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Dysfunctional eating behaviours and personality traits in severely obese patients undergoing bariatric surgery

The PSYMO study – a randomized controlled trial

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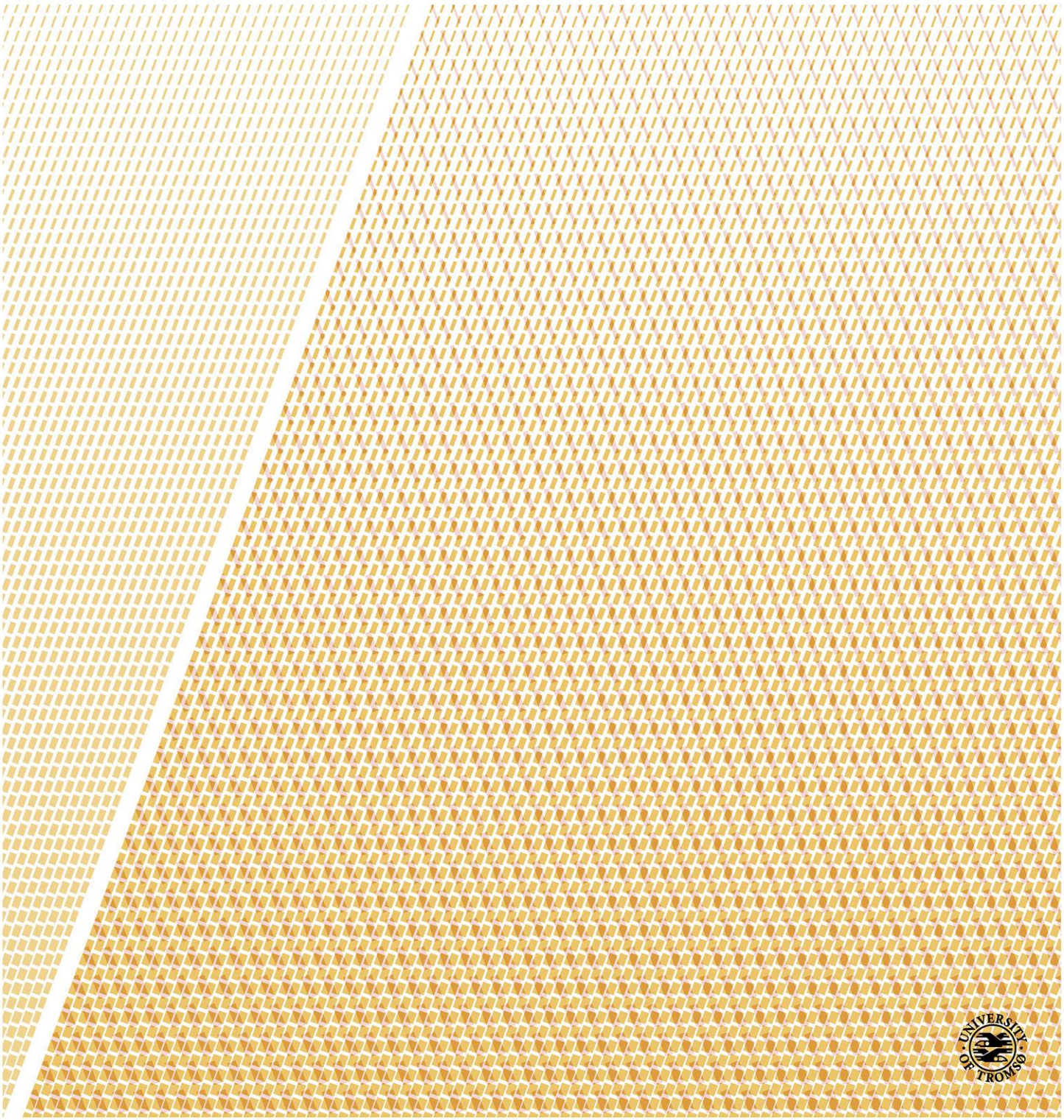

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Preface

The patients who have undergone bariatric surgery at the Morbid Obesity Centre, Vestfold Hospital Trust have given birth to the PSYMO-study. The research idea was born after a couple of years of working with post-surgical follow-up groups (2 years after surgery). In almost all groups, there were patients complaining that "their heads had not yet understood that their stomachs were much smaller" and that they still craved energy-dense food that they could not eat anymore. Despite comprehensive preparations before surgery, increasingly more patients in those groups claimed that they missed their dumping episodes that had the force to "punish" them every time they ate sugar and fatty food. Furthermore, some patients could tell that just a few weeks after surgery, they found out how much chocolate they could eat at a time without activating any dumping symptoms. My clinical experience was that too many of those patients had either re-developed the same dysfunctional eating behaviours as before the surgery, or developed new maladaptive eating patterns with grazing and emotional eating. For some patients, it was as if the body signals of dumping, which appear to work as *Skinnerian punishment mechanisms*, were not sufficient to change their eating behaviours permanently. In this light, bariatric surgery is not only a topic for surgeons and internists.

Therefore, this project hoped to expand knowledge and understanding of how cognitive behavioural therapy may improve dysfunctional eating behaviours and affective symptoms. Additionally, we aimed to give a deeper elaboration of important psychological features and mechanisms that underlie eating behaviours and how these may contribute in the understanding of morbid obesity.

Acknowledgements

This thesis is the result of the randomized controlled trial PSYMO carried out at the Morbid Obesity centre, Vestfold Hospital Trust, Tønsberg in the period 2011-2014. This work has been supported by an unrestricted educational grant from The Norwegian Health authorities, South-East, which funded my position as a PhD student.

My deepest gratitude goes to my former leader, Pediatrician Martin Handeland from whom I have learned a lot and who let me spend time at work to study and start writing my protocol. Thank you so much, Martin!

Morbid obesity is surely a *disease* that you can measure in pounds and centimetres, but nevertheless, in my clinical research and practice, I have recognized that the most important issue for patients may be the *illness* of being morbidly obese and their perceptions of having this disease. Therefore, I want to express my deepest thankfulness to all the PSYMO participants who have contributed with their willingness, positive attitudes and hours and hours of participation so that this research could be done.

As a PhD fellow, one does not research alone! Even though I worked as a health behaviour therapist with no external funding, professor and Head of the Morbid Obesity centre and my co-supervisor, Jøran Hjelmesæth has from the very beginning been extremely encouraging and supportive in designing a new study, and helped me out writing a research protocol so that we could apply for external funding. He deserves the greatest gratitude for the invaluable contributions he has made from the early beginning until the completion of this thesis!

In the search for an experienced and excellent main supervisor in psychology, I used Google! I found that professor Jan Rosenvinge at the The Arctic University of Norway, University of Tromsø, had done a lot of interesting work in the intersection of eating disorders, eating pathology and morbid obesity. He was (and still is!) a very busy man, but he was extremely positive and helpful from the very beginning. He also connected his colleague, my co-

supervisor professor Oddgeir Friborg, to this study. Please accept my heartfelt gratitude and thanks for all your helpfulness, support and the extremely important contributions and honest feedback in this study.

For a PhD fellow doing clinical research and especially a randomized controlled trial, one is completely dependent on a very well-organised clinic with well-planned logistic. I feel a special gratitude to Astrid Hillestad for having an excellent over-view of the patients admitted for bariatric surgery, Randi S. Lund for very interesting discussions of health related quality of life in women with obesity and Mariann Mathisen (Head of the Hospital Library) to set up systematic literature searches and alerts! ***Especially***, I am filled with profound gratitude to my former research assistant Åshild Skulstad-Hansen for organising patients' logistics with impressive accuracy at the one year follow-up measurements. I also extend gratitude to both former and current colleagues at the Morbid Obesity Centre for their contributions and support. Especially

In keeping up the good spirit, my special thanks go to my colleagues "The very best Line Kristin Johnson", Jan Magnus Fredheim and Njord Nordstrand – for your crazy humour and comments! My deep gratitude also goes to my colleague Dag Hofsø ("My Martin") who always showed exemplary organisational skills and offered his sincere willingness to help out making nice figures and tables for my oral presentation in Liverpool.

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To all my good friends, but first of all the group of girlfriends who I have had since childhood ("Flyfillene"): Ellen, Inger, Liv-Grethe, Rikke, Tone and Tove Gro (alphabetically sited). You have all been extremely curious, encouraging and responsive about my work. You are

simply the best! I also want to express my gratefulness to my dear friends in “the Beaujolais Nouveau group” who have given me so much joy and laughter.

My thoughts also go to my father, who died in 2001, but who gave me so much inspiration and encouragement in education and home-work when I grew up.

I am also extremely thankful for all the attention and interest in my work from my mother, my lovely daughters-in-law Eline and Kamma, my American family: “cousoeur” Lisa, her husband Jay and “my American son” Carl. Thank you, Lisa for patiently practicing with me over Skype when I was going to have my oral presentation at the European Congress of Obesity research (ECO) in 2013!

To you, my dearest Geir, my best friend, coach, mentor and husband: You are the very best!

And finally, my everlasting gratitude goes to my two sons, Sindre and Erlend. Thank you so much for being patient, helpful, funny and encouraging and for expressing that you are proud of Mum!

(I also do hope that my family will forgive me forgetting that I invited everyone over for family-dinner and that I was still working with this thesis when the dinner-guests arrived - without any dinner in the house. A PhD-project is overwhelming! It steals all the cognitive capacity that you may have.)

February, 2015

Hege Gade

I am now doing three things: concerts, conducting, and teaching, and they each support each other. I learn to see things from different perspectives and listen with different ears. The most important thing that you need to do is really listen.
(Itzhak Perlman)

Abbreviations

BE	Binge eating (symptoms)
BED	Binge eating disorder
BMI	Body Mass Index
BS	Bariatric surgery
CR	Cognitive restraint
DE	Dysfunctional eating behaviours
ED(s)	Eating disorders
EE	Emotional eating
EB(s)	Eating behaviour(s)
MO	Morbid obesity/Morbid obese
RYGB	Roux-en-Y Gastric Bypass
PSYMO	**Psy**chology in **m**orbid **o**besity
SG	Sleeve gastrectomy
UE	Uncontrolled eating

List of papers

Paper I: Gade H. Rosenvinge JH, Hjelmesæth J. Friborg O. Psychological correlates to dysfunctional eating patterns among morbidly obese patients accepted for bariatric surgery. *Obesity Facts* 2014

Paper II: Gade H, Hjelmesæth J, Rosenvinge JH, Friborg O. Effectiveness of a cognitive behavioral therapy for dysfunctional eating among patients admitted for bariatric Surgery: A randomized controlled trial. *Journal of Obesity* 2014

Paper III: Gade H, Friborg O, Rosenvinge JH, Småstuen MC, Hjelmesæth J. Impact of a pre-surgical cognitive behavioural therapy (CBT) on dysfunctional eating behaviours, affective symptoms and body weight one year after bariatric surgery. A randomised controlled trial", resubmitted 2015 to *Obesity Surgery.*

1 Introduction

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1.1 Classification and prevalence of obesity

The World Health Organization defines obesity as a chronic disease and has determined cut-off points for overweight and obesity (1). Using the Body Mass Index (BMI) (weight in kilograms divided by the square of height in meters (kg/m^2)) values between 25 and 29.9 define overweight, whilst a BMI score ≥ 30 indicates obesity. Being morbidly obese indicates a BMI ≥ 40 or a BMI ≥ 35 with at least one comorbid condition (i.e. hypertension, Type 2 diabetes, or sleep apnea).

According to the Norwegian epidemiological study HUNT (2), which includes 90.000 participants, the obesity epidemic in Norway is neither slowing down nor halting. Findings from this study indicate that there has been an increase in BMI in all weight groups, and around 4-5 % of the Norwegian population are estimated to have morbid obesity (MO). Moreover, the increase in BMI has been largest in the youngest age groups. These findings suggest that there is a strong demand for efficacious treatment strategies and intervention programmes that can help people to lose weight.

1.2 Eating and eating behaviours

Obesity has become a major cause of morbidity and mortality, and the obesity epidemic seems more related to an increase in energy intake than a more sedentary life style. Data from the American Food consumption surveys from 1977-2006 (3, 4) indicated that the total energy intake has increased significantly during this period, and so does the prevalence of obesity. The consumption of more energy-dense food, larger portion sizes and more frequent "snacking" and drinking have been characterised as important eating behaviours (EB)

explaining the significant increase in the prevalence of obesity (4, 5). Hence, positive energy balance is considered the most important aetiology factor for the development of obesity (6).

1.2.1 Hunger and dieting

The regulation of appetite and body-weight is a complicated process including neural systems and hunger- and appetite hormones (7). From neurophysiological and endocrinological perspectives, eating is a response to hunger- and appetite hormones like for instance ghrelin, a gut-hormone which stimulates the appetite and the growth hormone release (8). The seminal experiment *The Biology of Human Starvation* (9) presents insight in how starvation and hunger influence affective symptoms and mood, as well as a cognitive shift consisting of an intense preoccupations with thoughts of food. The participants in this study became depressed, irritable, their libido changed. However, hunger as an internal cue has a comprehensive impact on mood, cognition and EBs.

These early findings are of special interest considering how *dieting* may influence EB. The main change of EB in dieting is a cognitive restraint of eating, meaning that the intake of energy is below the energy expenditure. From an evolutionary perspective, dieting may be considered as an *abnormal* or *dysfunctional* eating behaviour (DE), as it in the long run may be life-threatening to consume fewer calories than needed. As an EB, dieting, or food restriction as shown in the starvation experiment (9), may influence both cognition and emotions, and in some people, the "dieting behaviour" itself also leads to overeating (10). From dieting to overeating - one DE may trigger another. The dieting may elicit shifts in cognition and a feeling of losing control when one is not completely compliant to the diet, and these cognitions and emotional states may trigger overeating. Furthermore, in persons who have had a massive weight loss, the overeating behaviour may be considered as a life-saving strategy. Moreover, in some studies, dieting behaviours have been found to predict increased risk for weight gain (11-13). According to the restraint theory (14), the self-induced restraint

of eating and overeating co-occur, and that it is the dieting that causes overeating when a break in a diet occurs. Ruining the diet temporarily seems to have a cognitive influence on the eating behaviour. The overeating may be interpreted as a physiological process, but the overeating seems to be strongly mediated by cognition (15).

1.2.2 Eating behaviours in morbidly obese

DE can be defined as exerting rigid control, or loss of control over eating, or eating for emotional reasons rather than hunger or appetite. This can aggravate to conditions close to or satisfying DSM-5-criteria for Other Specified Feeding or Eating Disorder (OSFED) or a binge eating disorder (BED).The development of obesity may be seen as a consequence of DE as the behaviour is associated with overconsumption of energy dense food (16-20). DE is maintained partly by the reinforcing effect of alleviating negative moods or experiences of stress and activation (21-23). DE **has, thus been operationalized as** *emotional eating* (EE), *uncontrolled eating* (UE) and *cognitive restraint* of eating (CR) (24).

EE serves to regulate negative mood states like feeling lonely, anxious or depressed. On the other hand, people with UE tend to lose control over their eating behaviour when feeling hungry while people with CR tend to overly restrict their food intake in order to control weight, body shape or body composition. All three patterns have all been identified among MO-patients (25). Recently, Gelinas et al (26) presented findings indicating that morbidly obese patients admitted for bariatric surgery (BS) perceive eating in response to negative affect, overeating and food desirability or eating in response to positive affect/social cues as three main reasons for their weight gain. The patients' perceptions concur with findings indicating that EE and UE are associated with higher body weight (27). Furthermore, in patients undergoing BS, less improvements in UE and EE postoperatively are related to a higher BMI (28).

Among patients with morbid obesity (MO) the DSM-IV definition of binge eating disorder (BED)vary between 6 – 49 % and binge eating (BE) symptoms (of any kind) between 6- 64 % (29-35). Moreover, 10-25 % of obese patients considered for or *completing* bariatric surgery (BS) have DE (33, 36-39). A recent study (40) comparing ED symptoms in normal, overweight and obese ED-patients, indicates that the obese patients display even more severe ED symptoms (poorer body-image and feelings of lacking control over food intake) than normal weight patients. Moreover, findings from the Look AHEAD-study (41) also demonstrate that participants with DE are significantly heavier than those without.

For patients eligible for bariatric surgery (BS), DE has been reported both prior (35, 42) to and after BS (21, 32, 43, 44), and DE may also impair sustained weight loss post-surgery (22, 32, 34, 45, 46). For most of those patients experiencing weight regain postoperatively, the major regain tend to start after one year post-surgically.

Thus, DE seems to be strongly related to obesity in general and weight regain in patients undergoing BS in particular, but less is known about the underlying mechanisms of DE.

1.3 What affects dysfunctional eating behaviours in morbidly obese?

1.3.1 The impact of personality

Personality is a psychological construct of large impact as it permeates most aspects of humans' cognitive and emotional life and behaviour (47). According to Ozer et al (48), the construct of personality refers to the stable personal characteristics that are revealed in a particular pattern of behaviour, and these characteristics are able to predict individual differences in behaviour. Additionally, personality relates to factors causing disease (49), and as such also life outcomes for the individual. The mechanisms underpinning DE are poorly understood, but certain personality traits may be good candidates. There are individual

differences in how different characteristics are distributed. (47). In a representation of the Five Factor Model of personality, five dimensions represent basic tendencies of thoughts, feelings and behaviours. Those basic tendencies are **Neuroticism** (anxiety, angry hostility, depression, self-consciousness, impulsiveness and vulnerability), **Extraversion** (warmth, gregariousness, assertiveness, activity, excitement seeking, positive emotions), **Openness for experience** (fantasy, aesthetic feelings, actions, ideas and values), **Agreeableness** (trust, straightforwardness, altruism, compliance, modesty and tender-mindedness) and **Conscientiousness** (competence, order, dutifulness, achievement striving, self-discipline and deliberation). These traits may be seen as individual characteristics accounting for particular patterns of behaviour across various contexts. People with higher scores on neuroticism have more pronounced emotional dysfunction with more negative feelings which may act as drivers in adopting dysfunctional behaviours. Particularly, higher levels of neuroticism are related to an impaired ability to cope with stress and stress-related disorders. Eating patterns may then be negatively influenced by neuroticism. In addition to the genetic factors of being predisposed to weight gain, body weight reflects our EB. DE observed among morbidly obese patients seem related with personality (29, 36, 50-52), and in particular, neuroticism (52-55). Neuroticism may imply a tendency to be sensitive towards excessive availability of food (52), and in the development of MO this trait may affect a disability to adjust EB. Recent findings indicate that the facet impulsiveness may be an important risk factor for the tendency to engage in addictive food-intake (56). Thus, neuroticism may be seen as a psychological risk factor in the development of MO. According to the findings of Canetti (57), EE mediated the relation between neuroticism and body weight, meaning that there may be a causal pathway via emotionally regulated eating explaining the mechanism between neuroticism and BMI. Personality traits may have a basic influence on how the individual adapt to the environment, and the characteristic adaptations may include personal strivings and attitudes.

As personality traits are quite stable over time (58), EB are persistently influenced by these traits. Hence, personality traits, like neuroticism, may be clinically important to address in addition to mood disorders as such traits are part of the psychological makeup of an individual that always exert a potential negative effect. In the study by Terracciano et al (59), they found that personality traits were associated with both underweight and overweight. Participants with overweight showed significantly lower scores on conscientiousness and significantly higher impulsiveness than those with normal weight. Additionally, those who scored in the higher levels of impulsiveness were in average 4 kg heavier. High levels of neuroticism imply a tendency to give in to sudden temptation to eat, and may be a strong predictor of the development of obesity and DE.

It is not known how gender, mood and affective symptoms, respectively may relate to personality traits and EB. Though, in female pre-bariatric obese patients, two broad personality subtypes have been observed (60). A more well-adjusted subtype shows a profile of high emotional stability, extraversion, conscientiousness and agreeableness, while a less well-adjusted subtype may be characterized by the opposite. The latter subtype also tends to display significantly more binge eating (BE) and DE in order to regulate emotions.

Concerning successful weigh reduction maintenance, *calorie restriction* has been identified as the strongest predictor of a sustained weight loss (61), and in a large Australian sample, conscientiousness was found to be inversely related to obesity (62). People who succeed in maintaining weight loss and calorie restriction tend to score significantly lower on neuroticism and hostility and higher on conscientiousness (63, 64).

1.3.2 Gender and personality traits

Women constitute the majority of morbidly obese persons including those seeking BS (65), and DE is also more prevalent among women in the general population (66). Moreover, EE seems to be more frequent among morbidly obese women (22). In the obesity literature small

to negligible gender differences have been reported in how EB and personality traits may be related (55), and inconsistent findings may be due to small sample sizes and failures to include BS-patients. Findings indicate that individual differences in personality traits and EB may account for the fact that some manage long-term calorie restriction and some do not (63). Thus, those who manage such long-term calorie restriction behaviours display lower neuroticism and low responsiveness to external cues. The fact that EE is more prominent in female patients admitted for BS, and that EE may be a "high risk" EB in both the development of obesity and weight regain after surgery, expanding knowledge about gender differences is important. The previously described associations between DE and gender in patients admitted for BS are not sufficient for the understanding of this relationship. Therefore, underlying mechanisms such as personality traits, symptoms of anxiety and depression need to be explored.

1.3.3 The role of poor mental health

Substantial comorbidity of mental disorders has been reported among MO patients (36, 39, 67-73), and this may be attenuated for MO patients admitted for BS compared with MO-patients seeking conservative obesity treatment (30). Studies indicate that in patients admitted for BS, affective and mood disorders might be more prevalent in female patients (72, 74). A recent review (72), however, shows widely disparate prevalence figures, i.e. 36-72% for lifetime, and 20-55% for a current DSM-IV symptom diagnosis of notably major depressive disorder, general anxiety disorder, BED and dysthymic disorder. This diversity of prevalence rates may to some extent be accounted for by sample characteristics and the use of different assessment methods (71).

Mood disorders, such as depression, may disrupt self-regulatory sleep and EB (75), and depression in obese patients may therefore affect the appetite and craving for energy-dense

food (76). Additionally, depression is associated with higher BMI in the population of obese (30).

As indicated in *Figure 1,* mood in general may affect self-rewarding mechanisms of food (77).

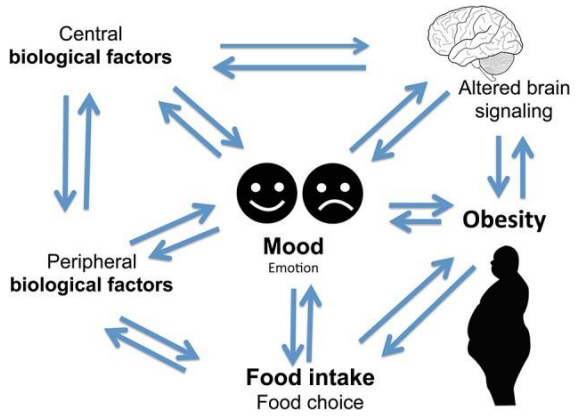

Figure 1. Complex two-way relationship linking food intake, mood and obesity
Figure by Singh, M. in *Mood, food and obesity* (78)

Recent findings show that the hunger hormone ghrelin is involved in stress and reward-oriented behaviours and regulates anxiety (79). Anxiety may in turn affect stress-related eating and the motivation to eat (80). Anxiety may further prompt worries about future consequences of non-sustainable changes and a reluctance to implement behavioural changes. As discussed by others (81, 82), the role of anxiety for eating behaviour may be different than that of depression. In the period following BS, symptoms of depression usually show a larger reduction than symptoms of anxiety (83, 84).

For MO patients it may be even more complicated to regulate eating: Neurophysiology studies (85, 86) indicate that glucocorticoids, like for instance the stress hormone cortisol, stimulate appetite and may have a major effect on food intake and fat storage (87-89). We may thus expect that the higher levels of anxiety and depression symptoms, which may imply higher cortisol-levels over time, also may have an impact on eating and the development of DE. To add, there are findings demonstrating that a stress-induced EB may have an impact on

food choices (80, 89), indicating that the nutrients that are "craved" tend to be energy-dense, often consisting of sugar and fat.

1.3.4 Environmental factors

Developmentally, EB can be viewed as patterns or habits of eating which have been acquired from the observing, imitating and internalizing of care-givers' food choices and feeding behaviours (90). Hence, the development of DE, as well as the child's weight status, seem closely related to parents' restrictive feeding behaviour (91), and the family environment. The availability of food has an impact on EB, and for a variety of reasons, we are tempted to eat when we are exposed to external cues like seeing or smelling food, either in commercial publicities or in real life (92, 93).

EB may as well be associated with positive emotions and cues (26) leading for instance, to more eating in familiar social settings (94).

1.3.5 Interactive processes

Environmental cues may also in turn influence the individuals' adaptations. One example may serve to illustrate a dynamic process: A patient with a high neuroticism – score (traits) has developed low self-esteem and a feeling of being a person who is unable to control needs or impulses (adaptations). In an obesogenic environment, the patient is constantly surrounded by highly available energy-dense food which smells and looks very tasty (environmental cues).

In an obesogenic environment, the high neuroticism-score may represent a *vulnerability* of developing both DE and obesity. Hence, given these circumstances, a high neuroticism-score may imply practical health consequences.

To conclude, most likely, there is a complicated interplay between homeostatic regulators, heritage, internal and external cues which interact in the development of both EB in general and DE in particular. Additionally, there are also good reasons to assume that people who are genetically more disposed for weight gain, struggle even more to maintain healthier self-

regulation in eating when they are exposed to environmental food cues. Due to the fact that females are overrepresented among individuals with MO, and among patients undergoing BS, there is a need to expand our knowledge about the interplay between gender, DE and personality.

1.4 Treatment of obesity

1.4.1 Nonsurgical treatment

From a medical point of view, a successful long-term treatment is weight reduction – to achieve a significantly lower BMI over time in order to prevent serious complications like hypertension, diabetes and sleep apnea (95). A significant meaningful weight loss of ≥ 5% may be achieved in different sorts of non-surgical interventions. In the Look AHEAD-study (96) 50% of obese participants with diabetes achieved this goal through intensive interventions to change lifestyle. Using calorie restriction, physical activity and psycho-educational guidance only, life style changes among MO patients is difficult, but weight loss of ≥ 5% may be achievable (97). Researchers are still searching for new approaches in the treatment of obesity, and the study of Wadden (98) demonstrates that one third of the patients achieved long-term clinically meaningful weight loss with enhanced counselling. A clinical meaningful weight loss may result in a significant improvement of physical health. On the other hand, a weight reduction of ≥ 5% of maximum weight may not be sufficient for many patients with MO in order to lower the risk of medical complications and psychological maladjustment. For those patients, surgical treatment may be an option.

1.4.2 Surgical treatment

BS is considered when other treatments fail. Selection criteria for BS include BMI ≥ 40, or BMI ≥ 35 with at least the presence of one co-morbid condition (i.e. diabetes, hypertension or sleep apnea) and previous failures of weight loss attempts (99-101). In Norway the Roux-en-

Y gastric bypass (RYGB) and the sleeve gastrectomy (GS) (*Figure 2*) are the most common BS-procedures (102). In RYGB, the effects of limiting food intake and the nutrition absorbed are combined, while GS is a restrictive surgical procedure which only limits food intake (65).

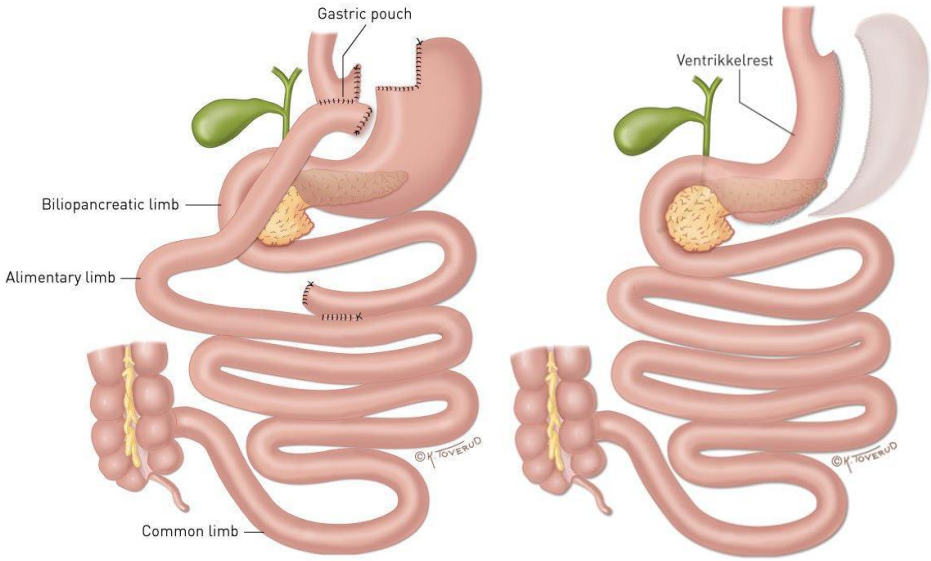

*Figure 2***.** Roux-en-Y gastric bypass (left), gastric sleeve (right).
(Figure by K. Toverud) (103)

1.4.3 The outcomes treatment

1.4.3.1 Surgical treatment

Both BS-procedures are designed to restrict food intake and nutrient malabsorption, and may result in significant long-term weight loss and reduction in DE (104-107).Other important factors explaining the significant weight reduction are also the decrease of hunger and the increase of satiety (108, 109). Nevertheless, long-term weight regain has been observed in approximately 20% of the patients (110-113) starting off rather immediately (within six months) after surgery (107). DE seems to increase the risk of relapse (22, 112-115). Findings from the SOS study (28) demonstrate that a) smaller decreases in UE during the first year and b) higher levels of UE at six months and 1 year postoperatively predict less-successful long-term weight loss after BS. In contrast, CR was unrelated to both short- and long-term weight

loss. These results confirm earlier findings (116) showing that loss of control over eating in the same post-surgical time interval predicted sub-optimal weight loss after surgery. The identification of post-surgical DE in these studies may be seen in relation to the development of different weight trajectories identified by Courcoulas et al (107) showing a variability in the amount of weight loss after the first postoperative year. These weight trajectories may be related to a redevelopment of DE postoperatively.

As many patients demonstrate DE both prior to (29, 33, 36-39) and after BS (21, 32, 39, 43, 44), the former may in fact predict a failure to maintain a successful weight loss (34, 45, 46) because such DE is characterised by overconsumption of energy dense food, overweight and obesity (17, 18, 89). Several attempts to identify possible psychological predictors in explaining sub-optimal weight loss after BS (22, 51, 115, 117-121) have, however, failed in providing consistent findings due to the diversity in measurements and terminology. One study (45) reports a development of grazing eating behaviours, but with less amount of food, while Toussi (121) identified BED as a predictor, while other studies report no predictive value (116), notably with respect to weight loss (115), and rather, that suboptimal weight loss is predicted by the level of *post*-operative BED-symptoms. Recently, Konttinen et al (28) published ten year follow-up data from the SOS study concerning the predictive values of eating behaviours and weight change after surgery. They concluded that lower levels of uncontrolled eating at 6 months and 1 year *after* surgery, and greater decrease in these EB, predicted significantly better short- and long-term weight loss (28).

1.4.3.2 Non-surgical treatments

In most of the nonsurgical treatment programmes including for instance mindfulness, cognitive behavioural therapy (CBT) and various forms of therapeutic support, weight reduction is the main target. In the literature a huge variety of behavioural interventions have been studied. A main conclusion is that patients lose weight, but most patients relapse into

substantial long-term weight-regain (122), and hence, the outcome is worse than for patients receiving BS. It has been reported that approximately 20% of overweight and obese may achieve a long term weight loss of 10% from the maximum weight (123, 124). In successful long-term weight losers, improved EBs have been identified (124).

This may demonstrate the difficulty in maintaining clinically meaningful weight loss, but also a treatment ineffectivity. Psychological treatments may delay weight regain, but the effects are modest (125, 126).

In contrast, the large multicentre randomised controlled trial Look AHEAD study (96), which included a manual-based intervention with CBT and motivational interviewing (MI) indicated that at least half of the patients in the intensive lifestyle group achieved a better restriction and control of their EB. The intensive intervention lasted for four years, and thereafter the participants were offered maintenance counselling. During the first year the participants were seen weekly for the first 6 months, and thereafter, group-sessions were offered every second week. In addition, individual visits were offered monthly. By comparison, the control group received educational- and social support sessions the first year in addition to the clinical diabetes visits. These results supported that a highly intensive manual-based intervention may have a favourable effects on EB and maintenance of weight loss.

1.4.3.3 Combining surgery and CBT

Concerning *pre-surgical* interventions, Ashton et al (127, 128) found that a brief group-based CBT-intervention improved binge eating behaviours before surgery. Additionally, a subgroup of patients who were identified as positive responders to the treatment had a significant larger weight loss 1 year after surgery. In the study of Abiles et al (129), psychological comorbidities were alleviated after a three months CBT intervention, both in patients with or without BED. In addition, other forms of pre-surgical intervention have been conducted, such

as preoperative counselling (130) which did not result in any significant group-differences in weight loss one year after surgery.

Regarding the impact of *post-surgical interventions*, Sarwer et al (131) found that dietary counselling had a short-term effect on DE and weight as the significant effects waned already six months following BS and stayed the same 1 year later, as well.

A number of studies have examined if CBT may alleviate BED symptoms (127, 128, 132-134). As DE might represent different variations or grades of severity of binge eating behaviour and that CBT is known to be the first-line therapy of treating BED (135), one might expect that a pre-surgical CBT intervention may alleviate DE. Additionally, CBT is among the best known empirically-supported treatment for a range of mental disorders, in particular anxiety and mood disorder (136). The studies of Ashton et al (127, 128) and Abiles et al (129) showed that pre-surgical interventions based on more psychological principles may reduce both affective symptoms and DE. These studies had a non-randomised design without control groups and do not permit causal conclusions about the effectiveness of a pre-surgical CBT intervention. Therefore, there is a lack of knowledge whether CBT before surgery may have an impact both before and 1 year after surgery.

Notably, CBT results in significant improvements in psychosocial functioning, but it does not produce weight loss (137). These findings may somehow be seen as contradictive: If the patients experience more control over EB and fewer episodes of overeating, one could logically presume that they would lose weight as well. Some of the core issues concerning weight reduction and changes in DE seem to be that weight- changes are affected by a spectrum of biological factors that strongly regulate weight, and EBs might therefore be considered as a moderator in weight regulation, and not necessarily a causal factor.

Therefore, regarding surgery for obesity, one may hypothesise that a combination of CBT and BS rather than BS alone may result in better maintenance of functional EB, and psychological health and possibly, weight loss as well.

In summary, approximately 70% of all patients undergoing BS are women, and an expanded knowledge concerning gender-related differences in DE in this population is needed. Moreover, high rates of DE are found both before and after surgery. First, based on data from the SOS study, uncontrolled eating following surgery seems to predict long-term suboptimal weight loss (28). Second, manual-based intensive interventions that include CBT techniques are related to significantly better weight loss and improvements in DE in obese patients with diabetes (96). Third, pre-surgical CBT interventions addressing DE (127, 128) are expected to facilitate weight loss. With this literature review in mind, there exist, to the best of our knowledge, no RCT that has examined whether a CBT intervention that is offered before surgery may improve DE, anxiety and depressive symptoms and weight loss 1 year after BS.

2 Aims of the thesis

The aims of this thesis are:

1. To explore gender-related differences in DE in patients admitted for BS, and the mediating role of specific personality traits, and symptoms of depression and anxiety, respectively.
2. To investigate whether CBT reduced DE in patients planned for BS.
3. To investigate one year after BS whether there were statistically significant differences between the two treatment arms with regard DE, mood and anxiety symptoms and weight, respectively.

3 Materials and methods

3.1 Study designs

All data were collected at baseline (Paper I), immediately after (Paper II), and one year after (Paper III) a 10-week CBT-intervention. Paper I used a cross-sectional design, while the remaining papers used a randomised controlled design.

3.1.1 Participants

A number of 167 patients were eligible during the inclusion period from September 2011 to April 2012. Patients were consecutively accepted for BS at the Morbid Obesity Centre (Vestfold Hospital Trust), and invited to participate in the present RCT study. This is a tertiary care centre located in Tønsberg, serving approximately one million inhabitants in Southern Norway.

3.2 Procedure

Four months prior to BS, immediately after the final CBT-intervention, and at a 1-year follow-up (December 2012-November 2013) patients were referred to a web-based form collecting demographic information as well as data about personality traits, eating behaviours, and affective and depressive symptoms. The questionnaires data based on validated and standardized instruments. The anthropometric measures were collected at the hospital. After the measurements, the patients were randomised into the treatment arm, i.e. a 10-week CBT-treatment and the usual care condition, by using a web-based programme (www.randomizer.org) to allocate blocks of patients. The participants were informed about the result of the randomization immediately after screening.

3.3 Attrition

Baseline data (Paper I) comprised the 102 patients who consented to participate, randomised to CBT (N= 50) or usual care (N=52), respectively. At the post-treatment (Paper II) four patients (two in each treatment condition) were lost early in the treatments due to trial fatigue. Despite an effort to reach out to the participants by invitation letters and offers to arrange flexible meetings 14 patients refused to participate (5 in the intervention - and 9 in the control group) in the follow-up (Paper III). The flow of the study participants is shown in *Figure 3*.

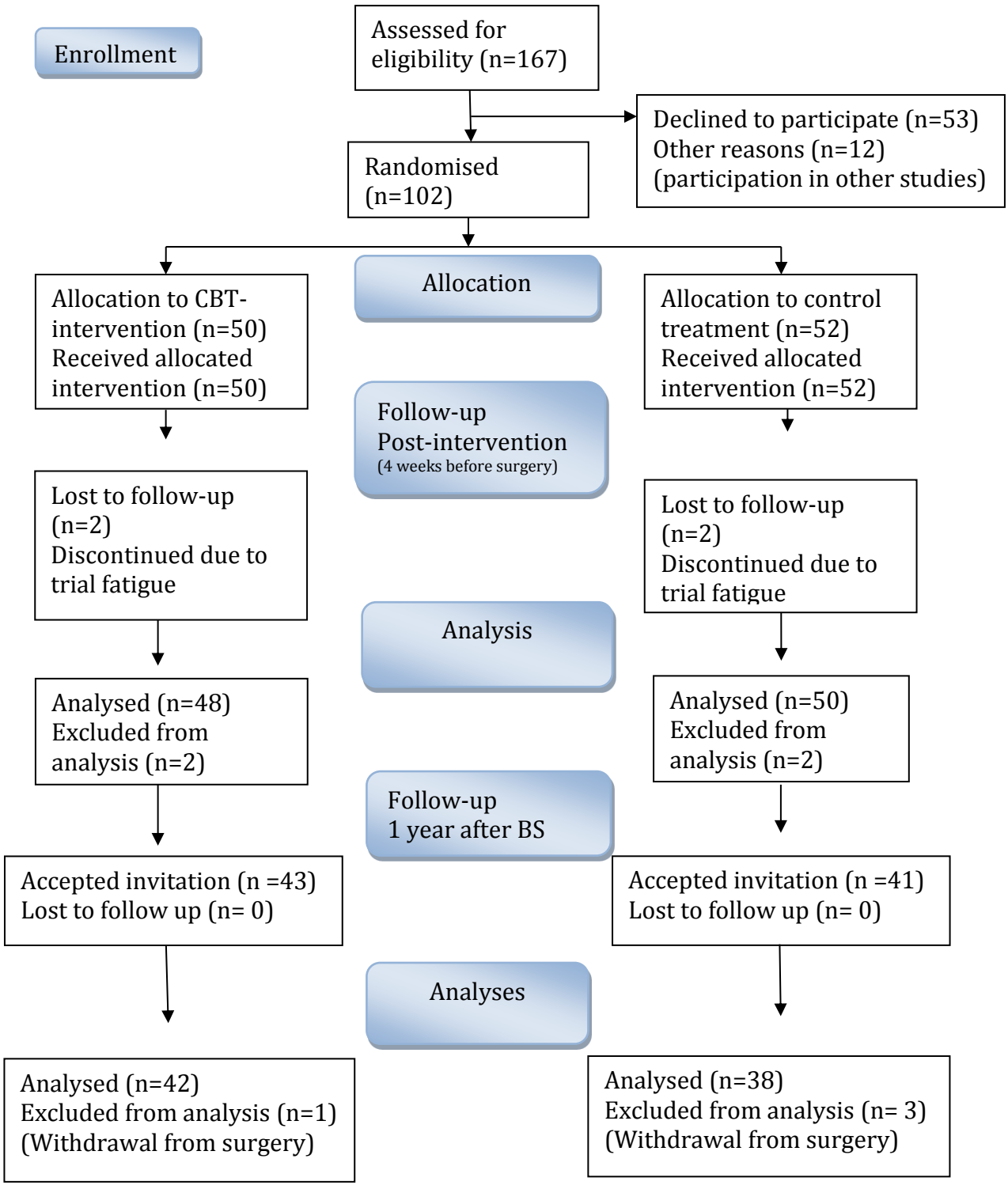

Figure 3. Flow of patients in the PSYMO study

3.4. Measurements/instruments

3.4.1 Anthropometric measures

Anthropometric measures were collected with patients in an upright position wearing light clothing and no shoes. Waist circumference was measured at the midpoint between the lowest rib margin and the iliac crest, whilst hip circumference was measured at the level of the major trochanter. Neck circumference was measured below the larynx and perpendicular to the long axis of the neck. Waist, hip and neck circumference were measured with a tape measure to the nearest cm. Height was measured with a wall mounted stadiometer to the nearest cm, whilst weight was measured to the nearest 0.5 kg.

3.4.2 Eating behaviours

The Norwegian version of the Three Factor Eating Questionnaire (TFEQ R-21) validated for measuring dysfunctional eating behaviours in obese individuals (24, 138) was used. It consists of 21 items comprising the subscales "emotional eating" (EE; 6 items), "uncontrolled eating" (UE; 9 items) and "cognitive restraint of eating" (CR; 6 items). The domain scores range was 0-100, and with 100 indicating maximal level of dysfunction.

3.4.3 Personality traits

The Norwegian version of the NEO Personality Inventory-Revised (NEO PI-R) (139) was used. The NEO PI-R is used world-wide and is based on the Five Factor Model (FFM) of personality (140). It consists of 240 items using a five-point Likert scale to measure five domains of personality: Neuroticism (N), Extraversion (E), Openness (O), Agreeableness (A) and Conscientiousness (C). The domain scores are each composed of six facet scores, and hence, mean raw scores were created for the five domain scores and the 30 facet scores and used in the correlation/regression analyses. Higher scores indicate that a trait is more present. Gender adjusted standardized T-scores (M=50, SD=10) were used for descriptive purposes.

3.4.4 Anxiety and depression

Anxiety and depression were measured by the Hospital Anxiety and Depression Scale (HADS) (141). The HADS is a self-report measure of non-vegetative affective symptoms (141, 142) where seven items assess depression (HADS-D) and seven items measure anxiety (HADS-A), respectively. Items are scored 0-3 yielding a range of 0-21 within each subscale. A cut-off ≥ 8 is used in Norway to indicate a probable diagnosis of depression or anxiety (143).

3.5 Pre-surgical interventions

3.5.1 Pre-surgery treatment

During the four months prior to surgery all patients (both groups) received up to three consultations, either from a medical doctor, a dietician, a nurse or a physiotherapist. The patients received educational materials concerning nutritional recommendations, detailed information about the mandatory low calorie diet the last three weeks before surgery as well as guidance about recommended physical activity level and intensity.

3.5.2 The CBT intervention

The intervention group received in addition a series of 10 sessions (five at the hospital and five as scheduled telephone calls to the patients at home) based on principles from self-determination theory and motivational interviewing, focusing on an accepting communication style to lower resistance and increase patients' perceived competence and intrinsic motivation to change (144). This was focused during the first two sessions. The remaining sessions were bases on principles from CBT. *Table 1* provides an overview of the contents of the sessions.

Table 1. Overview of the 10-week CBT-intervention

Sessions	Session content
Session 1 *(At the centre)* *Both groups*	• Establishing rapport with the patient in order to facilitate a good therapeutic working alliance. • Provide information about the interventions to all patients • Conduct the baseline measurements and perform the randomization and informing the patients about their allocated group.
Session 2 *(At the centre)*	• Introduction to the underlying principles of the therapy (working transparently, collaboratively, time-limited and using a manual) • Inform the patient about CBT and the treatment plans in the study • Psycho-education focusing on the relationships between eating behaviours, cognitive- and behavioral patterns, affect-regulation and obesity, thus introducing the patients for the CBT model. • Introducing and explaining home-work sheets for session 3 and 4
Sessions 3 + 4 *(By telephone-calls)*	• Reviewing the patient's home-work sheets • Recognizing and addressing dysfunctional eating behaviours • Working with the patient's behavioral eating patterns (what triggers eating), and the associated cognitions and emotions. • Providing the patient's means to assess their own perception about recognizing improvement in dysfunctional cognitions and eating behaviours.
Session 5 *(At the centre)*	• Coping with situational "triggers" that may lead to dysfunctional cognitive- and eating behavioral patterns • Working with the patient's cognitive- and behavioral eating patterns ("triggers", cognition, emotion and eating behaviour) • Introducing and explaining home-work sheets for sessions 6 & 7
Session 6 & 7 (*By telephone calls)*	• Reviewing the patient's home-work sheets • Continuing the intervention techniques • Reinforcing positive changes in eating behaviours
Session 8 *(At the centre)*	• Continuation or refining intervention techniques (as session 5) by guiding the patient in avoiding situational "triggers" and making a plan for practicing new eating behaviours. • Introducing and explaining home-work sheets for sessions 9 & 10
Session 9 & 10 (*By telephone calls)*	• Reviewing the patient's home-work sheets • Continuation or refining intervention techniques
Session 11 *(At the centre)*	• Relapse prevention • Ending of treatment and helping the patient to maintain positive changes

As for the CBT intervention, the manual (Table 1) was designed to fit this particular study sample, which may provide a better fit than using manuals developed for obese patients in general or for eating disorders (134, 145, 146). However, it was based on the fundamental principle from CBT. As most patients struggle with DE both before and after surgery, a key element of the intervention was to improve DE. However, patients also struggled with other facets of eating problem, and the intervention thus also contained interventions to address the regularity of eating or to change cognitive self-talk about affect-regulated eating and thereby establishing less emotional-triggered eating. Thus, the manual was based on the principles from eating disorder specific CBT manuals, i.e. that a behavioural change is the most powerful way of changing cognitive change (147).

However, both cognitive and behavioural techniques were used, including psycho-education, and the ABC-model to counter negative automatic thoughts, rumination and worries and how cognition and emotions influence DE. In most of the sessions, monitoring records (sheets) to depict associations between situational (triggers), cognition, emotional activation and eating were extensively used. These records were also used as home-work assignments in conjunction to the hospital sessions, but new home-work tasks were not initiated during the sessions run by telephone calls. In the initial session, functional analyses were included aiming to engage the patients in the treatment, to define which roles the DE-symptoms may have in the maintenance of DE and to "transform" the patients' symptoms of DE to useful aims of treatment.

3.6 Statistics

The SPSS version 17 was used for all statistical analyses. In all papers data are presented as mean (SD), mean (95 % CI) or number (%). Moreover, between- group differences were examined using either analysis of covariance (ANCOVA), independent sample t-test (for continuous data), linear mixed modelling analyses or Fisher's exact tests (categorical data).

ANCOVA compares the two post-test group mean scores after adjusting for baseline scores. Effect sizes are reported as Hedges' g indicating the differences between the groups in standard deviations. Values between 0.20 -0.49, from 0.50-0.79 and from 0.80 and higher are considered as small, moderate and high respectively (146). Associations between the continuous variables were examined with Pearson`s bivariate correlations and hierarchical multiple stepwise regression analyses. A hierarchical approach was used to assess how much of the variation in dysfunctional eating patterns that was related to three blocks of variables: 1) demographic information (i.e. age, gender, educational level status, and BMI), 2) affective symptoms (i.e. anxious and depressed mood), and 3) personality traits (i.e. NEO PI-R). Moreover, the multiple mediation analysis was conducted (Paper I) using an SPSS macro by Preacher & Hayes (148) as it allows covariate control. Given the number of analyses, the alpha levels needed to deem a statistical test as significant were set to < .001 in the bivariate analyses (tables 1 and 2) and < .01 in the multivariate analysis.

In *paper III*, linear mixed regression models were built including three factors: a within factor (examining change across time), a between factor (examining differences between the treatment arms), and a final random intercept factor (adjusting for individual differences at baseline). The within factor had three levels (repeated three times: T0, T1 and T3). All outcome variables measured at baseline (T0), post CBT-intervention/pre-surgically (T1), and one year post-surgery (T2) represented time factors. The between factor had two levels: the control (G0) and intervention (G1) group. The two-way interaction (*Group* × *Time*) thus represented a test of the hypothesized treatment effect. Conducting post-hoc tests were imperative in clarifying whether there were any remaining treatment effects at T2. The restricted maximum likelihood estimation method was used.

3.7 Ethics

The study was approved by the Regional Committee for Medical and Health Research Ethics (reference number 2010/2071a), registered in the ClinicalTrials.gov-registry (identifier NCT01403558), and conducted according to the Helsinki declaration.

3.8 Funding

The PSYMO-study has been fully funded by an unrestricted research grant from the **South-Eastern Regional Health Authority, Norway**

4 Results

4.1 Paper I

Psychological correlates to dysfunctional eating patterns among morbidly obese patients accepted for bariatric surgery

Our major finding was that personality traits were more firmly related to DE than mood disorder symptoms. Anxiety and depression were weakly related to such patterns when personality traits were controlled for. Neuroticism and conscientiousness were the prime personality traits involved in EE and UE/CR, and could be ascribed a vulnerable and protective role, respectively. Moreover, EE occurred more often in female patients as previously reported (22, 55, 149). A multiple mediation analysis was conducted to examine if personality, depression or anxiety played a role as mediators for the relationship between gender and EE. Personality was the only significant mediator. The initial direct path (gender-EE, controlled for BMI) was β = -.46 ($p < .001$), but dropped to β = -.28 ($p < .001$) after adding personality as the indirect path β = -.18 (CI 95% -.29 to -.09). The indirect path explained 39% (.18/.46) of the total variance in EE (*Figure 4*).

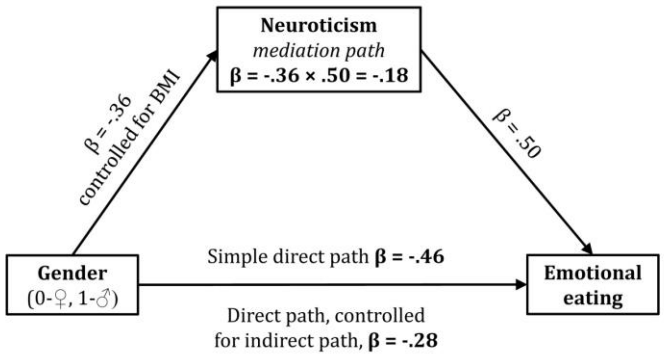

Figure 4. The mediation model with standardised beta coefficients

4.2 Paper II

Efficacy of a cognitive behavioural intervention on dysfunctional eating behaviours in patients admitted for bariatric surgery. A randomised controlled trial

The patients in the CBT group had significant improvements in DE, anxiety and depression compared with the control group patients. A significant reduction in BMI was also observed.

The between-group effect sizes for the improvements varied from high (uncontrolled eating (g = -.90, p = ≤.001), cognitive restraint (g = .92, p = ≤.001)), to moderate (emotional eating (g = -.67, p = ≤.001), anxiety (g = -.62, p = ≤.001), depression (g = -.73, p = ≤.001) and low (BMI (g = -.24, p = .004). Adjusted between group differences at follow-up for EE, UE and CR were -19 (95% CI, -26 to -12), -19 (95 % CI, -25 to -14) and 20 (95% CI, -28 to -13) respectively, all $p \leq .001$. For anxiety and depression the adjusted between group differences were -2.5 (95 % CI, -3.5 to -1.4) and -2.8 (95 % CI, -3.9 to -1.6) respectively, both $p \leq .001$. Concerning BMI and body weight, the adjusted between group differences were -1.1 (95 % CI, -1.8 to -.35, p = .004), and -3 (95 % CI, -5.1 to -.84, p = .004).

As shown in *Figure 5* and *6*, the patients in the intervention group changed their EBs, affective symptoms and mood significantly in a positive direction. (These change score-figures were given as an oral presentation at the European Conference of Obesity (ECO) in Liverpool, May 2013).

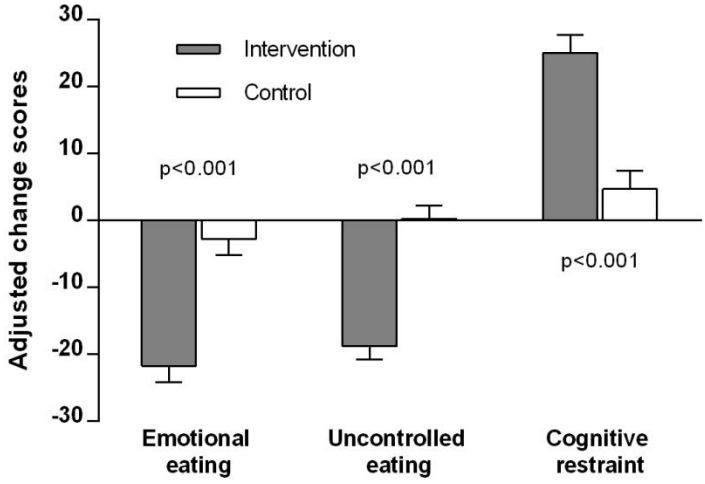

Figure 5. Post-intervention change scores in eating behaviours by treatment arm

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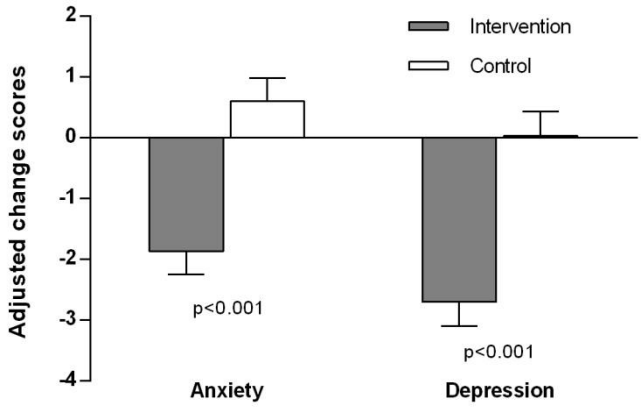

Figure 6. Post-intervention change scores in anxiety and depression by treatment arm

4.3 Paper III

The impact of a pre-surgical cognitive behavioral therapy (CBT) on dysfunctional eating behaviours, affective symptoms and body weight one year after bariatric surgery. A randomised controlled trial

Contrary to expectations, patients in both treatment arms were indistinguishable at the one-year follow-up. Importantly, however, there were positive changes in both groups over time,

and the two groups revealed different patterns of changes in all EBs and affective symptoms. In contrast, the patterns of weight loss did not differ between the groups.

Dysfunctional eating behaviours

Pairwise post-hoc tests revealed significant improvements in EE, UE and CR in the CBT group between T0 and T1 (M_{diff} = -22.62, M_{diff} = -19.49 and M_{diff} = 25.93, respectively, all p's < .001). Further improvements between T1 and T2 were evident for EE and UE only (M_{diff} = -8.48, p = .02; M_{diff} = -11.90, p < .001, respectively).

The improvement in the control group was only evident between T1 and T2 for EE and UE (M_{diff} = -19.97, p < .001; M_{diff} = -24.30, p < .001), and between T1 and T3 for CR (M_{diff} = 9.50, p = .02).

Group differences: The post-hoc tests revealed significant group differences favouring CBT only at T1 for EE, UE and CR (M_{diff} = -14.61, p < .01; M_{diff} = -15.60, p < .01; M_{diff} = -18.96, p < .001, respectively), but not at T2.

Anxiety and depression

There was a significant reduction in anxiety and depression symptoms in the CBT group between T0 and T1 (M_{diff} = -1.88 and M_{diff} = -2.69, respectively, both p's < .001), and between T1 and T2 for depression only (M_{diff} = -1.04, p = .03). In the control group, the anxiety score did not change significantly, whereas depression scores went down significantly between T1 and T2 (M_{diff} = -2.68, p < .001).

Group differences: No significant group differences were observed for anxiety, whereas a significant group difference favouring CBT was evident at T1 (M_{diff} = -1.71, p < .01), but not at T2.

BMI

BMI declined significantly in both groups after surgery, T1-T2 (CBT and control group; M_{diff} = -12.60 and -13.79, both p's < .001), however no significant between group differences were revealed at any time point.

5 Discussion

5.1 Main findings

The main findings from this thesis were:

- The personality traits neuroticism and conscientiousness were more strongly related to DE than symptoms of mood and anxiety. Anxiety and depression were weakly related to DE when personality traits were controlled for. An additional finding was that neuroticism partially mediated this relationship, thus indicating a putative mechanism. Moreover, EE occurred more often in female patients.
- The patients in the intervention group improved their DE significantly, thus supporting the potential benefit of using CBT intervention for this group. Patients were less inclined to eat for emotional reasons and to lose control of their eating following the intervention, as well as a better ability to regulate or stop eating when satisfied. A supplementary benefit was a reduction in the symptoms of anxiety and depression. Finally, a small, yet statistically significant reduction in BMI was found.
- One year after BS, the two groups were indistinguishable in terms of weight loss, and both groups had comparable improvements in DE and symptoms of anxiety and depression. However, the onset of improvement in the CBT-group was earlier in all DE, affective and mood symptoms.

5.2 Discussions of main findings

5.2.1 The impact of CBT in the treatment of DE in patients admitted for BS

Before surgery, the patients in the CBT group showed a significant reduction in DE and a moderate alleviation of anxiety and depression compared to the control group. In addition,

patients in the CBT group lost about 3 kg body weight. The follow-up one year after surgery, demonstrates that the two groups had comparable improvements in all outcomes. Importantly, however, there were positive changes in both groups over time, yet with different patterns of changes in all EBs and affective symptoms.

5.2.2 CBT and changes in DE before and after surgery

Previous studies (127-129) have found effects of a CBT-intervention for BE and psychological comorbidities. These finding are relevant considering the cognitive and behavioural similarities between BE and DE as outlined in the introduction part of this thesis. Also, CBT has proven effective to alleviate affective and mood disorders (136, 150). On the one hand, our findings (Paper II) concur with the previous sparse number of comparable studies: DE and symptoms of anxiety and depression were significantly improved before surgery. On the other, these studies had a non-randomised design with no control groups, thus it is uncertain whether the improvements were caused by the CBT intervention or by other factors. Our findings are based on a randomised controlled design which provides more reliable conclusions.

Considering the purpose of establishing an evidence base for a treatment effect of a psychological intervention additive to a BS-effect, the non-significant group differences at the one-year follow-up may be discouraging. Here, several models of explanation may be possible. A *delayed* effect may be concealed by the relatively brief follow-up interval. The fact that the great variability in the amount and trajectory of postoperative weight loss is firstly seen two years after BS (107), may indicate that the effects of a CBT intervention may take longer to show up. Furthermore, this may indicate that the effects of the CBT intervention may be stronger when the biological effects of BS start to wane after the first year of surgery.

Another model relates to an *incomplete delivery* of the intervention. For instance, half of the 10-week intervention was given as planned telephone calls. Even though these telephone-sessions included specific CBT elements, they may have been less effective. In the absence of face-to-face communication, the scheduled telephone appointments might have been perceived less seriously and, possibly, less committing than those given in the hospital setting. This option clearly points to the role of non-specific alliance factors as well as the genuine role of face-to-face contact to attain maximal effect of the CBT interventions. Additionally, due to the relatively brief CBT intervention, case formulations were not included. This limitation may have created an intervention less tailored to individual needs, thus possibly being experienced as less personally relevant, and further reducing personal commitments. On the other hand, functional analyses included in the intervention may have countered this potential negative effect.

Several studies (28, 151, 152) show that BS has a comprehensive impact on DE, affective symptoms and weight. In the PSYMO study, the results indicate that the CBT intervention exceeded usual care in terms of *an earlier onset* of reduction of DE, anxiety and depression, which in turn indicates more functional coping with daily stress and more control over eating. These patterns of results also indicate that the advantageous improvements before surgery might be less "weight-loss dependent", meaning that it is the significant weight loss itself, which promotes the improvements in DE, affective symptoms and mood.

An expanded understanding of DE and the change of these behaviours, the comprehensive effect of BS should be considered. BS patients have a reduced pouch after surgery. This makes it difficult to eat large portions of food, and bypassing the duodenum has most probably a major effect on the neuroendocrine regulation of hunger and appetite (153-155), DE as well as mood. In sum, these direct effects of BS may "rule out" any kind of long-term

effects from psychological interventions. As seen in the *Starvation experiment*" (9), hunger is a very strong driver and affects both cognition, emotions and DE. From a neuro-hormonal perspective, the postoperative altering of for instance the hunger hormone ghrelin might also partly explain the significant improvements in mental health in these patients, not only the weight loss itself.

5.2.3 The interrelationship between DE and body weight before surgery

Despite the significant improvements in DE in the CBT group before BS, the mean weight loss was only 3 kg. The CBT group reported significantly improvement in DE at the post-test, i.e. more restrained eating, less use of energy-dense food to regulate negative emotions, and fewer episodes of UE and impulse-driven snacking. Although the aim of the intervention was *not* weight reduction, with these improvements, one could expect that the intervention group on average would lose more than 3 kg during the 10 week intervention in terms of a significant lower intake of calories. Even in studies where weight reduction is the principal target, psychological methods *alone* might not have a significant impact on weight loss maintenance (145), meaning that weight reduction may in fact be rather resistant to psychological interventions. Studies of CBT for BE show the same tendencies: The patients report fewer episodes of DE after the therapy, but they do not lose weight (135). As discussed in part 1.3, the drivers for food intake are complex (156, 157) being controlled by signals from monitoring organs (158). This aspect may lend further support to the notion that perceiving significant improvements in DE may not automatically imply catabolic states and significant weight loss in all patients with MO.

5.2.4 Changes in body weight one year after BS

The postoperative weight outcomes may, to a certain extent, be comparable with a recent study (159) showing that including both pre- and post-surgical psychological support failed to provide significantly differences in weight loss one year after surgery. In the PSYMO study, patients in both groups reduced their weight on average approximately 40 kg. Most of the patients experience maximum weight change during the first year after BS. One may assume that the body may be resistant to further weight loss the first year after surgery (104, 160). In this regard, a pre-surgical CBT intervention will not have any additional impact beyond the surgery.

In retrospect, it might have been too optimistic to expect that the CBT intervention could have any *additional* effect beyond the surgery. However, additive effects may be conceived in a different way. Thus, the effect of an earlier onset of behavioural and cognitive change may be clinically important as the experience that change is possible even before the expectant change due to BS may promote self-efficacy and coping.

5.2.5 Common and specific factors

Both common and specific factors may affect the treatment outcome. The common factors include contextual factors, notably the therapist-patient alliance, which consists of the interplay between therapist and patient, including a mutual understanding of the goals of treatment and how to achieve them. It is known that satisfaction with treatment might be linked to how treatment is delivered (161, 162), and one important element in explaining therapeutic change is the quality of the therapeutic interaction between patient and therapist (163, 164).

Patients admitted for BS do definitely have common characteristics, but this is also a heterogeneous group with for instance different patterns of DE, and the way the CBT

treatment was delivered must be considered to be more flexible than strictly monitored by the manual. This implies that there may have been differences in the "doses" of the CBT treatment.

The failure not to include measurements of therapeutic alliance and manual-adherence makes it difficult to sort out common and specific effects of the CBT intervention. This opens the possibility of potent change factors unrelated to the CBT-specific treatment.

5.2.6 Gender-related differences in DE and the impact of mediators

General population studies (2, 165) show a higher probability of MO among women, and that around 70% of the patients admitted for BS are female (105). Moreover, in MO patients admitted for BS, higher rates of psychopathology are associated with female gender (72). One of the main findings (Paper I) was that female patients admitted for BS displayed significantly more DE. However, the exact nature of the relationship between gender and DE is not known. Gender differences in EBs in general may expand the understanding of gender and DE (166, 167). EBs may be explained both by genetic- and environmental factors concerning the regulation of energy balance, hunger, appetite and reward-seeking behaviour (168, 169). One possible explanation of "the female morbid obesity-DE –axis", is that emotion- regulated eating may be a determinant of food intake (157). EE is associated with higher consumption of sweet and fatty food, and this association is stronger in women (170, 171). A Finnish study including over 5000 adolescents showed that stress-related eating was more common in female than in male participants (172). One possible explanation is that a female vulnerability to emotion-regulated eating affects BMI, but one might also suspect the other way around – that being a women with a high BMI affects EE. The fact that morbidly obese women report having significantly lower health-related quality of life in obesity- specific emotional measurements than men (173), may also indicate that females with MO suffer significantly

more because of their weight. The negative emotional states of for instance shame, guilt and distress may in some women trigger EE.

A score ≥ 60 on the TFRQ R-21may be considered as dysfunctional for EE, yet this is not well established. Additional analyses (not shown in this thesis) indicate that 64% of the female patients and 15 % of the males had a score of ≥ 60 in EE at baseline. While gender-related differences were not the main focus of Paper III, additional sub-analyses showed that even one year after surgery, female patients display significantly more EE than males. In fact, there were significant correlations between postoperative EE and BMI in women but not in men.

DE observed in MO patients seems associated with personality traits (29, 36, 55) and particular with the trait neuroticism (53-55). The second major finding in Paper I demonstrates how personality traits and gender may account for individual differences in EE. The fact that patients admitted for BS have significantly higher levels of neuroticism than obese patients not admitted for surgery (174), indicates that the surgery group of MO are even further psychologically vulnerable than MO in general. The novelty of the findings indicates that neuroticism is a mechanism that partly explains the relationship between gender and EE. The mediating role of neuroticism may indicate that this trait is a key factor to modify in female bariatric surgery patients' DE. This stands in contrast to Elfhag and Morey (55) who found negligible gender differences in the correlations between DE and personality traits, like neuroticism. A highly likely explanation of these contradictive findings is that Elfhag and Morey (55) used standardised T-scores adjusting for gender differences whereas we used unadjusted raw-scores that do not mask naturally occurring gender differences in personality traits. Affective symptoms and mood, which have been identified in other studies as possible mechanisms (175) were in our study weakly related to EE when personality traits were

controlled for. One possible interpretation is that personality permeates most aspects of humans' cognitive and emotional life and behaviour, and it covers broader aspects of vulnerability than just depression and anxiety.

5.3 Methodological considerations

5.3.1. External validity

The target population in this study was patients with MO who were accepted for BS. The participants were consecutively admitted to surgery in a large tertiary care centre. Regarding the representativeness of the participants who were randomised, the baseline measurements concur with previous reports including BMI, gender, EB, anxiety and depression (28, 65, 151, 152, 176), personality (55, 60) and socio-economic status (177, 178). On the other hand, patients admitted for BS may be a heterogeneous population, and our findings may only be generalised to the population admitted for BS in public hospitals. Furthermore, while 167 patients were invited to participate, 53 patients declined for unknown reasons not further probed due to restrictions laid down in the ethical approval document. Another issue that may affect the external validity is "program contamination". The patients in the CBT-group were explicitly advised not to share homework sheets, or any other materials connected to the treatment, with the patients in the control group. Despite this solicitation, we have no guarantee whether any contamination of treatment effect may have occurred, meaning that participants potentially have "shared treatment" with the control group. Being aware of the fact that many patients admitted for BS prefer connecting to a variety of "BS-groups" on Facebook, a treatment contamination may have occurred.

5.3.2 Internal validity

The recording of anthropometric measures was conducted according to standards of such measurements, and they were performed by the same researcher, which may imply consistency in these assessments.

The measurements of personality traits, EBs, affective symptoms and mood are based on self-report assessments. The use of a web-based data collection method in a structured hospital setting have strengthened the reliability by reducing the loss of variance and the need for imputation due to scattered missing data. Being requested to complete instruments displayed by a computer may in principle be unfamiliar to some patients, yet the fact that only one participant had very limited computer skills, and that assistance was offered during the completion does not represent a limitation to this study. Furthermore, the instruments are commonly used in this patient population (151, 152, 176, 179, 180) and may imply a reduction of error variance.

Another concern relevant for validity is the impact of "impression management". In some countries like in the USA, pre-surgical evaluations may be decisive for being eligible for surgery. In these evaluations, underreporting of anxiety, depression and eating pathology has been identified (181). In the PSMO study, impression management measurements were not included, because at the time of the baseline measurements, all participants were already accepted for surgery. Therefore, we do not suspect any significant and systematic underreporting of symptoms.

In conclusion, using self-administered, well-established and validated assessment methods in a controlled hospital setting are methodological strengths.

Paper I had a cross-sectional design, which does not allow inferring causality. An attempt to identify predictors of DE was made, but the results of the mediation analyses need to be

replicated to generalise the findings. In considering the large number of significant test that were conducted implying an uncertainty of whether *n* was sufficient, the alpha levels were lowered to 0.001 in the bivariate analyses and 0.01 in the multivariate regression analyses.
In Paper II, analysis of covariance (ANCOVA) was used to examine the treatment effects. By having a randomised controlled design with non-significant differences between the two groups would minimise this source of error. Moreover, not losing statistical power and to reduce the within-group error variance were the main reasons for using ANCOVA.
The calculation of sample size was based on the primary outcomes in Paper II, and the sample-size in **Paper III** may have been under-powered. In evaluating the treatment effects, we conducted a multilevel model using linear mixed models to study the time development in the two groups. Mixed models analyses were well-suited in the setting where repeated measurements were made on the same population, as these analyses are better suited to handle drop-out of patients over time.

In principle, the randomised controlled design makes it possible to draw causal conclusions (182). However, in the present study, the failure to include measurements of therapeutic alliance and manual adherence makes it difficult to sort out common and specific effects of the CBT intervention. This opens the possibility of potent change factors unrelated to the CBT-specific treatment.
On the one hand, the PSYMO study may be considered as a *pragmatic* study in the sense that it was conducted within a routine clinical context for a specified patient group (183) (i.e. MO-patients seeking BS in a specialist care centre). Moreover, a manual-based intervention was used, the attrition rates were low to moderate, and there were no statistical significant differences in baseline measurements between participants and drop-outs. Of note, there was only one researcher doing the CBT intervention, and without an additional placebo group or a

group receiving another therapy intervention, there may be certain doubts whether this is a pragmatic study.

5.4 Implications for treatment

About 4-5% of MO people in Norway qualify for BS (2). The number of patients undergoing BS is high, but most probably the amount of BS has been stable in Scandinavia during the last two years (102, 184). We consider the data from the PSYMO study to be representative for public hospital treatment-seeking morbidly obese patients admitted for bariatric surgery. As our data show, the prevalence of general psychopathology and DE is very high, and our findings of gender-related differences may imply more individualised and tailored psychological treatments for this patient population. A pre-surgical treatment based on psychological principles may improve patients' control over food intake, reduce emotion-regulated eating as well as symptoms of anxiety and depression. Such improvements may result in better compliance with dietary recommendations.

5.5 Implications for further research

The use of RCT is sparse within this field, and should become a standard procedure in the search for treatment improvements. Also, future studies should search for clusters of personality traits which may predict relapse of DE and suboptimal weight loss or weight regain after BS.

Furthermore, the present findings highlight the need to focus on the nature and content of a CBT intervention as well as the optimal time of delivering it. Considering our one year follow-up results, it might be useful to test our CBT intervention post-surgically, as previous findings indicate that DE also may occur later than one year postoperatively and result in

impaired outcomes of BS. Finally, future studies should include a measure of therapeutic alliance to control for nonspecific factors initiating change.

6 Conclusions

This thesis may provide two main conclusions:

First, the thesis provides provisional new knowledge about how personality traits and gender may account for individual differences in EE among patients admitted for BS and the clinically relevant interplay between gender, psychological health and personality constructs.

Second, the thesis indicates an immediate impact of a pre-surgical CBT intervention on DE, affective symptoms, mood and body weight, but lends no support for a superior effect of CBT (plus BS) compared with a usual care (plus BS) at follow-up one year later.

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8 Appendix

- NEO PI-R
- Three Factor Eating Questionnaire (TFEQ R-21)
- Hospital Anxiety and Depression Scale (HADS)
- Paper I
- Paper II
- Paper III

NEO PI-R

Revidert NEO Personlighetsinventorium (NEO PI-R)

Spørsmålshefte

Paul T. Costa Jr., Ph. D. og Robert R. McCrae, Ph. D.

Instruksjoner for utfylling

Vær vennlig å lese alle instruksjonene under grundig før du begynner. Marker alle dine svar på svararket, og skriv bare der du skal. Ikke skriv i dette heftet.

På det vedlagte svarskjemaet kan du skrive navnet ditt eller en kode på den øverste linjen dersom du har blitt bedt om det. Skriv også ned datoen for utfylling, alder, kjønn, utdanning og yrke nederst på arket.

Spørreskjemaet inneholder 240 spørsmål. Les hvert spørsmål nøye, og sett en ring rundt det svaralternativet som passer best i forhold til om du er enig eller uenig i utsagnet.

Sett en ring rundt «SU» dersom utsagnet er helt uriktig eller dersom du er svært uenig.	(SU) U N E SE
Sett en ring rundt «U» dersom utsagnet er nokså uriktig eller dersom du er uenig.	SU (U) N E SE
Sett en ring rundt «N» dersom utsagnet er omtrent like riktig som uriktig, hvis du ikke kan bestemme deg, eller hvis du er nøytral i forhold til utsagnet.	SU U (N) E SE
Sett en ring rundt «E» dersom utsagnet stort sett er riktig eller hvis du er enig.	SU U N (E) SE
Sett en ring rundt «SE» dersom utsagnet er helt riktig eller dersom du er svært enig.	SU U N E (SE)

Det er ingen riktige eller gale svar, og du trenger ikke å være ekspert for å fylle ut spørreskjemaet. Beskriv deg selv på en ærlig måte, og uttrykk dine meninger så nøyaktig du kan.

Besvar alle spørsmålene/utsagnene. Merk deg at alle svarene er markert nedover i kolonnene på svararket. Vær vennlig å kontrollere at svaret for hvert spørsmål blir markert på korrekt sted på svararket.

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1. Jeg er ikke en person som pleier å bekymre seg.
2. Jeg liker virkelig de fleste mennesker jeg møter.
3. Jeg har en svært livlig fantasi.
4. Jeg har en tendens til å være kynisk og skeptisk til andres hensikter.
5. Jeg er kjent for min klokskap og sunne fornuft.
6. Jeg blir ofte sint over måten folk behandler meg på.
7. Jeg skyr menneskemengder.
8. Det som angår kunst og estetikk, er ikke spesielt viktig for meg.
9. Jeg er ikke utspekulert eller slu.
10. Jeg vil heller holde mine valgmuligheter åpne enn å planlegge alt på forhånd.
11. Jeg føler meg sjelden ensom eller nedfor.
12. Jeg er dominerende, sterk og selvhevdende.
13. Uten sterke følelser ville livet være uinteressant for meg.
14. Noen mennesker synes at jeg er selvopptatt og egoistisk.
15. Jeg forsøker å utføre alle oppgaver jeg blir pålagt på en samvittighetsfull måte.
16. I omgang med andre mennesker er jeg alltid redd for å dumme meg ut.
17. Jeg har en bedagelig stil i både arbeid og fritid.
18. Jeg har ganske faste vaner.
19. Jeg vil heller samarbeide med andre enn å konkurrere med dem.
20. Jeg engasjerer meg lite og tar ting som de kommer.
21. Jeg fråtser sjelden i noe.
22. Jeg higer ofte etter spenning.
23. Jeg liker ofte å leke med teorier eller abstrakte ideer.
24. Jeg har ingenting imot å skryte av meg selv og det jeg har utrettet.
25. Jeg er ganske flink til å tilpasse tempoet slik at jeg får gjort ting i tide.
26. Jeg føler meg ofte hjelpeløs og ønsker at andre skal løse problemene mine.
27. Jeg har aldri bokstavelig talt hoppet av glede.
28. Jeg tror at studenter bare blir forvirret og villedet av å høre kontroversielle talere.
29. Politiske ledere må være mer oppmerksomme på menneskelige sider av politikken sin.
30. I årenes løp har jeg gjort noen temmelig dumme ting.
31. Jeg blir lett skremt.
32. Jeg har ikke særlig glede av å småprate med folk.
33. Jeg forsøker å styre tankene mine inn i realistiske baner og unngå fantasiflukt.
34. Jeg tror at de fleste mennesker i bunn og grunn er velmenende.
35. Jeg tar ikke borgerplikter som f.eks. å stemme ved valg særlig alvorlig.
36. Jeg er en avbalansert person.
37. Jeg liker å ha mange mennesker rundt meg.
38. Jeg er noen ganger fullstendig oppslukt av den musikken jeg lytter til.
39. Om nødvendig er jeg villig til å manipulere folk for å få det som jeg vil.
40. Jeg holder mine eiendeler ordentlige og rene.
41. Noen ganger føler jeg meg fullstendig verdiløs.
42. Noen ganger klarer jeg ikke å hevde meg så mye som jeg burde.
43. Jeg har sjelden sterke følelser.
44. Jeg forsøker å være høflig mot alle jeg møter.

45. Noen ganger er jeg ikke så pålitelig eller til å stole på som jeg burde være.
46. Jeg føler meg sjelden sjenert når jeg er blant folk.
47. Når jeg gjør noe, gjør jeg det med stor iver og energi.
48. Jeg synes det er interessant å lære og utvikle nye hobbyer.
49. Jeg kan være sarkastisk og skarp når jeg må.
50. Jeg har klare mål og arbeider systematisk for å nå dem.
51. Jeg har problemer med å motstå mine lyster.
52. Jeg ville ikke like å feriere på et sted med mye natteliv, spill og underholdning.
53. Jeg synes filosofiske diskusjoner er kjedelige.
54. Jeg vil helst ikke snakke om meg selv og det jeg har utrettet.
55. Jeg kaster bort mye tid før jeg kommer i gang med arbeidet.
56. Jeg føler at jeg er i stand til å mestre de fleste av mine problemer.
57. Jeg har noen ganger opplevd intens glede eller ekstase.
58. Jeg tror at lover og velferdspolitikk burde endres for å avspeile behovene i en verden i endring.
59. Jeg er nøktern og usentimental i mine holdninger.
60. Jeg tenker gjennom ting før jeg tar en beslutning.
61. Jeg føler meg sjelden redd eller engstelig.
62. Jeg er kjent som en varm og vennlig person.
63. Jeg har et rikt fantasiliv.
64. Jeg tror de fleste mennesker vil utnytte deg hvis du lar dem gjøre det.
65. Jeg holder meg informert og tar vanligvis fornuftige avgjørelser.
66. Jeg er kjent for å være en hissig og oppfarende person.
67. Jeg foretrekker vanligvis å gjøre ting alene.
68. Jeg synes det er kjedelig å se på ballett eller moderne dans.
69. Jeg kunne ikke lure noen selv om jeg ønsket det.
70. Jeg er ikke en utpreget systematisk person.
71. Jeg er sjelden trist eller deprimert.
72. Jeg har ofte vært leder i grupper jeg har tilhørt.
73. Det er viktig for meg hva jeg føler om ting.
74. Noen mennesker ser på meg som kald og beregnende.
75. Jeg betaler min gjeld punktlig og i sin helhet.
76. Til tider har jeg vært så skamfull at jeg bare har ønsket å gjemme meg.
77. Jeg har en tendens til å arbeide langsomt, men jevnt og trutt.
78. Når jeg først finner den riktige måten å gjøre noe på, holder jeg meg til den.
79. Jeg nøler med å uttrykke mitt sinne selv når det er berettiget.
80. Når jeg starter et selvforbedringsopplegg, lar jeg det vanligvis skli ut etter noen få dager.
81. Jeg har ikke særlig problemer med å motstå fristelser.
82. Jeg har av og til gjort ting bare for «kicket» eller spenningens skyld.
83. Jeg liker å løse problemer eller «tankenøtter».
84. Jeg er bedre enn de fleste mennesker, og jeg vet det.
85. Jeg er en produktiv person som alltid får arbeidet unna.
86. Når jeg er svært stresset, føles det av og til som jeg går i stykker.
87. Jeg er ikke en munter optimist.

88. Jeg synes vi skal holde oss til våre religiøse autoriteter når det gjelder avgjørelser i moralske spørsmål.
89. Vi kan aldri gjøre for mye for de fattige og de eldre.
90. Av og til handler jeg først og tenker etterpå.
91. Jeg føler meg ofte anspent og nervøs.
92. Mange ser på meg som noe kald og fjern.
93. Jeg liker ikke å kaste bort tiden på å dagdrømme.
94. Jeg tror at de fleste mennesker jeg har med å gjøre, er ærlige og til å stole på.
95. Jeg kommer ofte opp i situasjoner uten å være fullt forberedt.
96. Jeg blir ikke betraktet som en nærtakende eller temperamentsfull person.
97. Jeg føler virkelig behov for andre mennesker når jeg er lenge for meg selv.
98. Jeg er fascinert av de mønstrene jeg finner i kunst og natur.
99. Det å være helt ærlig er en dårlig måte å gjøre forretninger på.
100. Jeg liker å ha alt på sin plass slik at jeg vet akkurat hvor det er.
101. Jeg har av og til opplevd en dyp følelse av skyld eller synd.
102. I møter lar jeg vanligvis andre stå for snakkingen.
103. Jeg legger sjelden vekt på de følelser jeg har i øyeblikket.
104. Jeg forsøker som regel å være omtenksom og hensynsfull.
105. Av og til jukser jeg når jeg legger kabal.
106. Jeg blir ikke særlig forlegen om folk gjør narr av meg og erter meg.
107. Jeg føler det ofte som om jeg strutter av energi.
108. Jeg prøver ofte ny og fremmed mat.
109. Hvis jeg ikke liker folk, lar jeg dem få vite det.
110. Jeg arbeider hardt for å nå mine mål.
111. Når jeg får livrettene mine, har jeg en tendens til å spise for mye.
112. Jeg unngår helst filmer som er sjokkerende eller skremmende.
113. Noen ganger mister jeg interessen når folk snakker om svært abstrakte, teoretiske ting.
114. Jeg forsøker å være ydmyk.
115. Jeg har problemer med å få meg selv til å gjøre det jeg burde gjøre.
116. Jeg holder hodet kaldt i krisesituasjoner.
117. Noen ganger bobler jeg av lykke.
118. Jeg tror at de forskjellige oppfatninger av rett og galt som folk i andre samfunn har, kan være riktige for dem.
119. Jeg har ingen sympati for tiggere.
120. Jeg vurderer alltid konsekvensene før jeg handler.
121. Jeg er sjelden bekymret for fremtiden.
122. Jeg liker veldig godt å snakke med folk.
123. Jeg liker å konsentrere meg om en fantasi eller dagdrøm, utforske dens muligheter og la den vokse og utvikle seg.
124. Jeg blir mistenksom når noen gjør noe hyggelig mot meg.
125. Jeg er stolt av min gode vurderingsevne.
126. Jeg føler ofte avsky for folk jeg er nødt til å ha med å gjøre.
127. Jeg foretrekker jobber der jeg kan arbeide alene uten å bli forstyrret av andre mennesker.
128. Dikt og poesi har liten eller ingen virkning på meg.

129. Jeg ville hate å bli ansett som en hykler.
130. Det virker som om jeg aldri greier å organisere meg selv.
131. Jeg har en tendens til bebreide meg selv når noe går galt.
132. Andre holder seg ofte til meg når avgjørelser skal tas.
133. Jeg opplever et bredt spekter av stemninger og følelser.
134. Jeg er ikke kjent for min gavmildhet eller sjenerøsitet.
135. Når jeg forplikter meg til noe, kan en alltid stole på at jeg følger opp.
136. Jeg føler meg ofte underlegen i forhold til andre.
137. Jeg er ikke så kvikk og livlig som andre mennesker.
138. Jeg foretrekker å holde meg i kjente omgivelser.
139. Når jeg er blitt fornærmet, forsøker jeg bare å tilgi og glemme.
140. Det føles ikke som om noe driver meg fremover.
141. Jeg gir sjelden etter for mine impulser eller innfall.
142. Jeg liker å være der det skjer noe.
143. Jeg liker å arbeide med «tankenøtter» hvor jeg må vri hjernen.
144. Jeg har svært høye tanker om meg selv.
145. Når jeg starter på et prosjekt, fullfører jeg det nesten alltid.
146. Jeg har ofte vanskelig for å bestemme meg.
147. Jeg betrakter meg ikke som spesielt lett til sinns.
148. Jeg tror det er viktigere å være lojal mot sine idealer og prinsipper enn å ha et «åpent sinn».
149. Menneskelige behov burde alltid prioriteres fremfor økonomiske hensyn.
150. Jeg tar ofte ting på «på sparket».
151. Jeg bekymrer meg ofte for ting som kan gå galt.
152. Jeg synes det er lett å være blid og utadvendt overfor fremmede.
153. Hvis jeg føler at jeg holder på å drømme meg bort, sørger jeg som regel for å konsentrere meg om arbeid eller annen aktivitet i stedet.
154. Min umiddelbare reaksjon er å stole på folk.
155. Det ser ut som om jeg ikke lykkes helt med noe som helst.
156. Det skal mye til for å få meg sint.
157. Jeg vil heller feriere på en populær strand enn i en isolert hytte i skogen.
158. Visse typer musikk slutter aldri å fascinere meg.
159. Noen ganger lurer jeg folk til å gjøre det jeg vil de skal gjøre.
160. Jeg har en tendens til å være noe kresen og nøyaktig.
161. Jeg har ikke høye tanker om meg selv.
162. Jeg vil heller gå mine egne veier enn å være en leder for andre.
163. Jeg registrerer sjelden stemninger eller følelser som ulike omgivelser kan skape.
164. De fleste jeg kjenner, liker meg.
165. Jeg holder meg strengt til mine etiske prinsipper.
166. Jeg føler meg vel i nærvær av mine sjefer eller andre autoriteter.
167. Det virker som om jeg vanligvis har det travelt.
168. Av og til gjør jeg forandringer i huset bare for å prøve noe annet.
169. Hvis noen starter en krangel, er jeg parat til å ta igjen.
170. Jeg anstrenger meg for å utrette så mye jeg kan.

171. Noen ganger spiser jeg så mye at jeg blir kvalm.
172. Jeg elsker spenningen på berg og dalbaner.
173. Jeg er lite interessert i å spekulere over universets natur eller menneskets vilkår.
174. Jeg føler ikke at jeg er bedre enn andre, uansett hvilken tilstand de er i.
175. Når et prosjekt blir for vanskelig, er jeg tilbøyelig til å starte på et nytt.
176. Jeg greier meg ganske bra i en krise.
177. Jeg er en munter, livlig person.
178. Jeg anser meg selv for å være vidsynt og tolerant overfor andre menneskers livsstil.
179. Jeg mener at alle mennesker fortjener respekt.
180. Jeg tar sjelden forhastede avgjørelser.
181. Jeg har færre redsler enn folk flest.
182. Jeg er sterkt følelsesmessig knyttet til vennene mine.
183. Som barn likte jeg sjelden fantasilek.
184. Jeg pleier å tro det beste om folk.
185. Jeg er en svært kompetent person.
186. Til tider har jeg følt meg harm og forbitret.
187. Sosiale sammenkomster er vanligvis kjedelige for meg.
188. Noen ganger når jeg leser dikt eller ser et kunstverk, føler jeg en gysning eller en bølge av begeistring.
189. Til tider herser eller smisker jeg med folk for å få dem til å gjøre det jeg vil.
190. Jeg er ikke tvangsmessig opptatt av rengjøring.
191. Noen ganger ser alt nokså trist og håpløst ut for meg.
192. I samtaler har jeg en tendens til å stå for det meste av snakkingen.
193. Jeg synes det er lett å være empatisk – å selv føle det andre føler.
194. Jeg ser på meg selv som en nestekjærlig person.
195. Jeg forsøker å gjøre ting nøyaktig slik at de ikke må gjøres om igjen.
196. Hvis jeg har sagt eller gjort noe galt mot noen, har jeg fryktelig vanskelig for å se dem i øynene etterpå.
197. Livet mitt er hektisk.
198. Når jeg skal feriere, foretrekker jeg å dra tilbake til et kjent og kjært sted.
199. Jeg er sta og egenrådig.
200. Jeg streber etter å gjøre det utmerket i alt jeg gjør.
201. Noen ganger gjør jeg ting på impuls som jeg senere angrer på.
202. Jeg er tiltrukket av sterke farger og en prangende stil.
203. Jeg har mye intellektuell nysgjerrighet.
204. Jeg vil heller rose andre enn selv bli rost.
205. Det er så mange småjobber som må gjøres at jeg av og til ganske enkelt ignorerer dem.
206. Når alt ser ut til å gå galt, kan jeg likevel ta gode avgjørelser.
207. Jeg benytter sjelden ord som «fantastisk» eller «sensasjonelt» for å beskrive mine opplevelser.
208. Jeg mener at hvis folk ikke vet hva de tror på når de er 25, er det noe galt med dem.
209. Jeg har sympati for dem som har vært mindre heldige enn meg.
210. Jeg planlegger nøye på forhånd før jeg skal ut på en reise.
211. Av og til får jeg skremmende tanker.

212. Jeg er personlig interessert i de menneskene jeg arbeider sammen med.
213. Jeg ville ha vansker med bare å la tankene vandre uten kontroll eller styring.
214. Jeg har stor tiltro til menneskets natur.
215. Jeg er effektiv og produktiv i mitt arbeid.
216. Selv mindre ergrelser kan være frustrerende for meg.
217. Jeg liker selskaper med mange mennesker.
218. Jeg liker å lese dikt som legger vekt på følelser og forestillinger fremfor rene fortellinger.
219. Jeg er stolt av min skarpsindighet i å håndtere mennesker.
220. Jeg bruker mye tid på å lete etter ting jeg har forlagt.
221. Når noe går galt, blir jeg altfor ofte motløs og får lyst til å gi opp.
222. Det er ikke lett for meg å ta føringen i en situasjon.
223. Merkelige ting – som visse dufter eller navn på fjerne steder – kan vekke sterke sinnsstemninger i meg.
224. Jeg strekker meg langt for å hjelpe andre mennesker hvis jeg kan.
225. Jeg må virkelig være syk for å bli borte fra jobben en dag.
226. Når folk jeg kjenner gjør noe dumt, blir jeg flau på deres vegne.
227. Jeg er en svært aktiv person.
228. Jeg følger alltid den samme ruten når jeg skal et sted.
229. Jeg kommer ofte opp i argumentering/uenighet med min familie og mine kolleger.
230. Jeg er litt av en arbeidsnarkoman.
231. Jeg er alltid i stand til å holde mine følelser under kontroll.
232. Jeg liker å være i tilskuermengden ved sportsarrangementer.
233. Jeg har et vidt spekter av intellektuelle interesser.
234. Jeg er en suveren person.
235. Jeg har stor selvdisiplin.
236. Jeg er ganske følelsesmessig stabil.
237. Jeg har lett for å le.
238. Jeg mener at den «nye moral», som tillater det meste, ikke er noen moral i det hele tatt.
239. Jeg vil heller være kjent som «barmhjertig» enn som «rettferdig».
240. Jeg tenker meg om to ganger før jeg svarer på et spørsmål.

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*

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.

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*

HAD

Hospital Anxiety & Depression Scale (januar 1999)

Navn: _____ Fødselsdato: _____

Dato for utfylling: _____ Pasient nr.: _____

Behandler: _____

Rettledning

Legen er klar over at følelser spiller en stor rolle ved de fleste sykdommer. Hvis legen vet mer om følelser, vil han/hun bli bedre i stand til å hjelpe deg.

Her kommer noen spørsmål om hvorledes du føler deg. For hvert spørsmål setter du kryss for ett av de fire svarene som best beskriver dine følelser den siste uken. Ikke tenk for lenge på svaret – de spontane svarene er best.

1. Jeg føler meg nervøs og urolig

- ❑ 3 Mesteparten av tiden
- ❑ 2 Mye av tiden
- ❑ 1 Fra tid til annen
- ❑ 0 Ikke i det hele tatt

2. Jeg gleder meg fortsatt over tingene slik jeg pleide før

- ❑ 0 Avgjort like mye
- ❑ 1 Ikke fullt så mye
- ❑ 2 Bare lite grann
- ❑ 3 Ikke i det hele tatt

3. Jeg har en urofølelse som om noe forferdelig vil skje

- ❑ 3 Ja, og noe svært ille
- ❑ 2 Ja, ikke så veldig ille
- ❑ 1 Litt, bekymrer meg lite
- ❑ 0 Ikke i det hele tatt

4. Jeg kan le og se det morsomme i situasjoner

- ❑ 0 Like mye nå som før
- ❑ 1 Ikke like mye nå som før
- ❑ 2 Avgjort ikke som før
- ❑ 3 Ikke i det hele tatt

5. Jeg har hodet fullt av bekymringer

- ❑ 3 Veldig ofte
- ❑ 2 Ganske ofte
- ❑ 1 Av og til
- ❑ 0 En gang i blant

6. Jeg er i godt humør

- ❑ 3 Aldri
- ❑ 2 Noen ganger
- ❑ 1 Ganske ofte
- ❑ 0 For det meste

7. Jeg kan sitte i fred og ro og kjenne meg avslappet

- ❑ 0 Ja, helt klart
- ❑ 1 Vanligvis
- ❑ 2 Ikke så ofte
- ❑ 3 Ikke i det hele tatt

8. Jeg føler meg som om alt går langsommere

- ❑ 3 Nesten hele tiden
- ❑ 2 Svært ofte
- ❑ 1 Fra tid til annen
- ❑ 0 Ikke i det hele tatt

9. Jeg føler meg urolig som om jeg har sommerfugler i magen

- ❑ 0 Ikke i det hele tatt
- ❑ 1 Fra tid til annen
- ❑ 2 Ganske ofte
- ❑ 3 Svært ofte

10. Jeg bryr meg ikke lenger om hvordan jeg ser ut

- ❑ 3 Ja, jeg har sluttet å bry meg
- ❑ 2 Ikke som jeg burde
- ❑ 1 Kan hende ikke nok
- ❑ 0 Bryr meg som før

11. Jeg er rastløs som om jeg stadig må være aktiv

- ❑ 3 Uten tvil svært mye
- ❑ 2 Ganske mye
- ❑ 1 Ikke så veldig mye
- ❑ 0 Ikke i det hele tatt

12. Jeg ser med glede frem til hendelser og ting

- ❑ 0 Like mye som før
- ❑ 1 Heller mindre enn før
- ❑ 2 Avgjort mindre enn før
- ❑ 3 Nesten ikke i det hele tatt

13. Jeg kan plutselig få en følelse av panikk

- ❑ 3 Uten tvil svært ofte
- ❑ 2 Ganske ofte
- ❑ 1 Ikke så veldig ofte
- ❑ 0 Ikke i det hele tatt

14. Jeg kan glede meg over gode bøker, radio og TV

- ❑ 0 Ofte
- ❑ 1 Fra tid til annen
- ❑ 2 Ikke så ofte
- ❑ 3 Svært sjelden

Takk for utfyllingen!

Sum A:

1+3+5+7+9+11+13= _____

Sum D:

2+4+6+8+10+12+14= _____

Sum A + D: _____

Skåringsveiledning til HAD

(Hospital Anxiety and Depression Scale)

Selvutfylling på sju angst- og depresjonsspørsmål.

Sum A eller Sum D:
En skår på 11 eller mer regnes for å være et tilfelle av angst eller depresjon som vil trenge nærmere utredning (med SPIFA for eksempel) og eventuelt behandling. En skår på 8-10 anses som et mulig tilfelle, og lavere skår uttrykker en viss symptombelastning, som kan ha betydning samlet sett, men som i seg selv ikke krever spesifikk behandling av angst eller depresjon.

Sum A + Sum D:
Det er også mulig å legge sammen angst- og depresjonsskåren til en totalskår fordi en del pasienter har en blanding av angst og depresjon. Et tilfelle vil da ha en totalskår på 19 eller mer. Et mulig tilfelle vil ha en skår på 15-18. Skår på over 15 vil trenge oppfølging og eventuelt behandling.

Dersom inntil to spørsmål på HAD er ubesvart, vil det være mulig å beregne totalskår. Sumskåren deles med antallet besvarte spørsmål og svaret ganges med 14. Dette gir estimert totalskår.

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Original Article

Psychological Correlates to Dysfunctional Eating Patterns among Morbidly Obese Patients Accepted for Bariatric Surgery

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Key Words
Obesity · Dysfunctional eating behaviours · Personality traits · Affective symptoms · Bariatric surgery

Abstract
Objective: To examine the relationships between dysfunctional eating patterns, personality, anxiety and depression in morbidly obese patients accepted for bariatric surgery. ***Design***: The study used cross-sectional data collected by running a randomized controlled trial (*http://clinicaltrials.gov/ct2/show/NCT01403558*). ***Subjects***: A total of 102 patients (69 women, 33 men) with a mean (SD) age of 42.6 (9.8) years and a mean BMI of 43.5 (4.4) kg/m^2 participated. ***Measurements***: Measurements included the NEO-PI-R (personality: neuroticism, extroversion, openness, conscientiousness and agreeableness), the TFEQ-R-21 (dysfunctional eating: emotional eating (EE), uncontrolled eating (UE) and cognitive restraint of eating (CR)) and the HADS (anxiety and depression). ***Results***: The personality traits neuroticism and conscientiousness were more strongly correlated with dysfunctional eating than anxiety and depression. These differences were most pronounced for emotional and cognitive restraint of eating. Emotional eating occurred more often in female than in male patients, a finding that was partially mediated by neuroticism but not by anxiety and depression. ***Conclusion***: Personality traits may be important to address in the clinical management of morbidly obese patients seeking bariatric surgery as neuroticism is particularly salient in female patients displaying an emotional eating behaviour.

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Introduction

Approximately 10–25% of obese patients considered for or treated by bariatric surgery have dysfunctional eating patterns [1–4]. Dysfunctional eating patterns can be operationalized as emotional eating (EE), uncontrolled eating (UE) and cognitive restraint of eating (CR) [5]. EE serves to regulate negative mood states like feeling lonely, anxious or depressed. On the other hand, people with UE tend to lose control over their eating behaviour when feeling hungry while people with CR tend to overly restrict their food intake in order to control weight, body shape or body composition. Hence, all three patterns serve to regulate or suppress negative emotional states. These patterns may result in overconsumption of energy-dense food [6], and extreme variations may qualify for a DSM-IV-defined unspecified eating disorder or a binge eating disorder (BED) [7].

The clinical importance of pre-surgical BED symptoms for the outcome of bariatric surgery is unsettled. One study [8] reported a development of grazing eating behaviours, but with less amount of food. Other studies reported no predictive value [9], notably with respect to weight loss [10], but rather that suboptimal weight loss was predicted by the level of post-operative BED symptoms.

Measures of dysfunctional eating seem to show a more consistent picture [3, 11–14] for predicting weight regain and impaired post-operative weight loss.

Depression and anxiety are common comorbid conditions in patients seeking bariatric surgery [15, 16] – with a prevalence of 16% and 22%, respectively [17]. Depression is known to disrupt self-regulatory sleep and eating behaviours [18] related to impaired motivation and anhedonia. Anxiety on the other hand may prompt worries about future consequences of non-sustainable changes and a reluctance to implement behavioural changes. Also, mood disorders appear to be a consequence of being morbidly obese [19].

A psychological construct of large impact is personality as it permeates most aspects of humans' cognitive and emotional life and behaviour [20], including eating behaviours. Dysfunctional eating patterns observed among morbidly obese patients seem to be related to personality traits [2, 21–23] and, in particular, to neurotic personality traits [24–26]. However, the exact nature of this relationship in conjunction with gender and symptoms of mood disorders is not known. Though, in female pre-bariatric obese patients, two broad personality subtypes have been observed [27]: one normal and a second psychopathological type. Patients of the latter type score higher on 'neuroticism', and lower on the traits 'extraversion', 'agreeableness' and 'conscientiousness'. They also display significantly more binge eating and dysfunctional eating behaviours that may have emotion-regulating functions.

Women constitute the majority of morbidly obese persons including those seeking bariatric surgery [28]. Disturbed eating patterns are also more prevalent among women in the general population [29], and EE seems to be more frequent among morbidly obese women [30]. The gender differences in the correlation between eating behaviours and personality traits have been reported as small to negligible in the obesity literature [24]. The findings have also been inconsistent due to a small number of participating patients and failures to include bariatric surgery patients. In the current study we therefore aimed to explore gender-related differences in dysfunctional eating behaviours in patients admitted for bariatric surgery and the mediating role of specific personality traits as well as of symptoms of depression and anxiety.

We hypothesized that 1) female morbidly obese patients accepted for bariatric surgery had a higher level of emotional eating than men, and 2) that personality traits, anxiety and depression may partly explain hypothesis 1 indirectly via mediation.

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Material and Methods

Participants

The inclusion criteria were obesity grade 2 (BMI 35.0–39.9 kg/m^2) and at least one obesity-related comorbidity or obesity grade 3 (BMI ≥ 40.0 kg/m^2) and being accepted for bariatric surgery at the Morbid Obesity Centre at Vestfold Hospital Trust. There were no exclusion criteria.

We included 102 patients (69 females and 33 males) consecutively admitted between September 2011 and June 2012 for bariatric surgery at the Morbid Obesity Centre at Vestfold Hospital Trust in Norway. The mean BMI was 43.5 (SD 4.9) kg/m^2, and the majority (79.4% n = 81) had obesity grade 3.

Measures

Descriptive variables comprised age, gender, educational level as well as body weight and body height.

The Norwegian version of the Three Factor Eating Questionnaire (TFEQ-R-21) validated for measuring eating behaviours in obese individuals [5, 31] was used to measure dysfunctional eating patterns. It consists of 21 items comprising the subscales 'emotional eating' (EE; 6 items), 'uncontrolled eating' (UE; 9 items) and 'cognitive restraint of eating' (CR; 6 items). The domain scores range from 0 to 100. The level of dysfunction is indicated by higher scores on all subscales. All subscales have shown high internal consistency (Cronbach's α ranging from 0.70 to 0.90) [31].

The Norwegian version of the NEO Personality Inventory-Revised (NEO-PI-R) [32] was used to assess personality traits. The NEO-PI-R is used world-wide and is based on the Five Factor Model (FFM) of personality [33]. It consists of 240 items using a five-point Likert scale to measure five domains of personality: neuroticism (N), extraversion (E), openness (O), agreeableness (A) and conscientiousness (C). The domain scores are each composed of six facet scores, and hence, mean raw scores were created for the five domain scores and the 30 facet scores and used in the correlation/regression analyses. Higher scores indicated more of the trait. Gender-adjusted standardized T-scores (M = 50, SD = 10) were additionally used for descriptive purposes.

Anxiety and depression were measured by the Hospital Anxiety and Depression Scale (HADS) [34]. HADS is a self-report measure of non-vegetative affective symptoms [34, 35] where seven items assess depression (HADS-D) and seven items measure anxiety (HADS-A). Items are scored 0–3 yielding a range of 0–21 within each subscale. In Norway a cut-off ≥ 8 is used to indicate a probable diagnosis of depression or anxiety [36]. The Cronbach's α for HADS-A vary from 0.68 to 0.93 (mean 0.83), and for HADS-D from 0.67 to 0.90 (mean 0.82) [34, 36].

Procedures

Four months prior to bariatric surgery and after the completion of the informed consent form, data were collected by a web-based solution (Metreno and FluidSurveys) during the hospital visit.

Height, using a wall-mounted stadiometer, and weight were measured to calculate the BMI (kg/m^2).

The procedures were initiated after the study had been approved by the Regional Committee for Medical and Health Research Ethics (2010/2071a).

Statistical Analyses

The SPSS version 17 was used for all statistical analyses. Group differences were examined with independent sample t-tests (continuous data) and Fisher's exact tests (categorical data). Associations between the continuous variables were examined with Pearson's bivariate correlations and hierarchical multiple stepwise regression analyses. A hierarchical approach was used to assess how much of the variation in dysfunctional eating patterns was related to three blocks of variables: 1) demographic information (i.e., age, gender, educational level and BMI), 2) affective symptoms (i.e., anxious and depressed mood) and 3) personality traits (i.e., NEO-PI-R).

Given the number of analyses, the α levels were set to 0.001 in the bivariate analyses (tables 1, 2) and 0.01 in the multivariate analysis (table 3), and, accordingly, p values ≤ 0.001 and ≤ 0.01 were considered significant.

Multiple Mediation Analysis

A multiple mediation analysis was conducted using an SPSS macro by Preacher and Hayes [37] allowing covariate control. The contributions by multiple mediators, i.e. personality, anxiety and depression, were simultaneously examined as indirect paths. Bootstrapped standard errors were estimated through 1,000 re-samplings [37] to obtain correct confidence intervals for the indirect path or the mediator coefficient.

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Table 1. Descriptive total sample and gender specific statistics for BMI, age, eating behaviours, anxiety, depression and personality for patients accepted for bariatric surgery (N = 102)

	Men (n = 33), M (SD)	Women (n = 69), M (SD)	t	g	α
BMI	43.3 (5.7)	43.6 (4.4)	0.29	0.06	
Age	45.6 (10.3)	41.2 (9.3)	2.17	–0.46	
Eating behaviors					
Emotional eating	35.0 (20.9)	60.7 (24.1)	5.26*	1.11	0.92
Uncontrolled eating	46.4 (19.2)	50.2 (18.7)	0.96	0.20	0.73
Cognitive restraint	42.8 (19.3)	44.8 (21.1)	0.46	0.10	0.84
Anxiety	4.9 (3.5)	7.6 (3.9)	3.40*	0.72	0.84
Depression	4.9 (3.5)	5.2 (3.4)	0.47	0.10	0.78
Personality traits					
Unadjusted raw scores					
Neuroticism	82.0 (26.7)	103.3 (25.4)	3.89*	0.97	0.89
Extraversion	103.4 (23.8)	109.9 (19.8)	1.46	0.31	0.78
Openness	108.5 (15.5)	104.4 (17.7)	1.16	–0.24	0.65
Agreeableness	129.4 (16.1)	122.6 (20.2)	1.82	–0.36	0.75
Conscientiousness	115.0 (22.6)	111.8 (19.5)	–0.74	–0.16	0.84
Adjusted T scores					
Neuroticism	52.8 (11.1)	54.9 (11.1)	0.77	0.18	
Extraversion	42.2 (11.2)	45.8 (9.8)	0.27	0.35	
Openness	44.6 (8.0)	43.0 (9.6)	0.19	–0.17	
Agreeableness	52.7 (12.5)	53.3 (10.6)	0.13	0.06	
Conscientiousness	48.9 (11.0)	49.5 (10.3)	0.72	0.05	

t = Student's t-test; * $p < 0.001$; g = Hedge's g effect size; α = Cronbach's α.

Results

The 102 participants (69 women, 33 men) had a mean (SD) age of 42.6 (9.8) years and a BMI of 43.5 (4.9) kg/m^2. Women reported significantly more emotional eating and anxiety symptoms. The unadjusted personality scores showed higher neuroticism in women, but no gender differences were found in the norm-adjusted T-scores (table 1).

Anxiety and Depression

Using the HADS cut-off score ≥ 8 for a possible diagnosis, no gender differences were found for the prevalence of anxiety (men 33%, women 45%, $p = 0.29$) or depression (men 24%, women 24%, $p = 0.999$).

Bivariate Associations between Personality Traits, Eating Behaviours and Mood

The personality trait neuroticism was strongly positively correlated with EE, UE, anxiety and depression, and negatively with CR and age. Conscientiousness was negatively correlated with EE, UE, depression and anxiety, and positively correlated with CR.

Regression Analyses Predicting Emotional Eating, Uncontrolled Eating and Cognitive Restraint

The hierarchical regression models (table 3) showed in the first step that women reported significantly more EE than men, while high BMI was related to higher levels of dysfunction on all three eating pattern measures. In the second step, patients with more anxiety and, but to a lesser extent, more depression reported more EE relative to those with less anxiety or depression. Depressive symptoms were positively related with a dysfunctional eating pattern

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Table 2. Correlation coefficients between the demographic variables, eating behaviours, anxiety, depression and personality traits (N = 102)*

	1	2	3	4	5	6	7	8	9	10	11	12
1 Emotional eating												
2 Uncontrolled eating	0.61											
3 Cognitive restraint	–0.36	–0.42										
4 Anxiety	0.52	0.32	–0.19									
5 Depression	0.38	0.45	–0.28	0.44								
6 Age	–0.09	0.11	0.12	–0.26	0.03							
7 BMI	0.27	0.20	–0.22	0.05	0.19	–0.04						
8 Neuroticism	0.63	0.44	–0.37	0.70	0.49	–0.31	0.21					
9 Extraversion	–0.12	–0.21	0.17	–0.11	–0.42	–0.08	–0.07	–0.32				
10 Openness	–0.08	–0.06	0.04	–0.04	–0.08	0.06	0.01	–0.13	0.43			
11 Agreeableness	–0.00	–0.18	0.28	–0.01	–0.16	0.21	–0.04	–0.18	0.03	0.14		
12 Conscientiousness	–0.43	–0.43	0.42	–0.27	–0.35	0.16	–0.24	–0.56	0.32	0.23	0.32	

*Pearson correlations above 0.31 are significant at $p < 0.001$.

Table 3. Hierarchical stepwise multiple regression analyses predicting dysfunctional eating behaviours

Step / variable	Emotional eating			Uncontrolled eating			Cognitive restraint		
	initial β	final β	adj R^2	initial β	final β	adj R^2	initial β	final β	adj R^2
Step 1									
Gender	–0.47***	–0.29***							
BMI	0.26**	0.16	0.27	0.20	0.07	0.03	–0.22	–0.11	0.04
Step 2									
Anxiety	0.40***	0.12							
Depression	0.18	0.11	0.43	0.43***	0.33***	0.20	–0.24	–0.13	0.09
Step 3									
Neuroticism		0.36**	0.48						
Conscientiousness					–0.30**	0.27		0.34***	0.18

β = Standardized regression coefficient; *** $p < 0.001$; ** $p < 0.01$.

adj R^2 = Adjusted R-square. Variables excluded from the model: Age and socio-economic status in step 1, and extraversion, openness and agreeableness in step 3. An empty table cell implies that the included variable was non-significant in the prediction of the relevant outcome measure.

across all three measures (i.e., EE, UE and CR), having the largest negative impact on UE. Adding of personality traits in the last step showed that neuroticism was positively associated with EE, but importantly, this personality trait also rendered anxiety and depression non-significant. For the other two outcomes, conscientiousness was the most significant personality trait, also substituting the relation depression had with CR. In both cases conscientiousness represented a protective effect. The fact that no outliers and non-linear regression effects were detected argues for the generalizability of the regression models.

Due to the statistically significant domain scores for neuroticism and conscientiousness, follow-up regression analyses were conducted by replacing these domain scores with their facet scores. For EE, impulsiveness (β = 0.28, p = 0.001) and depression (β = 0.29, p = 0.006)

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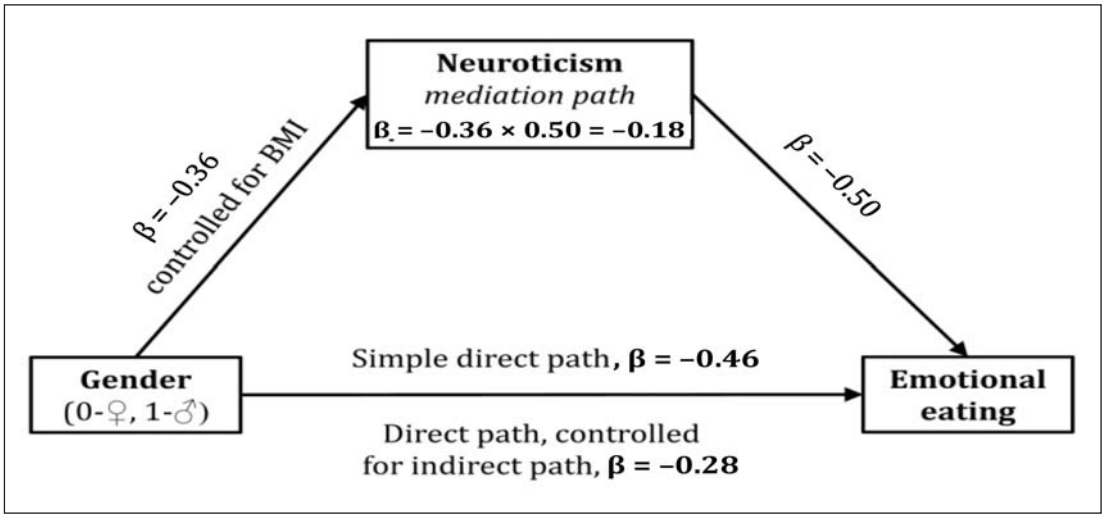

Fig. 1. The mediation model with standardized beta coefficients.

were the most important neuroticism facets in explaining EE (total $R^2 = 0.56$). For UE, the neuroticism facets impulsiveness (β = 0.26, p = 0.005) and vulnerability (β = 0.27, p = 0.007) contributed most to the regression model (total $R^2 = 0.35$). For CR, the conscientiousness and neuroticism facets dutifulness (β = 0.34, p = 0.001) and impulsiveness (β = –0.34, p = 0.001), respectively, contributed most to the model (total $R^2 = 0.33$). Using facet rather than domain scores increased the amount of variance explained (R^2) considerably, and in particular for CR.

Mediation Analysis of the Gender Differences in EE

A multiple mediation analysis was conducted to examine if personality, depression or anxiety played a role as mediators for the relationship between gender and EE. Personality was the only significant mediator. The initial direct path (gender-EE, controlled for BMI) was β = –0.46 (p < 0.001), but dropped to β = –0.28 (p < 0.001) after adding personality as the indirect path β = –0.18 (95% CI –0.29 to –0.09). The indirect path explained 39% (0.18/0.46) of the total variance in EE (fig. 1).

Taken together, the findings indicated a stronger impact of personality on dysfunctional eating compared with anxiety and depression.

Discussion

We hypothesized that female morbidly obese patients accepted for bariatric surgery had a higher level of emotional eating than men and, secondly, that personality traits, anxiety and depression partly explained the variation in eating behaviours.

Our major finding was that personality traits were more firmly related to dysfunctional eating patterns than mood disorder symptoms. Anxiety and depression were weakly related to such patterns when personality traits were controlled for. Neuroticism and conscientiousness were the prime personality traits involved in EE and UE/CR and could be ascribed a vulnerable and protective role, respectively. Moreover, EE occurred more often in female patients as previously reported [24, 25, 30]. We also found that neuroticism partially mediated this relationship, thus indicating a putative mechanism.

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The Role of Personality in Gender Differences in Emotional Eating

As reported by others [38, 39], women displayed more neuroticism and emotional eating. However, our results extended these findings by indicating that the gender-related differences in EE were partially explained by neuroticism (explaining almost half of the total variance), but neither by anxiety nor depression. The mediating role of neuroticism may indicate that this trait is a key factor to modify dysfunctional eating patterns of female bariatric surgery patients. This stands in contrast to Elfhag and Morey [24] who found negligible gender differences in the correlations between dysfunctional eating patterns and personality traits, such as neuroticism. We suspect that the contradictive results may be due to the use of standardized T-scores adjusting for gender differences in the study of Elfhag and Morey [24], whereas our analyses are based on unadjusted raw scores which do not mask naturally occurring gender-specific personality differences. The generally larger effect sizes for the gender differences in the unadjusted raw scores compared with the adjusted standardized T-scores make this interpretation plausible.

Symptoms, Personality and Emotional Eating

Our findings concur with previous studies [1, 24, 25] supporting the role of personality traits in disordered eating patterns. As personality traits are quite stable over time [19], eating behaviours are persistently influenced by these traits in contrast to the fluctuating influence of more time-limited psychological conditions like mood disorders. Hence, personality traits, like neuroticism, may be clinically important to address in addition to mood disorders as such traits are part of the psychological makeup of an individual that always exert a potential negative effect. A neurotic person is continuously exposed to emotional turmoil elicited by the liability to react to negative life events. Improved self-regulation of inherent neurotic traits may facilitate the achievement of weight maintenance goals by reducing the tendency to use dysregulated and dysfunctional eating patterns to cope with stress and daily hassles that may otherwise elicit anxiety or depression.

Notably, the neuroticism facet 'impulsiveness' contributed to explain a variation in all kinds of dysfunctional eating patterns. This finding concurs with a recent review article [40], which indicates that obese patients with or without BED display dysregulated eating behaviours due to the tendency of more spontaneous and 'impulse-driven' food intake. One possible implication of these findings is that this personality facet might be a 'key factor' in many different types of dysfunctional eating patterns. This personality trait may thus act as a vulnerability factor in an obesogenic society with a high exposure to energy-dense food and beverages, increasing the liability to develop dysfunctional eating patterns.

The personality trait conscientiousness is manifested by characteristic behaviours such as being organized and having a mental focus on order, control and systematics. High-scoring individuals may therefore stand out as rigid and inefficient if they lack the ability to flexibly adjust their behaviour according to changing situational demands. In the present study, though, the highly conscientious bariatric surgery patients seemed more able to take control over eating. In this respect, conscientiousness may protect against UE and CR and dispose for a higher ability to resist weight gain.

Strengths and Limitations

Strengths of the present study include the use of a web-based data collection method in a structured hospital setting. This may have reduced loss of variance and the need for imputation due to scattered missing data. Important limitations were the failure to include a diagnostic or self-report measure of BED as well as the cross-sectional design. The latter precludes any causal interpretations and tempers the conclusion that neuroticism may be an intermediate link between gender and emotional eating. Longitudinal data from this research project

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are underway that may more validly examine the direct and indirect causal role of personality factors, anxiety and depression for patients' ability to maintain an adequate post-surgery target weight.

Conclusions

The present study confirms previous findings [24–27]. Despite being limited by the cross-sectional design, it provides provisional new insights into how personality traits and gender may account for individual differences in emotional eating behaviour among patients admitted for bariatric surgery. Secondly, the study highlights that the interplay between gender, psychological health (i.e., anxiety and depression) and personality constructs (i.e., neuroticism and conscientiousness) are clinically relevant topics for future research.

Disclosure Statement

No conflicts of interest reported.

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Clinical Study

Effectiveness of a Cognitive Behavioral Therapy for Dysfunctional Eating among Patients Admitted for Bariatric Surgery: A Randomized Controlled Trial

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Objective. To examine whether cognitive behavioral therapy (CBT) alleviates dysfunctional eating (DE) patterns and symptoms of anxiety and depression in morbidly obese patients planned for bariatric surgery. *Design and Methods.* A total of 98 (68 females) patients with a mean (SD) age of 43 (10) years and BMI 43.5 (4.9) kg/m^2 were randomly assigned to a CBT-group or a control group receiving usual care (i.e., nutritional support and education). The CBT-group received ten weekly intervention sessions. DE, anxiety, and depression were assessed by the TFEQ R-21 and HADS, respectively. *Results.* Compared with controls, the CBT-patients showed significantly less DE, affective symptoms, and a larger weight loss at follow-up. The effect sizes were large (DE-cognitive restraint, $g = -.92$, $P \leq .001$; DE-uncontrolled eating, $g = -.90$, $P \leq .001$), moderate (HADS-depression, $g = -.73$, $P \leq .001$; DE-emotional eating, $g = -.67$, $P \leq .001$; HADS-anxiety, $g = -.62$, $P = .003$), and low (BMI, $g = -.24$, $P = .004$). *Conclusion.* This study supports the use of CBT in helping patients preparing for bariatric surgery to reduce DE and to improve mental health. This clinical trial is registered with NCT01403558.

1. Introduction

Bariatric surgery may result in significant weight loss, however with large individual differences [1, 2]. In patients eligible for bariatric surgery (BS), dysfunctional eating (DE) has been found among 10–25% of obese patients considered for or completing bariatric surgery [3, 4], and DE has been reported both prior [3–6] to and after BS [7–10]. DE can be operationalized as exerting rigid control, or loss of control over eating, or eating for emotional reasons rather than hunger or appetite. DE, in particular emotionally regulated eating, may be negatively reinforced if used to alleviate negative mood or feelings of stress [11].

DE is associated with overconsumption of energy dense food [12–15], which may impair sustained weight loss post-surgically [7, 11, 16–18]. Conversely, psychological treatments which target DE may increase the possibility of sustained weight loss following BS.

In addition to DE, patients with morbid obesity may suffer from symptoms of anxiety and depression. The prevalence of any mood disorder is about 16% and 24%, respectively [19]. Theoretically, improving affective symptoms might improve control over eating as there are fewer negative affects that one needs food to regulate. Moreover, alleviations in depression may facilitate experiences of self-efficacy and hence the motivation to implement the necessary behavioral changes in terms of adhering to dietary recommendations [20]. Both disorders may be effectively treated by cognitive behavioral therapy (CBT) [21].

To our knowledge, no previous controlled studies have tested the efficacy of a CBT-intervention aimed at reducing DE in obese patients selected to BS. However, several sources of knowledge indicate that such an intervention could be feasible. The convincing body of knowledge from controlled trials has established CBT as the treatment of choice for the spectrum of eating disorders according to diagnoses and

Table 1: Overview of the 10-week CBT-intervention.

Sessions	Session content
Session 1 *(at the center) both groups*	(i) Establishing rapport with the patient in order to facilitate a good therapeutic working alliance. (ii) Providing information about the interventions to all patients. (iii) Conducting the baseline measurements and performing the randomization and informing the patients about their allocated group.
Session 2 *(at the center)*	(i) Introduction to the underlying principles of the therapy (working transparently, collaboratively, being time-limited, and using a manual). (ii) Informing the patient about CBT and the treatment plans in the study. (iii) Psychoeducation focusing on the relationships between eating behaviors, cognitive and behavioral patterns, affect-regulation, and obesity, thus introducing the patients for the CBT model. (iv) Introducing and explaining home-work sheets for sessions 3 and 4.
Sessions 3 + 4 *(by telephone calls)*	(i) Reviewing the patient's home-work sheets. (ii) Recognizing and addressing dysfunctional eating behaviors. (iii) Working with the patient's behavioral eating patterns (what triggers eating), and the associated cognitions and emotions. (iv) Providing the patients' means to assess their own perception about recognizing improvement in dysfunctional cognitions and eating behaviors.
Session 5 *(at the center)*	(i) Coping with situational "triggers" that may lead to dysfunctional cognitive and eating behavioral patterns. (ii) Working with the patient's cognitive and behavioral eating patterns ("triggers," cognition, emotion, and eating behavior). (iii) Introducing and explaining home-work sheets for sessions 6 & 7.
Session 6 & 7 (*by telephone calls)*	(i) Reviewing the patient's home-work sheets. (ii) Continuing the intervention techniques. (iii) Reinforcing positive changes in eating behaviors.
Session 8 *(at the center)*	(i) Continuation or refining intervention techniques (as session 5) by guiding the patient in avoiding situational "triggers" and making a plan for practicing new eating behaviors. (ii) Introducing and explaining home-work sheets for sessions 9 & 10.
Session 9 & 10 (*by telephone calls)*	(i) Reviewing the patient's home-work sheets. (ii) Continuation or refining intervention techniques.
Session 11 *(at the center)*	(i) Relapse prevention. (ii) Ending of treatment and helping the patient to maintain positive changes.

clinical severity [22] including binge eating disorder (BED) [21]. DE may be considered as a milder variant of BED. Hence, a treatment working for the severe variant should logically also work for the milder one. Other sources of knowledge come from a case study of a patient admitted to BS [23] as well as from uncontrolled pre-post studies of larger series of patients, indicating that CBT might be an appropriate approach [21, 24].

Using a randomized controlled design, the purpose of this study was to examine the efficacy of a CBT-intervention in improving DE as well as affective symptoms. We hypothesized that the intervention would be superior to usual care, particularly with respect to reducing emotional and uncontrolled eating and increasing cognitive restraint of eating.

2. Methods

2.1. Participants. A total of 102 eligible (69 females and 33 males) consecutive morbidly obese patients admitted for bariatric surgery agreed to participate. All patients participated based on informed consent.

2.2. Study Design. This randomized controlled trial (http://clinicaltrials.gov/ct2/show/NCT01403558) used a mixed design: one between-group factor (intervention versus usual care) and one within-group factor (pre- and postmeasures). The time-interval between pre- and postmeasurements was 10 weeks.

2.3. Randomization. A block randomization procedure (http://www.randomizer.org) was employed (with blocks of 4) to ensure balance between the groups. Two research assistants at the treatment center, with no affiliation to the study, had access to the key to the randomization file. After having read and signed the informed consent letter and completed the baseline measurements, the patients as well as the first author were informed about the allocated treatment arm. The allocation ratio was 1 : 1.

2.4. Procedures before Surgery (All Patients). During the four months prior to surgery, patients in both treatment arms were offered up to three consultations from either a medical doctor, a dietician, a nurse, or a physiotherapist. These consultations were voluntary and were based on the patients' individual needs. Here the patients received educational materials concerning nutritional recommendations, detailed information about the mandatory low calorie diet the last three weeks

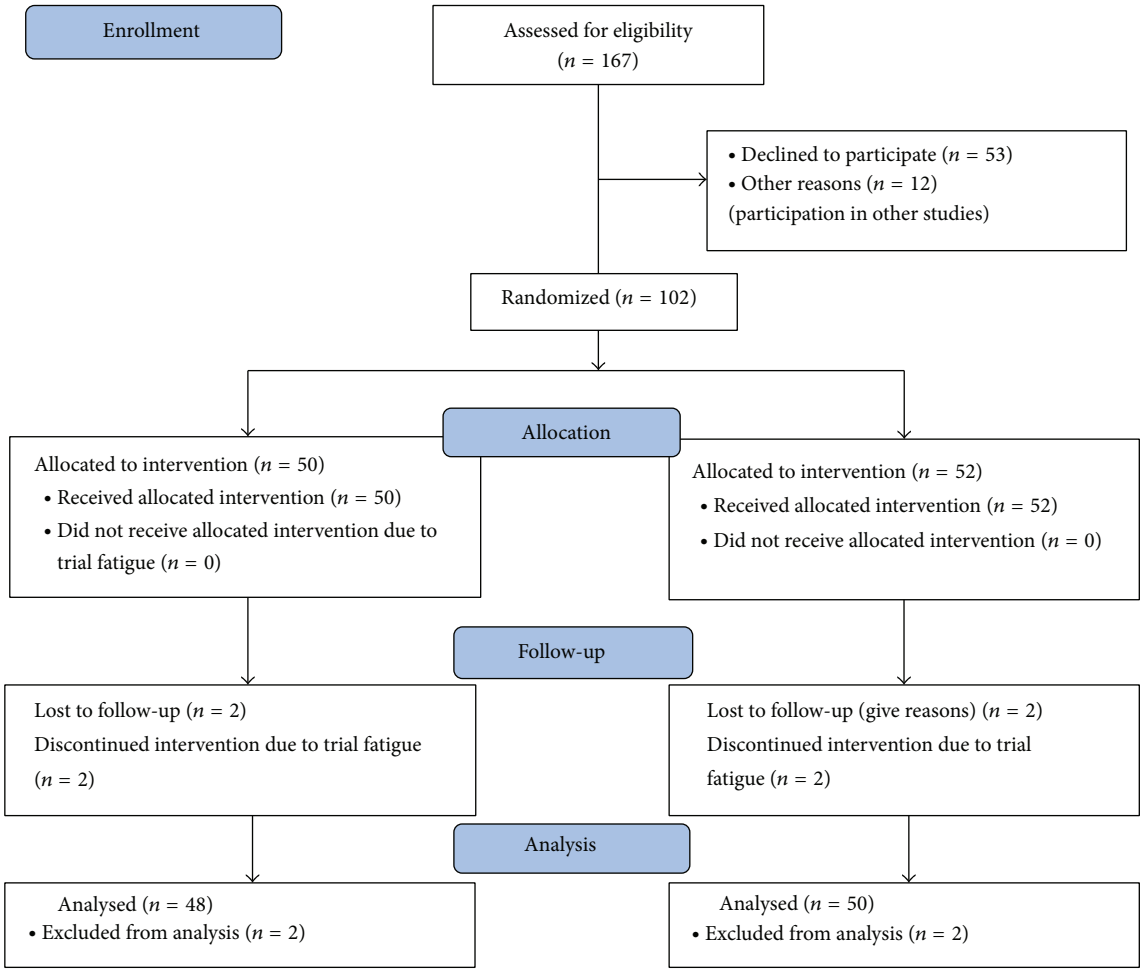

Figure 1: Participant flow (morbidly obese patients admitted for bariatric surgery).

before surgery, and guidance about recommended physical activity level and intensity.

2.5. Intervention Group. The patients in the intervention group received ten sessions based on theoretical principles from CBT, that is, learning to recognize triggers of DE, identifying associated cognitions and emotions, initiating plans for change, and use of home-work task in between the sessions. Sessions 1-2 included strategies to enhance intrinsic motivation and addressed resistance to change [25]. Sessions 2–11 were based on CBT-principles. Table 1 provides an overview of the contents of all sessions. Five sessions were carried out at the treatment center, and the remaining six as scheduled telephone calls.

2.6. Measurements and Outcomes. Demographic and clinical variables comprised age, gender, educational level, employment, and BMI.

2.7. Dysfunctional Eating (DE). The primary outcome measures were changes in DE as measured by the Three-Factor Eating Questionnaire (TFEQ R-21) which has been validated for use in obese individuals [26, 27]. It consists of 21 items comprising three subscales: "emotional eating" (EE; 6 items; Cronbach's $\alpha = .92$), "uncontrolled eating" (UE; 9 items; $\alpha = .73$), and "cognitive restraint of eating" (CR; 6 items, $\alpha = .84$). According to the manual, the three subscales were transformed to a 0–100 scale to become comparable [26]. Higher scores indicated more severe dysfunction. The reliability of the subscales in the present study was comparable to previous reports [26].

2.8. Affective Symptoms. Secondary outcome measures were symptoms of anxiety and depression, measured by the Hospital Anxiety and Depression Scale (HADS) [28]. HADS is a self-report measure of nonvegetative affective symptoms [28, 29] where seven items assess depression (HADS-D) and seven items measure anxiety (HADS-A), respectively. Items are scored 0–3 yielding a range of 0–21 within each subscale. A cut-off ≥8 is used in Norway to indicate a clinically probable impairment due to depression or anxiety [30]. Cronbach's alphas for HADS-A and HADS-D were .84 and .78, respectively.

The procedures were initiated after the study had been approved by the Regional Committee for Medical and Health Research Ethics (2010/2071a).

2.9. Sample Size . Based on clinical experience, reductions in the emotional and uncontrolled eating scores of 15% or more were considered to be clinically meaningful. A conservative estimate was that no patients in the control group, and at least 30% in the intervention group, would achieve this treatment goal. Given this difference in treatment effect, a 90% statistical power, a significance level of 5%, and a dropout rate of 40%, a minimum sample size of 80 patients was required. To allow for a 20% withdrawal rate, we included 102 patients in the current study. The statistical power was excellent for all analyses (>.99).

2.10. Statistical Methods. Data were analyzed by the Statistical Package of the Social Science (SPSS) for Windows, version 17 (SPSS, Chicago, IL, USA).

The intervention effects were examined by analysis of covariance (ANCOVA), comparing the two posttest group mean scores adjusted for baseline scores. Effect sizes were reported as Hedges' g indicating the differences between the groups in number of standard deviations. Effect sizes of 0.20, 0.50, and 0.8 were regarded as small, moderate, and large [31].

3. Results

3.1. Recruitment and Participant Flow. Hundred and two patients agreed to participate; four patients were lost to follow-up, leaving data from 98 patients for analysis (Figure 1). A completers-only analysis was conducted at follow-up as attrition was minor.

3.2. Baseline Data. Clinical baseline data are presented in Table 2 showing that most participants (82%) had finished upper secondary school (≥12th grade), 54% were employed, and 40% received disability pension or a temporary pension while assessing work ability.

The prevalences of clinically relevant symptoms of anxiety and depression (HADS ≥ 8) were 41% and 25%, respectively.

3.3. Effect of the Intervention. The patients in the CBT-group had significant improvements in DE, anxiety, and depression compared with the control group patients. A significant reduction in BMI was also observed.

The intervention effects are presented in Figures 2 and 3 showing postinterventional scores for eating behaviors and affective symptoms by treatment. The between-group effect sizes for the improvements varied from high (uncontrolled eating ($g = -.90, P \leq .001$), cognitive restraint ($g = .92, P \leq .001$)) to moderate (emotional eating ($g = -.67, P \leq .001$), anxiety ($g = -.62, P \leq .001$), depression ($g = -.73, P = \leq .001$)) and low (BMI ($g = -.24, P = .004$)).

Adjusted between-group differences at follow-up for EE, UE, and CR were −19 (95% CI, −26 to −12), −19 (95% CI, −25 to −14), and 20 (95% CI, −28 to −13), respectively, all $P \leq .001$. For anxiety and depression the adjusted between-group differences were −2.5 (95% CI, −3.5 to −1.4) and −2.8 (95% CI, −3.9 to −1.6), respectively, both $P \leq .001$. Concerning BMI and body weight, the adjusted between-group differences were −1.1 kg/m^2 (95% CI, −1.8 to −.35, $P = .004$) and −3 kg (95% CI, −5.1 to −.84, $P = .004$).

Table 2: Baseline demographics, eating behaviors, anxiety, and depression among 102 patients admitted for bariatric surgery by treatment arm.

	Total ($n = 102$)	Intervention ($n = 50$)	Controls ($n = 52$)
BMI (kg/m^2)	43.5 (4.9)	43.6 (5.1)	43.5 (4.7)
Weight (kg)	128.0 (19.1)	129.1 (18.0)	126.9 (20.1)
Gender			
Female	69	31	38
Male	33	19	14
Age (years)	42.6 (9.8)	44.1 (9.8)	41.2 (9.6)
Educational level			
<12th grade	84 (82.4)	41 (82.0)	43 (82.7)
High school/college degree	18 (17.6)	9 (18.0)	9 (17.3)
Employment			
Employed	55 (53.9)	26 (52.0)	29 (55.8)
Unemployed	6 (5.9)	3 (6.0)	3 (5.8)
Temporary pension	21 (20.6)	11 (22.0)	10 (19.2)
Disabled	20 (19.6)	10 (20.0)	10 (19.2)
Eating behaviors			
Emotional eating	52.4 (26.0)	53.4 (27.1)	51.4 (25.0)
Uncontrolled eating	49.0 (18.8)	50.5 (17.7)	47.4 (20.0)
Cognitive restraint	44.1 (20.5)	42.7 (19.7)	45.5 (21.2)
Affective symptoms			
Anxiety	6.7 (3.9)	7.0 (4.2)	6.5 (3.7)
Depression	5.1 (3.4)	5.5 (3.7)	4.7 (3.0)

Number (%) or mean (SD). The Three-Factor Eating Questionnaire (TFEQ R-21) was used to measure the three domains of eating behaviors, and the Hospital Anxiety and Depression Scale (HADS) was used to measure anxiety and depression.

4. Discussion

This study contributes to the literature as being the first randomized controlled trial of a CBT-intervention to treat dysfunctional eating behaviors among severely obese patients scheduled for bariatric surgery. It demonstrated that patients in the CBT-group showed a strong reduction in DE and a moderate alleviation of anxiety and depression following the 10-week intervention compared to the control group. In addition, the CBT-group lost about 3 kg body weight.

To our knowledge, no previous controlled study has assessed a CBT program in the treatment of DE. Nevertheless, DE is closely linked to BED both cognitively and behaviorally in terms of eating patterns and the use of food to regulate negative mood. Although BED was not assessed in the present study, a comparison with previous BED-studies may

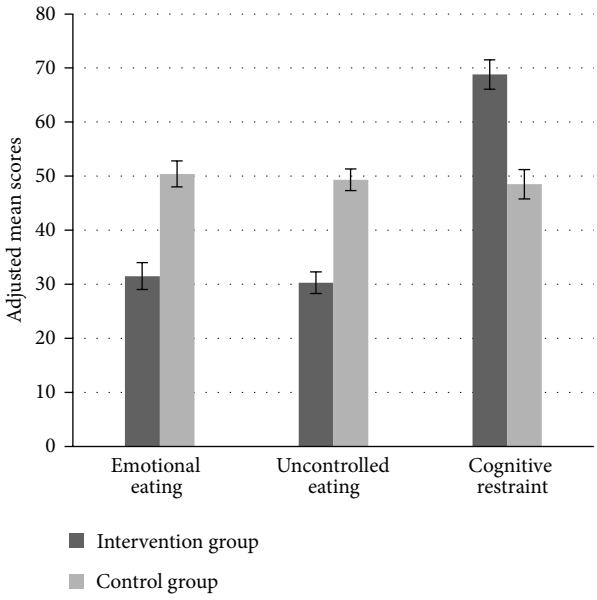

Figure 2: Postintervention (10-week) scores for eating behaviors by treatment arm. Data expressed as adjusted mean scores. Error bars expressed as standard errors of the mean. The Three-Factor Eating Questionnaire (TFEQ R-21) was used to measure the three domains of eating behaviors.

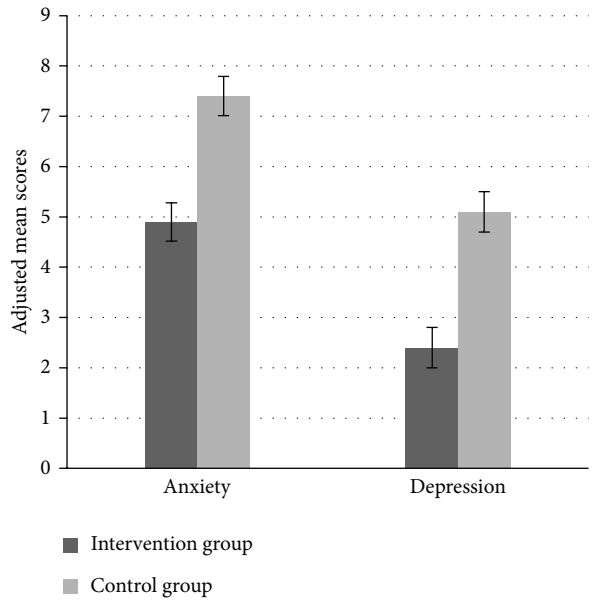

Figure 3: Postintervention (10-week) scores for anxiety and depression by treatment arm. Data expressed as adjusted mean scores. Error bars expressed as standard errors of the mean. The Hospital Anxiety and Depression Scale (HADS) was used to measure anxiety and depression.

be warranted. Hence, previous BED-studies [21, 24] support our findings in the sense that a CBT-intervention effectively reduced binge eating symptoms and associated cognitions, as well as increasing postoperative weight loss. On the other hand, comparisons across studies may be difficult due to incomplete treatment descriptions [21] and divergent study designs [24].

As DE often includes overeating in relation to negative mood states or a tendency to lose control over eating, it includes cognitive, emotional, and behavioral elements. It is plausible that a targeted CBT-program could have beneficially affected all these elements. As repeated measures following each session were not taken, it was impossible to discern which of the specific parts of the intervention produced the improvements. In addition, a nonspecific, independent effect of the therapist and the therapeutic alliance cannot be excluded.

The reduction of affective symptoms might be considered an adjuvant effect of the CBT-intervention. Although the intervention did not address symptoms of poor mental health specifically, it addressed how to detect and improve the tolerance of negative emotions triggering DE behaviors and associated dysfunctional cognitions. Moreover, as the intervention included home-sessions with practical tasks that were possible to accomplish for all patients, feelings of coping and mastery might partly explain the apparent antidepressive and anxiety reducing effect.

Strengths of the study are the randomized controlled treatment design and the low attrition rate. In addition, the recruitment of consecutive treatment seeking white morbidly obese patients preparing for bariatric surgery in a large tertiary care center suggests that our results may be generalizable to similar populations.

As the first trial addresses DE and the effect of CBT before bariatric surgery, our results need replication trials. Indeed, such replications will need to take this pioneer study's limitations into account. Notably, one needs to sort out common versus specific effects by including more than one therapist as well as possible treatment component effects. In addition, further studies should develop a control treatment condition in more detail and with a number of sessions equal to the CBT-condition. Furthermore, future studies should also collect additional data such as binge eating symptoms, which was not done in the current study.

This study shows the success of a 10-week CBT-intervention program in improving DE behaviors and affective symptoms in morbidly obese patients admitted for bariatric surgery. Future research should investigate whether these proximal effects are sustained and whether presurgical improvement in DE behaviors and affective symptoms do provide an additive benefit to bariatric surgery in terms of a stabilization of weight loss.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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The impact of a pre-surgical cognitive behavioural therapy (CBT) on dysfunctional eating behaviours, affective symptoms and body weight one year after bariatric surgery. A randomised controlled trial

Original contribution

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Abstract

Objective

To examine whether a pre-surgical cognitive behavioural therapy (CBT) intervention exceeds usual care in the improvements of dysfunctional eating behaviours, mood, affective symptoms and body weight one year after bariatric surgery.

Design and methods

This is a 1-year follow up of a single centre parallel-group randomised controlled trial (http://clinicaltrials.gov/ct2/show/NCT01403558). A total of 80 (55 females) patients with mean age of 44 years (SD 10) were included. The intervention group received 10 weeks of CBT *prior* to bariatric surgery, and the control group received nutritional support and education. Both groups were assessed at baseline (T0), post CBT-intervention/pre-surgically (T1), and one year post-surgery (T2). Using a mixed modelling statistical approach, we examined if the CBT group improved more across time than the control group.

Results

Our hypothesis was not supported as both groups had comparable improvements in all outcomes except for anxiety symptoms. Body weight declined by 30.2% (37.3 kg) in the CBT group and by 31.2% (40.0 kg) in the control group from baseline to follow-up, $p = .82$. There were statistically significant reductions in anxiety and depression symptoms in the CBT group between T0 and T1 and between T1 and T2 for depression only. However, in the control group, the anxiety score did not change significantly. The CBT group showed an earlier onset of improvements in all eating behaviours and affective symptoms than the control group.

Conclusion

The 10-week pre-surgical CBT intervention showed beneficial effects pre-surgically, but the non-significant group differences post-surgically indicate a genuine effect of surgery.

Introduction

In patients with extreme obesity undergoing bariatric surgery, there is a high rate of dysfunctional eating behaviours (DE) (i.e. emotional eating, uncontrolled eating and cognitive restraint) both prior to (1-5) and after surgery (5-7). Gastric bypass, a common bariatric procedure, promotes weight loss mainly by reducing appetite, thereby helping the patient to change eating behaviours (8, 9). Between 20 and 30% of patients undergoing gastric bypass regain weight around two years after surgery (10, 11), and the notable individual differences in the amount of weight loss (12, 13) may be partly accounted for by sustained DE (14). Data from the LABS-study (13) reporting 3 years change in weight show that the majority of patients reach their nadir weight one year after surgery. Additionally, five sub-groups with different patterns of weight loss were identified. These patterns showed a variability of weight changes starting at six months postoperatively. Cognitive restraint (15) and emotional eating (16) before surgery have been identified as predictors for postoperative body weight. Thus, while higher cognitive restraint may predict greater weight loss (15), increased emotional eating may predict suboptimal weight loss or weight regain (16).

Symptoms of mood and anxiety are also highly prevalent in this patient population (5, 17-19). Except for short-term improvements of mood and affective symptoms after surgery (17, 20), mood and anxiety *disorders* diagnosed preoperatively tend to remain unchanged long-term (17).

Psychological interventions may alleviate both these comorbidities and DE. Some studies (21, 22) show that such kind of pre-surgical interventions may reduce both psychological comorbidity and DE. In the studies of Ashton et al (21, 23), a brief cognitive behavioural therapy (CBT) intervention of four weeks reduced binge eating behaviours before surgery. Additionally, the patients who improved their eating behaviours lost significantly more weight one year after surgery than those who did not. Abiles et al (22) showed that a 12 week

group-based CBT intervention reduced psychological comorbidity both in patients with or without binge eating disorder (BED). In the second stage of the pre-surgical intervention, the patients were offered weekly follow-ups including dietary counselling and were recommended to follow a 1500 kcal diet. More than half of the patients had a pre-surgical weight loss of ≥ 10%.

Other studies have provided additional information on the effects of post-surgical interventions. The results from a pilot study by Sarwer et al (24) indicate that dietary counselling may have a short-term (6 months) effect on weight loss and eating behaviours In contrast, a controlled study (25) did not lend support to the effect of unspecified psychological support, yet this study restricted the outcome variable to weight reduction only.

Less is known though, about the longer-term impact of pre- or post-surgical behavioural interventions on DE, psychological comorbidity and weight loss. One systematic review and metaanalysis of post-surgical behavioural interventions (26) concluded that greater weight loss may be achieved. However, the validity of these findings is restricted by uncontrolled study designs, measurements, and contents of interventions.

In the evaluation of the impact of such interventions, the temporal aspect of post-surgical follow-up is of importance. One year after surgery may be considered "short-term", as significant differences in outcomes of body weight and DE may be seen at later stages.

Using an RCT-design we recently demonstrated (27) that compared with usual care, a 10 week pre-surgical cognitive behavioural treatment (CBT) significantly improved DE as well as affective symptoms immediately before the time of surgery. Whether these beneficial effects are sustained, or whether the pre-surgical intervention may give any *additional* positive effects beyond surgery remain unknown.

In the present study we anticipated that the outcome variables change differently across time depending on the consultations offered (CBT vs usual care). The hypothesis was

that a pre-surgical CBT intervention would perform better than usual care in reducing DE and body weight as well as mood and anxiety symptoms at a 1-year follow-up after surgery.

Methods

Trial design and setting

This is the second part (1-year follow up) of a single centre parallel-group randomised controlled trial (http://clinicaltrials.gov/ct2/show/NCT01403558) conducted at a tertiary care centre in Norway between September 2011 and December 2013 (2, 27).

The study design was mixed factorial. The within factor had three levels (a repeated time factor: T0, T1 and T2). All outcome variables were measured at baseline (T0), post CBT intervention/pre-surgically (T1), and one year post-surgery (T2). The between factor had two levels: the control (G0) and the intervention (G1) group. The two-way interaction (*Group* × *Time*) thus represented a test of the hypothesised between-group mean difference in the outcome variables over time.

Participants

All recruited participants were accepted for bariatric surgery. Those who agreed to take part in the study were included after providing written informed consent, both at inclusion and at one year post-operative follow-up. Unlike in North America, a pre-surgical psychological evaluation is not standard practise in Norway. Therefore, no patients were excluded from the study based on a psychological evaluation. Of note, all patients who were invited to participate in the study were already accepted for bariatric surgery.

Interventions

During the four months prior to surgery, patients in both treatment arms were offered up to three voluntary consultations from either a medical doctor, a dietician, a nurse or a physical therapist tailored to the patients' individual needs. The CBT-intervention has been described

in more detail elsewhere (28), but this 10-week treatment condition consisted of learning to recognise triggers of DE, i.e. identifying how automatic thoughts and dysfunctional cognitions, negative moods, and overeating are interrelated. Moreover, weekly home-work tasks were used to break the DE-patterns which are a common problem for patients suffering from extreme obesity. Thus, the main purpose of the intervention was to improve self-monitoring and self-regulation of eating behaviour. Some individual adjustments during the course of therapy were allowed to accommodate for the fact that some patients spent more time working on obtaining more regularity in eating, while others addressed cognitive negative self-talk in order to reduce emotionally triggered eating behaviour.

In the year following the surgery, all patients were invited to attend one group session with a clinical nutritionist and another with a physical therapist. The patients were additionally offered two individual consultations with a physician.

Covariates and outcomes

The demographic variables at T0 comprised age, gender, educational level, and employment status. Body weight and height were measured with light clothing and no shoes using a digital scale (Soehnle Professional 2755, http://www.soehnle.de/) and a wall mounted stadiometer (Seca 240, http://www.stadiometer.com/), respectively. Body weight was measured at all three time points.

The clinical variables were collected at all-time points through a web-based solution (http://fluidsurveys.com/ and https://metreno.com/) , and comprised the Three–Factor Eating Questionnaire (TFEQ R-21) (28, 29) measuring DE, and the Hospital Anxiety and Depression Scale (HADS) (30, 31) measuring symptoms of anxiety and depression, respectively.

The TFEQ R-21 has been validated for use in obese individual (28). It consists of 21 items comprising three subscales, i.e. “Emotional eating” (EE; 6 items; Cronbach’s α =.92), “uncontrolled eating” (UE; 9 items; α = .81) and “cognitive restraint of eating” (CR; 6 items,

α =.74). According to the manual, the three subscales were transformed to a 0-100 scale to become comparable (28). Higher scores indicate higher levels of dysfunction. The reliability of the subscales in the present study was comparable to previous reports (28).

The HADS (30) is a self-report measure of anxiety and non-vegetative affective symptoms (30, 31). Seven items assess depression (HADS-D) and seven items measure anxiety (HADS-A), respectively. Items are scored 0-3 yielding a range of 0-21 within each subscale. A cut-off ≥ 8 is used in Norway to indicate a clinically probable impairment due to depression or anxiety (32). In the current study, the Cronbach's alphas for HADS-A and HADS-D were .77 and .70, respectively.

Statistical methods

Continuous variables were described using means and standard deviations (SD), categorical data as counts and percentages. Crude differences between the groups were assessed with t-tests (continuous data) and chi-square tests (categorical data). Linear mixed model regression analyses were used in order to estimate both random and fixed effects. Time, group and their interaction represented fixed effects, while individual differences at baseline were accounted for by a random intercept parameter. A diagonal covariance structure was specified to accommodate for heterogeneous residual variances across time. Restricted maximum likelihood estimation was used to produce unbiased estimates of the model parameters. All overall effects were analysed using the F-tests. The results were presented as estimated means with 95% confidence intervals (CI). Least significant difference (LSD) post-hoc tests were used to compare selected means at given time points. Due to the exploratory nature of our study we did not use any correction for multiple testing. P-values < 0.05 were considered statistically significant and all tests were two-tailed. The software IBM SPSS version 17 (SPSS, Chicago, IL, USA) was used to conduct all analyses.

Sample size

The sample size was based on pre-estimations from the pre-surgical intervention. According to clinical experience, reductions in the emotional and uncontrolled eating scores of 15% or more are considered to be clinically meaningful. A conservative estimate was that no patients in the control group, and at least 30% in the intervention group, would achieve this treatment goal before surgery. Given this difference between the treatment groups, a 90% statistical power, a significance level of 5% and a dropout rate of 40%, a minimum sample size of 80 patients was required at baseline. To allow for a 20% withdrawal rate, we included 102 patients at baseline, and 83 patients completed all assessments one year after surgery (27).

Randomisation

A block randomisation procedure (http://www.randomizer.org) was employed (with blocks of 4) to ensure balance between the groups. Two research assistants at the treatment centre with no affiliation to the study, had access to the randomisation file. After having read and signed the informed consent letter and completed the baseline measurements, the patients as well as the first author were informed about the allocated treatment arm. The allocation ratio was 1:1 (27).

Results

Recruitment and participant flow

Eighty four patients accepted to participate at follow-up, but as four patients refused surgery the final sample included 80 patients (*Figure 1*). The majority of patients (69%) were female, and the mean (SD) BMI was at T0 43.7 (4.9) kg/m^2. There were no statistically significant differences between the groups regarding gender, BMI or level of education (*Table*

1). Eighty six percent in the intervention group and 82% in the control group underwent Roux-en-Y gastric bypass. The remaining patients underwent sleeve gastrectomy.

Analyses of treatment effects

Neither of the groups had any significant change in body weight from T0 to T1 (data not shown). In contrast, body weight declined significantly after surgery (T1-T2) in both the CBT (M_{diff} = -37.3 (CI: -40.4-34.2)), -37.3 kg and control group (M_{diff} = -40.0 (CI: -43.3-36.7)) – 40 kg, both *p*'s < .001). To summarize, in the CBT and the control group, the body weight was reduced by 30.9% and 31.2% respectively from baseline to 1-year follow up (p = .816).

The unadjusted means for all other outcome variables are presented in *Table* 2. The differential change in eating behaviours and affective symptoms across time and groups are presented in *Figures* 2 and *3*. The main effect of *time* was significant (p < 0.001) indicating an improvement in both groups across time (from T0 to T2). The interaction *group* × *time* was statistically significant for all outcome variables (all *p*'s < 0.01), except BMI, thus indicating that the improvement occurred at different time points in the two groups. Follow up post-hoc tests were therefore needed to pin-point which *group* differences were present at T1 and T2. There were no statistically significant differences in changes in body weight and DE between the patients who underwent RYGB and GS (all *p*-values above).

Post-hoc tests

Dysfunctional eating

Pairwise post-hoc tests were used to examine mean differences (M_{diff}) between time (T0-T1-T2) and groups (CBT vs control). They revealed significant improvements in EE, UE and CR in the CBT group between T0 and T1 (M_{diff} = -22.62, M_{diff} = -19.49 and M_{diff} = 25.93, respectively, all *p*'s < .001). Further improvements between T1 and T2 were evident for EE and UE only (M_{diff} = -8.48, p = .02; M_{diff} = -11.90, p < .001, respectively).

The improvement in the control group was only evident between T1 and T2 for EE and UE (M_{diff} = -19.97, $p < .001$; M_{diff} = -24.30, $p < .001$), and between T0 and T2 (Reviewer #1, comment #1) for CR (M_{diff} = 9.50, $p = .02$).

Group differences: The post-hoc tests revealed significant group differences favouring CBT only at T1 for EE, UE and CR (M_{diff} = -14.61, $p < .01$; M_{diff} = -15.60, $p < .01$; M_{diff} = -18.96, $p < .001$, respectively), but not at T2.

Anxiety and depression

There was a significant reduction in anxiety and depression symptoms in the CBT group between T0 and T1 (M_{diff} = -1.88 and M_{diff} = -2.69, respectively, both p's < .001), and between T1 and T2 for depression only (M_{diff} = -1.04, $p = .03$). In the control group, the anxiety score did not change significantly, whereas depression scores went down significantly between T1 and T2 (M_{diff} = -2.68, $p < .001$).

Group differences: No significant group differences were observed for anxiety, whereas a significant group difference favouring CBT was evident at T1 (M_{diff} = -1.71, $p < .01$) for depression, but not at T2.

Discussion

Our hypothesis that CBT would improve dysfunctional eating patterns, mood and anxiety symptoms one year after surgery was not supported. Apart from a comparable weight loss, the two groups revealed different patterns of changes in all eating behaviours and affective symptoms during the follow-up time.

Treatment effects comparisons with other studies are difficult as no other studies have examined the impact of a CBT intervention versus a control group over time. Our findings do however concur with a pilot-study of Sarwer et al (24), which showed that initial short-term

effects on weight loss and eating behaviours after post-surgical dietary counselling waned off after the first four months. As shown in *Figures 2* and *3*, the patterns of change suggest that the benefit of the CBT intervention exceeded usual care *before* surgery. However, at the one-year follow-up the CBT treatment did not have any *additional* effects beyond the surgery on eating behaviours, affective symptoms or body weight. The CBT intervention thus exceeded usual care in terms of *an earlier onset* of reduction of DE and affective symptoms, which represents a beneficial improvement in mental health in terms of facilitating functional coping with daily stress as well as control over eating. These findings are in line with Abiles et al (22) indicating that a pre-surgical CBT intervention may improve psychological comorbidity. Indicated by the present, as well as by previous research (17, 18), depressive symptoms usually drops more than symptoms of anxiety following bariatric surgery.

With respect to the course, our findings concur with studies (8, 9, 12, 18) showing more positive enduring changes in DE, affective symptoms and body weight, and that the surgery itself had a comprehensive effect on DE by limiting the possibility of consuming large amounts of food. However, the effects of bariatric surgery on DE and affective symptoms seem to decrease over time (18). It may be the case that the CBT intervention was not potent enough to maintain further improvements that exceeded usual care one year after bariatric surgery. The clinical value of the treatment may thus be limited, or at least indicating a need to address issues related to maintenance of effects more carefully in psychological treatments. On the other hand, as bariatric surgery had a comprehensive effect on all these outcomes, our expectations of CBT being superior to usual care one year after surgery might have been too optimistic. According to the findings of Courcoulas et al (13), the majority of the patients reached their maximum weight loss one year postoperatively, a period that has been labelled "the honeymoon phase". As the biological effects of the surgery have not yet started to wane, and the majority of the patients are at their nadir weight, it may be difficult to identify any

additional psychological effects at this time. On the other hand, the LABS-study (13) showed that the variability in the weight loss trajectories do increase from about six months postoperatively. Considering the profound biological effects that surgery provides during the first year, a one year follow-up gap may be considered rather short-term. Hence, the CBT-intervention may still exert an influence, particularly when the problems with maintaining the weight reduction start.

A minor objection may relate to the lack of statistical power as the sample size was calculated to detect pre-surgical (T1) treatment effects. As Table 2 reports, there were differences between the groups in the hypothesized directions at T2 that a larger sample size might have deemed significant. These differences would on the other hand probably have minor clinical importance.

Our study had considerable strengths, notably the randomised controlled design and the unbiased selection of patients due to the consecutive recruitment procedure. Moreover, CBT covers a number of different approaches which differ in their emphasis on cognitive versus behavioural principles and techniques. In contrast to previous studies, we have (27) designed a treatment manual with a detailed outline of the intervention, which makes replications more accurate. A limitation may be noted for the one year follow-up time. Accordingly, a later follow-up assessment might clarify whether the effects on DE and body weight would become more prominent when the biological effects of the surgery wane.

In case of a clinical replication, common versus specific psychotherapy effects need to be sorted out by including more than one therapist as well as including measures of treatment alliance and therapist competence. Furthermore, future studies should consider including a control group having an equal number of attendance sessions, hence ruling out alternative explanations related to differences in dose-response and placebo. Future studies should also

include a measure of binge eating symptoms, and address the issue of treatment potency in terms of content and time of delivery.

Conclusions

Our results confirmed that eating behaviours, affective symptoms and body weight improve the first year after bariatric surgery. The pre-surgical CBT initiated a faster improvement in dysfunctional eating and affective symptoms, but it was no longer superior to usual care following surgery.

Other information

Registration

http://clinicaltrials.gov/show/NCT01403558

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Conflict of interest:

None disclosed

Figure legends:

Figure 1: Participant flow: Patients with extreme obesity assessed for eligibility, randomisation and follow-up.

Figure 2: Changes in the three facets of dysfunctional eating behaviours (DE) by treatment arm measured by the TFEQ R-21: Emotional eating, Uncontrolled eating and Cognitive restraint. B = Baseline (4 months before surgery), post-CBT = after CBT-intervention and before surgery, 1y = 1 year after surgery. Values presented as estimated means with 95 % CI from linear mixed-effects models. High scores represent more emotional eating, uncontrolled eating or cognitive restraint.

Figure 3: Changes in symptoms of anxiety and depression by treatment arm measured by the HADS. B = Baseline (4 months prior to surgery), post-CBT = after CBT-intervention and before surgery, 1y = 1 year after surgery. Values presented as estimated means with 95% CI from linear mixed-effects models. High scores represent more anxiety or depression.

***Table 1**. Baseline demographics among 80 patients who underwent bariatric surgery by treatment arm.*

	Total (n = 80)	**Intervention** (n = 42)	**Controls** (n = 38)	***p*-values**
BMI (kg/m^2)	43.7 (4.9)	43.6 (5.1)	43.5 (4.7)	.742
Weight (kg)	128.7 (18.1)	129.5 (17.2)	127.7 (19.2)	.661
Gender				
Female	55	27	28	.369
Male	25	15	10	
Age (years)	44.3 (10)	44.1 (9.8)	41.2 (9.6)	.152
Educational level				
$< 12^{th}$ grade	66 (82.5)	34 (81.0)	32 (84.2)	.705
High school/college degree	14 (17.5)	8 (19)	6 (15.8)	
Employment				
Employed	45 (56.3)	22 (52.4)	23 (60.5)	
Unemployed	4 (5)	3 (67.1)	1 (2.6)	.671
Temporary pension	17 (21.3)	9(21.4)	8 (21.1)	
Disabled	14 (17.5)	8 (19.0)	6 (15.8)	

Data presented as observed mean (SD) or number (%).

Table 2: Treatment effects across time by treatment arm.

Outcomes	**EE**		**UE**		**CR**		**Anxiety**		**Depression**	
	CBT	Control	CBT	Control	CBT	Control	CBT	Control	CBT	Control
	M 95% CI	M 95% CI	M 95% CI	M 95% CI	M 95% CI	M 95% CI	M 95% CI	M 95% CI	M 95% CI	M 95% CI
Baseline	53.7 46.2-61.2	48.1 40.2-56.0	49.6 44.6-54.7	45.5 40.2-50.8	43.3 37.5-49.0	47.8 41.7-53.8	6.8 5.7-7.9	6.3 5.2-7.4	5.3 4.5-6.2	4.2 3.3-5.1
Post-intervention	31.1 23.6-38.6	45.7 37.7-53.7	30.2 25.1-35.2	45.8 40.4-51.2	69.2 63.4-75.0	50.2 44.0-56.4	5.0 3.9-6.0	6.4 5.3-7.6	2.6 1.8-3.5	4.4 3.4-5.3
Follow-up	22.6 15.1-30.2	25.7 17.8-33.6	18.3 13.2-23.3	21.4 16.2-26.7	62.3 56.5-68.2	57.3 51.2-63.4	4.4 3.4-5.5	5.7 4.6-6.8	1.6 0.7-2.5	1.7 0.8-2.6

Data presented as unadjusted means with confidential intervals (CI). EE (emotional eating), UE (uncontrolled eating) and CR (cognitive restraint) were measured by the Three-Factor Eating Questionnaire (TFEQ R-21), and symptoms of anxiety and depression were measured by the Hospital Anxiety and Depression Scale (HADS).

Figure 1

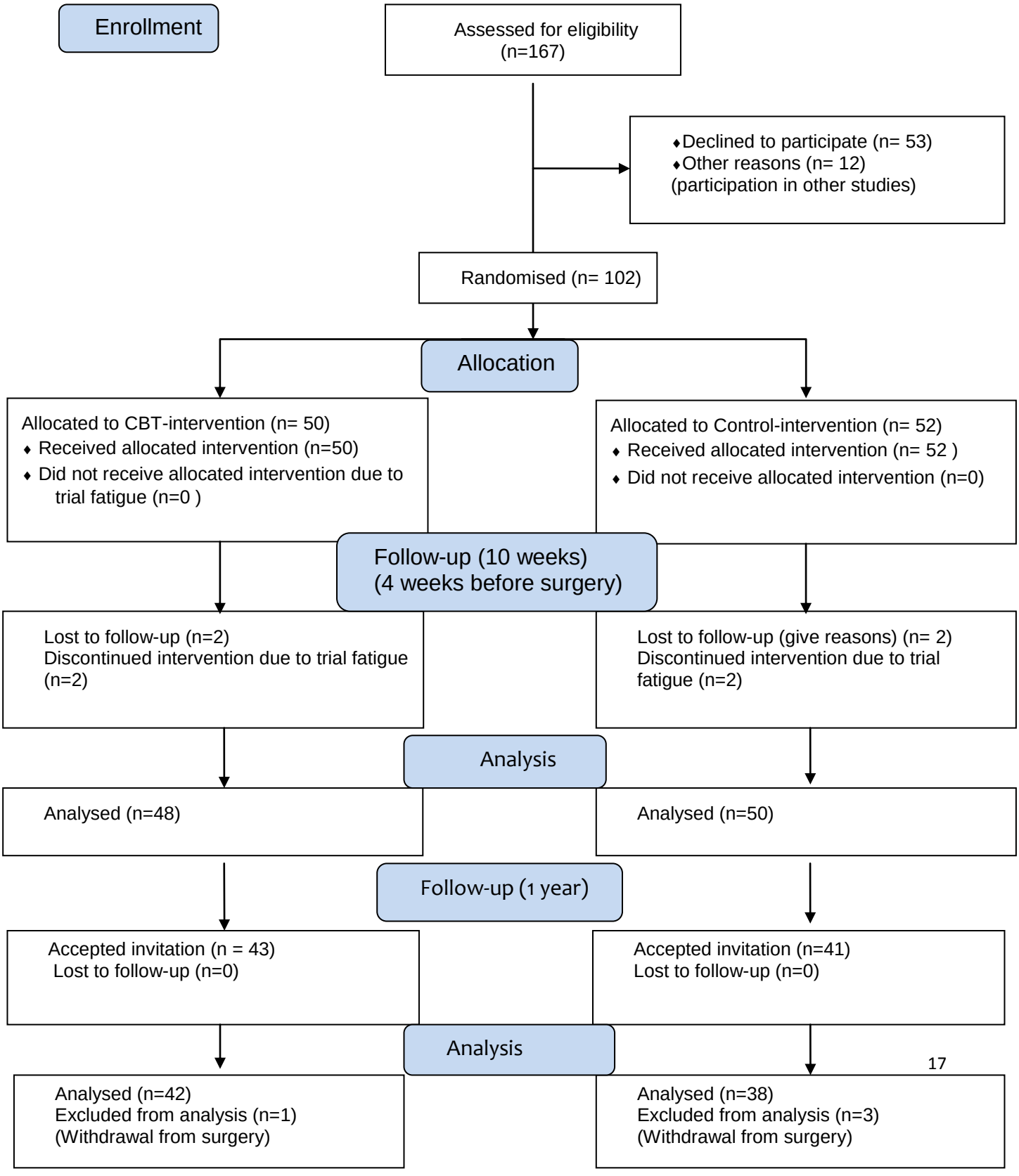

Enrollment
Assessed for eligibility (n=167)
♦Declined to participate (n= 53)
♦Other reasons (n= 12)
(participation in other studies)
Randomised (n= 102)
Allocation
Allocated to CBT-intervention (n= 50)
♦ Received allocated intervention (n=50)
♦ Did not receive allocated intervention due to trial fatigue (n=0)
Allocated to Control-intervention (n= 52)
♦ Received allocated intervention (n= 52)
♦ Did not receive allocated intervention (n=0)
Follow-up (10 weeks)
(4 weeks before surgery)
Lost to follow-up (n=2)
Discontinued intervention due to trial fatigue (n=2)
Lost to follow-up (give reasons) (n= 2)
Discontinued intervention due to trial fatigue (n=2)
Analysis
Analysed (n=48)
Analysed (n=50)
Follow-up (1 year)
Accepted invitation (n = 43)
Lost to follow-up (n=0)
Accepted invitation (n=41)
Lost to follow-up (n=0)
Analysis
Analysed (n=42)
Excluded from analysis (n=1)
(Withdrawal from surgery)
Analysed (n=38)
Excluded from analysis (n=3)
(Withdrawal from surgery)

Figure 2

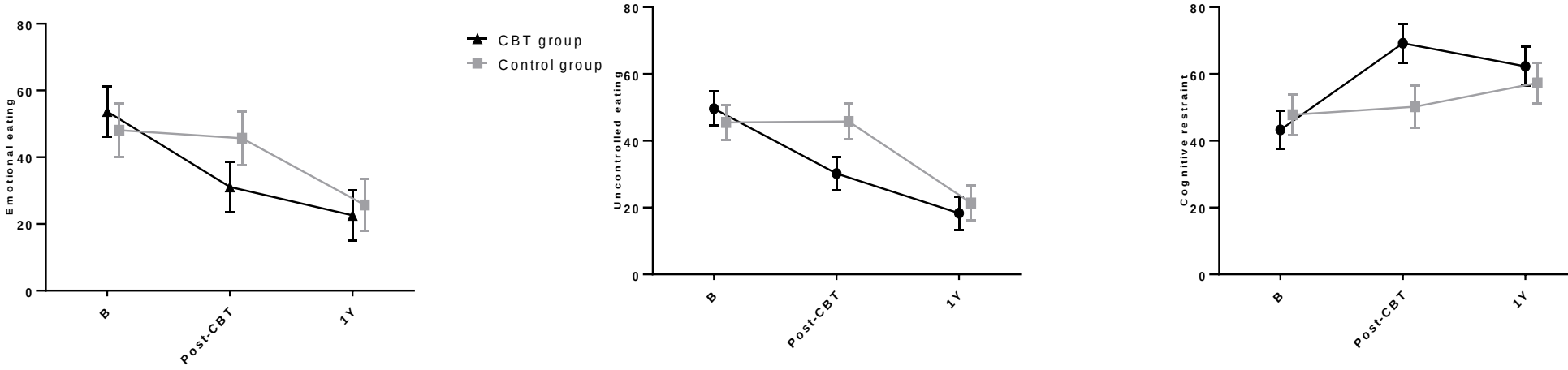
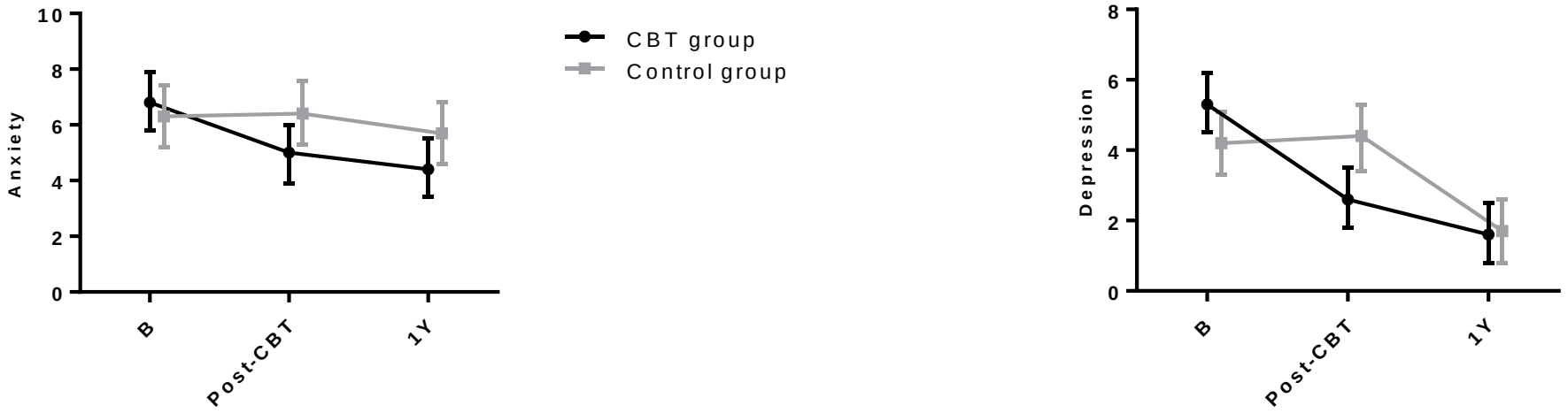

CBT group
Control group
Emotional eating
Uncontrolled eating
Cognitive restraint
0
20
40
60
80
B
Post-CBT
1Y

Figure 3


Anxiety
10
8
6
4
2
0
B
Post-CBT
1Y
CBT group
Control group
Depression
8
6
4
2
0
B
Post-CBT
1Y

Conflict of Interest Disclosure:

None of the authors has any potential financial conflict of interest related to the manuscript.

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