stemmer helt*
- ☐ *Stemmer ganske bra*
- ☐ *Stemmer ikke særlig bra*
- ☐ *Stemmer ikke i det hele tatt*

14. Hvis jeg kjenner meg ille til mote, forsøker jeg å dempe ubehaget med å spise.

- ☐ *Stemmer helt*
- ☐ *Stemmer ganske bra*
- ☐ *Stemmer ikke særlig bra*
- ☐ *Stemmer ikke i det hele tatt*

15. Når jeg ser noe som ser veldig godt ut, får jeg ofte så lyst på det at jeg må det spise med en gang.

- ☐ *Stemmer helt*
- ☐ *Stemmer ganske bra*
- ☐ *Stemmer ikke særlig bra*
- ☐ *Stemmer ikke i det hele tatt*

16. Når jeg føler meg dyster til sinns eller lei meg, vil jeg ha noe å spise.

- ☐ *Stemmer helt*
- ☐ *Stemmer ganske bra*
- ☐ *Stemmer ikke særlig bra*
- ☐ *Stemmer ikke i det hele tatt*

17. Hvor ofte unngår du å ha fristende mat tilgjengelig?

- ☐ *Nesten aldri*
- ☐ *Sjelden*
- ☐ *Ofte*
- ☐ *Nesten alltid*

18. Hvor sannsynlig er det at du bevisst spiser mindre enn det du vil ha?

- ☐ *Usannsynlig*
- ☐ *Ikke særlig sannsynlig*
- ☐ *Ganske sannsynlig*
- ☐ *Veldig sannsynlig*

19. Fortsetter du å spise selv om du ikke er sulten lenger?

- ☐ *Aldri*
- ☐ *Sjelden*
- ☐ *Iblant*
- ☐ *Minst en gang i uken*

20. Hvor ofte har du lyst på mat?

- ☐ *Bare til måltidene*
- ☐ *Iblant mellom måltidene*
- ☐ *Ofte mellom måltidene*
- ☐ *Nesten alltid*

21. På en skala fra 1 til 8, der 1 står for ingen begrensning (spiser hva jeg vil, når jeg vil) og 8 står for streng begrensning (begrenser alltid matinntaket, gir aldri etter), hvor på skalaen befinner du deg?

Sett en ring rundt det tallet som passer best for deg.

1	2	3	4	5	6	7	8
Spiser hva jeg vil, når jeg vil							*Begrenser alltid matinntaket, gir aldri etter*

Appendix 6 – Mean scores, changes, and differences between subgroups in eating behavior during treatment

		1 BN-CBT	2 BN-PED-t	3 BED-CBT	4 BED-PED-t	0 Control	p-values for group differences
n		29	34	14	20	11	
				Mean score (SD)			
BES							
	Pre	41.8 (5.9)	43.0 (6.9)	44.2 (5.8)	44.2 (6.5)	41.9 (8.4)	
	Post	32.8 (7.6)*	32.4 (8.7)*	35.4 (6.9)	34.2 (7.7)	38.9 (10.4)	0-1: 0.04; 0-2: 0.02.
	Change	-9.0 (6.0)*	-10.6 (9.4)*	-8.9 (4.9)*	-10.0 (7.1)*	-3.0 (7.2)	1-0: 0.03; 2-0: <0.01; 3-0: 0.05; 4-0: 0.02.
CR							
	Pre	18.1 (2.6)*	16.8 (3.3)	16.8 (3.3)	16.3 (3.1)	17.3 (2.8)	1-4: 0.04.
	Post	17.2 (3.4)*	15.3 (3.2)	16.7 (3.3)	15.1 (2.7)	16.0 (4.0)	1-2: 0.02; 1-4: 0.03.
	Change	-1.3 (3.5)	-1.6 (3.2)	-0.1 (2.4)	-1.2 (3.0)	-1.3 (3.5)	
UE							
	Pre	26.4 (5.3)	27.6 (4.5)	29.0 (3.5)	28.8 (3.6)	28.4 (6.2)	
	Post	22.1 (4.8)*	21.3 (5.8)*	24.8 (4.0)	23.7 (6.0)	26.5 (6.0)	0-1: 0.02; 0-2: <0.01; 3-2: 0.04.
	Change	-4.4 (5.4)	-6.3 (5.2)*	-4.2 (2.8)	-5.2 (4.9)	-1.8 (4.9)	2-0: 0.01.
EE							
	Pre	17.6 (4.5)	18.9 (3.3)	19.7 (3.2)	18.2 (4.4)	16.6 (4.5)	
	Post	14.9 (3.8)	14.4 (4.2)	17.7 (2.6)*	15.8 (4.6)	17.0 (4.2)	3-1: 0.03; 3-2: 0.01.
	Change	-2.7 (3.9)*	-4.5 (4.1)*	-2.0 (3.2)	-2.4 (3.9)	0.4 (3.7)	1-0: 0.03; 2-0: <0.01; 2-3: 0.04.

BN: Bulimia Nervosa; BED: Binge Eating Disorder; CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; BES: Binge Eating Scale; CR: Cognitive restraint; UE: Uncontrolled eating; EE: Emotional eating; SD: Standard Deviation; *Significant difference between groups.

Appendix 7 – Detailed overview of change in nutrient intake and food selection during treatment

	CBT n = 54			PED-t n = 62		
	Mean (SD)					
	Baseline	Post treatment	Change	Baseline	Post treatment	Change
Energy						
Kilojoule, kJ	8898.5 (3164.4)	8823.5 (5221.8)	-75.0 (5457.5)	10926.6 (8305.0)	9991.9 (5993.5)	-934.7 (6640.9)
Kilocalories, kcal	2122.7 (754.9)	2104.6 (1246.2)	-18.1 (1301.9)	2608.0 (1979.7)	2397.6 (1452.8)	-210.4 (1587.4)
Energy-yielding nutrients (g)						
Fat	86.1 (39.9)	87.5 (60.3)	1.4 (64.6)	110.7 (87.6)	95.6 (61.2)	-15.1 (76.6)
Saturated fat	31.7 (19.7)	28.9 (25.7)	-2.8 (31.3)	40.8 (38.4)	34.5 (24.5)	-6.3 (34.5)
Transunsaturated fat	0.6 (0.6)	0.6 (0.8)	-0.1 (0.9)	0.7 (1.0)	0.7 (1.0)	-0.1 (0.9)
Monounsaturated fat	28.5 (17.3)	29.3 (22.3)	0.8 (23.5)	38.5 (34.9)	31.3 (19.4)	-7.3 (32.0)
Polyunsaturated fat	12.1 (7.0)	14.0 (9.4)	1.7 (9.5)	15.9 (12.6)	15.5 (12.5)	-0.4 (13.8)
Omega-3	2.3 (1.7)	3.0 (2.5)	0.7 (2.5)	3.4 (4.5)	3.4 (3.6)	-0.1 (4.2)
Omega-6	9.3 (5.9)	10.1 (3.6)	0.8 (7.7)	11.3 (9.0)	11.3 (8.3)	0.0 (9.0)
Cholesterol	274.3 (232.8)	259.2 (194.9)	-15.1 (280.7)	386.3 (631.9)	377.3 (309.4)	-9.0 (500.0)
Carbohydrate	247.1 (107.5)	244.7 (162.2)	-2.4 (174.1)	295.4 (237.9)	277.0 (182.7)	-18.4 (199.1)
Starch	98.7 (59.3)	101.5 (81.5)	2.8 (87.9)	113.5 (110.0)	115.7 (65.5)	2.2 (98.9)
Mono- and disaccharides	99.0 (56.3)	97.1 (76.5)	-1.8 (94.3)	123.3 (127.1)	109.9 (103.0)	-13.4 (109.4)
Added sugar	43.0 (45.5)	40.8 (50.3)	-2.2 (71.0)	56.4 (68.5)	45.6 (74.7)	-10.7 (83.6)
Dietary fiber	27.8 (14.4)	28.3 (17.5)	0.5 (12.8)	28.8 (21.6)	28.9 (19.1)	0.1 (17.0)
Protein	97.8 (35.5)	92.3 (37.8)	-5.5 (44.6)	114.2 (92.9)	116.2 (72.5)	2.0 (72.3)
Alcohol	1.2 (7.8)	1.1 (2.5)	-0.6 (8.3)	0.8 (3.4)	1.7 (7.5)	0.9 (6.3)

Vitamins, minerals and trace elements						
Salt	6.0 (3.9)	5.9 (3.8)	-0.1 (5.5)	7.0 (6.2)	7.4 (4.2)	0.4 (6.6)
Sodium, mg	2467.5 (1590.5)	2442.8 (1583.3)	-24.7 (2191.5)	2854.6 (2533.5)	3046.1 (1811.4)	191.5 (2702.7)
Vitamin A, RAE	814.6 (981.4)	838.2 (940.2)	23.6 (1367.8)	879.9 (865.3)	938.8 (858.5)	58.9 (1008.4)
Retinol, ug	515.0 (968.5)	602.9 (924.7)	87.9 (1337.6)	561.1 (735.8)	679.2 (815.9)	118.1 (886.5)
Betakaroten, ug	3029.1 (4243.1)	2394.0 (2839.3)	-635.1 (4136.1)	3129.1 (3575.3)	2556.6 (2842.5)	-572.5 (4147.3)
Vitamin D, ug	6.7 (9.0)	7.5 (7.5)	0.8 (11.4)	10.4 (12.9)	9.9 (10.2)	-0.5 (12.9)
Vitamin E, u-TE	15.0 (10.6)	14.6 (9.7)	-0.4 (12.2)	18.3 (16.0)	17.4 (12.3)	-0.9 (12.2)
Thiamin (B1), mg	1.7 (1.2)	1.5 (0.9)	-0.2 (1.3)	2.0 (1.7)	2.0 (1.4)	0.1 (1.6)
Riboflavin (B2), mg	1.9 (1.3)	1.7 (0.9)	-0.2 (1.5)	2.7 (3.3)	2.3 (2.1)	-0.4 (2.6)
Niacin (B3), mg	20.7 (13.5)	18.6 (8.7)	-2.1 (16.1)	21.9 (18.3)	26.4 (25.2)	4.5 (26.1)
Pyridoxine (B6), mg	1.9 (1.1)	1.8 (0.8)	-0.2 (1.2)	2.0 (1.6)	2.3 (1.7)	0.3 (1.8)
Folate, ug	318.2 (190.8)	301.2 (175.8)	-17.0 (228.8)	283.2 (232.3)	313.2 (163.9)	30.0 (200.2)
Vitamin B12, mg	5.5 (3.3)	6.1 (3.7)	0.6 (5.1)	8.2 (8.8)	8.4 (7.5)	0.2 (6.5)
Vitamin C, mg	134.3 (126.5)	127.6 (154.2)	-6.7 (196.0)	90.8 (90.8)	131.1 (89.7)	40.2 (114.1)
Calcium, mg	939.7 (536.5)	896.8 (538.2)	-42.9 (615.2)	1264.3 (1436.4)	1149.1 (712.9)	-115.2 (1226.0)
Iron, mg	10.6 (7.5)	10.0 (6.1)	-0.7 (9.4)	11.3 (9.2)	12.1 (7.7)	0.8 (8.5)
Potassium, mg	3453.4 (1512.7)	3320.2 (1616.8)	-133.2 (1594.8)	4227.8 (3942.1)	3770.0 (2041.9)	-457.8 (3283.4)
Magnesium, mg	350.3 (145.3)	328.9 (172.8)	-21.3 (165.0)	386.2 (285.1)	394.4 (229.3)	8.1 (234,6)
Zinc, mg	10.6 (7.3)	9.7 (5.4)	-0.9 (9.6)	12.5 (11.6)	12.6 (7.2)	0.1 (9.9)
Selenium, ug	47.5 (39.2)	45.0 (24.4)	-2.5 (44.2)	60.2 (57.1)	68.7 (68.2)	8.5 (57.6)
Copper, mg	1.4 (0.9)	1.3 (0.7)	-0.1 (1.0)	1.6 (1.3)	1.5 (1.3)	-0.1 (1.3)
Phosphorus, mg	1603.8 (628.0)	1549.3 (788.1)	-54.5 (850.1)	1993.3 (1834.9)	1942.1 (1107.3)	-51.3 (1471.9)
Iodine, ug	91.5 (115.9)	84.1 (65.3)	-7.5 (115.4)	132.0 (211.1)	103.6 (81.5)	-28.3 (179.8)

CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; SD: Standard deviation.

Appendix 8 – Changes in nutrient intake and food selection for treatment subgroups

	1 BN-CBT	2 BN-PED-t	3 BED-CBT	4 BED-PED-t	p-values for group differences
n	33	36	16	21	
			Mean (SD)		
Kilocalories					
Energy					
Pre	2085.9 (671.9)*	3048.9 (2420.5)*	2245.2 (955.7)*	2010.8 (897.9)	1-4: <0.01; 2-3: <0.01; 2-4: <0.01; 3-1: <0.01.
Post	2284.6 (1413.1)	2561.8 (1768.3)	1853.2 (923.7)	2242.6 (896.8)	
Difference	198.7 (1470.7)	-487.1 (1921.6)	-392.0 (866.2)	231.8 (899.2)	
Grams					
Fat					
Pre	86.4 (39.5)	128.7 (104.3)*	93.5 (42.6)	86.8 (52.6)	2-1: 0.02; 2-4: 0.03.
Post	93.9 (69.2)	101.0 (73.4)	80.5 (43.8)	88.2 (43.3)	
Difference	7.5 (70.7)*	-27.8 (90.0)	-13.0 (53.5)	1.3 (52.9)	1-2: 0.05.
Carbohydrate					
Pre	235.7 (88.4)	344.4 (293.1)*	261.6 (139.3)	228.3 (102.9)	2-1: 0.02; 2-4: 0.03.
Post	275.3 (182.9)	295.8 (220.6)	200.5 (118.0)	267.4 (115.1)	
Difference	39.6 (193.2)	-48.6 (241.8)	-61.1 (109.7)#	39.1 (112.9)	
Dietary fiber					
Pre	30.2 (14.3)	34.0 (26.6)*	24.3 (15.2)	21.5 (8.0)	2-4: 0.02.
Post	32.6 (18.3)*	30.3 (23.7)	20.7(14.6)	28.8 (9.5)	1-3: 0.04.
Difference	2.4 (13.9)	-3.6 (20.2)	-3.6 (8.9)	7.3 (9.0)*#	4-2: <0.01; 4-3: 0.03.
Protein					
Pre	97.7 (35.4)	134.6 (114.4)*	100.0 (39.7)	86.6 (39.8)	2-1: 0.04. 2-4: 0.02.
Post	96.2 (39.2)	127.4 (89.6)*	86.9 (35.2)	104.9 (35.2)	2-1: 0.03. 2-3: 0.03.
Difference	-1.5 (50.6)	-7.1 (84.8)	-13.1 (29.7)	18.3 (51.4)	

Fruits, berries and vegetables					
Pre	474.1 (264.0)*	422.3 (356.8)	346.5 (354.9)	281.0 (210.0)	1-4: 0.03.
Post	209.1 (281.4)	147.8 (89.6)	294.6 (346.1)	283.7 (228.2)	
Difference	-265.0 (375.1)[#]	-274.5 (403.7)[#]	-52.0 (347.8)*	2.7 (290.5)*	1-4: 0.01; 2-3: 0.05; 2-4: <0.01.
Carbohydrate staple foods					
Pre	208.7 (125.7)	255.0 (237.3)	264.8 (176.5)	273.2 (192.3)	
Post	239.2 (171.5)	297.4 (156.8)	226.6 (136.2)	294.3 (96.8)	
Difference	30.5 (203.0)	42.4 (191.2)	-38.2 (219.4)	21.1 (196.2)	
Whole grain sources					
Pre	102.3 (99.3)	117.7 (147.7)	76.1 (97.6)	67.6 (65.9)	
Post	122.8 (103.9)	152.6 (153.2)	92.2 (83.3)	165.6 (103.6)	
Difference	20.5 (143.4)	34.9 (143.0)	16.1 (112.2)	98.0 (136.3)*[#]	4-1: 0.05.
Milk and dairy products					
Pre	354.5 (307.5)	496.1 (688.7)	292.3 (296.5)	388.4 (534.3)	
Post	419.5 (266.2)	401.6 (384.6)	272.4 (249.7)	441.0 (254.0)	
Difference	65.1 (289.0)	-94.5 (529.7)	-19.9 (257.7)	52.6 (603.5)	
Fat meat					
Pre	62.1 (91.9)	54.7 (117.3)	75.8 (129.2)	52.9 (124.6)	
Post	34.2 (75.9)	67.9 (107.6)	35.8 (58.8)	49.9 (88.0)	
Difference	-27.9 (101.7)	13.2 (146.2)	-39.9 (111.2)	-3.0 (163.6)	

BN: Bulimia Nervosa; BED: Binge Eating Disorder; CBT: Cognitive Behavior Therapy; PED-t: Physical Exercise and Dietary Therapy; SD: Standard Deviation; [#]Significant change from baseline; *Significant difference between treatment subgroups.