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Impulsivity, Negative Mood, and Disordered Eating in Obesity

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Abstract

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Bariatric surgery is a life-altering procedure that leads to substantial weight loss for most patients with obesity. Psychiatric conditions that may interfere with eating behavior and other behavioral prescriptions after surgery are common. Disordered eating is an established risk factor for inferior weight loss but the effects of negative mood and impulsivity are largely unknown. This thesis aims to investigate the prevalence of and associations between these potential risk factors and eating behavior in bariatric surgery patients.

Study I assessed the prevalence of adult Attention Deficits/Hyperactivity Disorder (ADHD) symptoms in bariatric surgery patients. Symptoms of adult ADHD were elevated compared to the normal population and associated with symptoms of disordered eating, anxiety, and depression.

Study II investigated whether treatment with Behavioral Activation (BA) could ameliorate binge eating and other symptoms of disordered eating in patients with obesity and Binge Eating Disorder. The results showed that BA was effective in increasing activity levels and improving mood but not in ameliorating binge eating in these patients.

Study III was a prospective study on disordered eating, symptoms of depression and anxiety, symptoms of adult ADHD, and alcohol risk consumption before surgery and at follow-up after 12 months. After controlling for age, no variable measured before surgery could predict weight loss after surgery. Disordered eating after surgery was associated with inferior weight loss in men and a subgroup of older female participants.

The present thesis concludes that symptoms of adult ADHD are common among bariatric surgery patients and associated with disordered eating. There is no indication that symptoms of adult ADHD are associated with short-term inferior weight loss after surgery. However, adult ADHD may be a risk factor for postsurgical alcohol abuse. The treatment study showed no direct association among activity, mood, and binge eating. BA, while effective in improving mood, was found not to be an effective treatment for BED, at least in the short group format investigated.

Keywords: Obesity, Eating disorder, Depression, ADHD

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To my primary reinforcer

List of Papers

This thesis is based on the following papers, which are referred to in the text by their Roman numerals.

I   Alfonsson, S., Parling, T., Ghaderi, A. (2012). Screening of adult ADHD among patients presenting for bariatric surgery, *Obesity Surgery*, 22, 918-926.

II   Alfonsson, S., Parling, T., Ghaderi, A. (2014). Group Behavioral activation for patients with severe obesity and Binge eating disorder: A randomized controlled trial. *Manuscript submitted for publication.*

III   Alfonsson, S., Sundbom, M., Ghaderi, A. (2014). Is age a better predictor of weight loss one year after Gastric bypass than symptoms of disordered eating, depression, adult ADHD, and alcohol consumption? *Manuscript submitted for publication.*

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Contents

Abbreviations

%EBMIL	Percent Excess BMI Loss
ADHD	Attention Deficits/Hyperactivity Disorder
ANOVA	Analysis of Variance
ASRS	Adult ADHD Self-Report Scale
AUDIT	Alcohol Use Disorder Identification Test
BA	Behavioral Activation
BADS	Behavioral Activation in Depression Scale
BED	Binge Eating Disorder
BMI	Body Mass Index
CBT	Cognitive Behavior Therapy
CT	Cognitive Therapy
DBT	Dialectical Behavior Therapy
DRO	Discriminative Reinforcement of Other
DSM	Diagnostic and Statistical Manual of Mental Disorders
EDE	Eating Disorder Examination
EDE-Q	Eating Disorder Examination Questionnaire
EDO	Eating Disorders in Obesity Questionnaire
EROS	Environmental Reward Observation Scale
GFCQT	General Food Cravings Questionnaire Trait
HADS	Hospital Anxiety Depression Scale
IPT	Interpersonal Therapy
MMRM	Mixed-effects Models Repeated Measures
POA	Person Oriented Approach/Person Oriented Analysis
RYGBP/GBP	Roux-en-Y Gastric Bypass
SAS-M	Social Adjustment Scale Modified

Introduction

In the last fifty years, obesity has become one of the largest health concerns in the developed world. Not only has the prevalence increased but attempts to find effective treatments have largely been without success (Malik, Willett, & Hu, 2013). When bariatric surgery procedures were developed and proven safe, persons with severe obesity had for the first time access to treatments with long term effects (Sjöström et al., 2004). However, bariatric surgery is not equally effective for everyone and 5-10% of patients regain most of their lost weight within a few years after surgery (Hörchner & Schweitzer, 2013; Sjöström, et al., 2004). Binge eating and depression were early identified as two possible risk factors for less successful weight loss and it is widely recommended to screen all patients for such symptoms as well as other psychiatric disorders prior to surgery (Mechanick et al., 2009). But prospective studies have failed to conclusively associate presurgical binge eating and depression with inferior weight loss, indicating that the associations between risk factors and weight outcome are complex or not well understood. The specific relationship between two major risk factors, negative mood and binge eating needs further investigation.

After surgery, non-adherence to behavioral recommendations is associated with inferior weight loss (Toussi, Fujioka, & Coleman, 2009; Vidal et al., 2014). This includes not following dietary guidelines and recommendations for physical activity, and failing to attend follow-up appointments. Binge eating and other forms of disordered eating naturally affect patients' ability to follow dietary prescriptions, but the reasons why many find it hard to follow other recommendations are unclear. In order to help more patients achieve successful outcomes after bariatric surgery, we must identify and evaluate new potential risk factors. We can then investigate new, effective treatments that target these risk factors while also giving patients better help to follow behavioral prescriptions after surgery.

Obesity

The past half-century has seen a massive rise in obesity in the developed world due to changes in lifestyle patterns and living environment (Caterson & Gill, 2002). Foods have become richer in fat and sugar, and thus contain more energy. At the same time, physical activity both during work- and lei-

sure time has decreased, reducing energy expenditure for many. In this context the rising levels of obesity are unsurprising, though still alarming. Obesity, and especially severe obesity, is associated with the most common cause of death, cardiac failure, and constitutes a major risk factor for severe diseases like diabetes mellitus, hypertension, and some forms of cancer (Flegal, Graubard, Williamson, & Gail, 2007; Ogden, Yanovski, Carroll, & Flegal, 2007). Furthermore, obesity is associated with many other health related problems, such as pregnancy complications (Linne, 2004).

While the increase in obesity seems to have leveled of over the past decade, at least in Sweden, the proportion of people with obesity still represents a formidable health issue (Rokholm, Baker, & Sørensen, 2010). Why the prevalence of obesity has leveled of is unclear but, since it seems to be a general trend affecting all age groups, environmental factors may play a major role. With the possible exception of Japan, there seems to be no country where obesity is on the decline. Obesity will thus continue to be a major health issue in most countries in the foreseeable future.

Obesity is often assessed by measuring Body Mass Index (BMI), which is weight divided by squared height. A person with a BMI above 25 is considered overweight while one with a BMI over 30 is considered to have obesity. However, BMI does not take into account how or where fat is stored in the body. This is crucial since visceral adiposity, fat stored in the upper body around the waist, is more strongly associated with health problems than fat stored in the lower body (Montague & O'Rahilly, 2000). Fat deposition in the body is mainly governed by genetic factors and there is a clear gender difference in that men are more prone to excess visceral fat deposits than women.

The genetics and physiology of obesity

Although obesity has increased because of environmental factors, the genetic component still accounts for about 50% of the risk of developing obesity (Walley, Asher, & Froguel, 2009). The exact mechanisms of this genetic influence have been difficult to establish and probably include epigenetic effects as well. A number of genes have been shown to be involved in the development of obesity, through many different pathways. Genes that affect metabolism and gastric uptake are primarily involved but genes that have more indirect associations may also be very important (Cummings & Schwartz, 2003). This includes genes that affect eating behaviors, feelings of satiety and physical activity (e.g., Qi et al., 2012). On a physiological level, there are several biological pathways associated with developing obesity. First, there must be an energy surplus: the body must receive more energy than it expends. Energy balance and regulation, homeostasis, is governed by complex neurological and hormonal interactions. Both the central nervous system and the endocrine system are involved in homeostasis and energy balance is regulated through behaviors as well as the processes of inner or-

gans (Berthoud & Morrison, 2008). The physiology of the energy-regulating systems can change as a result of developing obesity. The hormonal systems, including the important hormone Leptin, that help regulate dietary intake seem to become less effective after prolonged obesity. It has been hypothesized that some degree of hormonal resistance develops (Morrison & Berthoud, 2007).

The other major regulatory system for homeostasis, the nervous system, is involved in a constant feedback loop involving appetite and eating behavior. It is noteworthy that the neurological mechanisms for controlling homeostasis, just like the endocrine system, seem to be centered on avoiding undernutrition, not overnutrition. This is most probably a consequence of evolutionary pressure since nutritional energy has historically been a scarce resource for humankind. Today however, these mechanisms may be the main reasons why it is so difficult to lose weight after having developed obesity (Morton, Cummings, Baskin, Barsh, & Schwartz, 2006). The neurological signals that affect eating behaviors are governed primarily by the hindbrain which integrates endocrine signals with sensory signals. This integrated information can affect behaviors directly, but these are often regulated by the forebrain. Consequently, eating behaviors are closely associated with conscious cognitions and emotions. The neurological systems for regulating eating behaviors are under the influence of the reward systems, mainly the opioid and dopamine systems, which govern feelings of pleasure and relief (Morrison & Berthoud, 2007; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008). The complex interaction between reward systems and eating behavior implies that many different processes may affect eating. In some people, an inefficient reward system may result in increased eating to achieve the same level of pleasure as in people with more efficient reward systems. Other people may have a powerful association between eating and reward, resulting in a strong positive loop where eating leads to further eating. Changes in these reward systems have been observed in people with obesity but the temporal and causal mechanisms are unclear. Further, the reward systems are subject to desensitization so an increased activation may lead to reduced reward activation for a constant stimulus (Volkow, Wang, & Baler, 2011). This, in turn, may increase motivation for eating and a need to consume more energy in order to experience previous levels of reward.

As learning theory would predict, obesity may lead to a shift toward anticipatory reward for eating, such as that the mere prospect of eating activates the dopamine reward system (Stice, et al., 2008) . It is known that the dopamine system affects inhibitory control, and disruptions in dopamine processes may affect cortical functions such as delay discounting, though these mechanisms are still elusive. In conclusion, eating behavior and other behaviors involved in homeostasis are thus under the influence of the unconscious reward systems but also governed by conscious efforts to control behavior. This

interaction between more basal neurological systems and higher cortical systems that govern eating behavior can in turn be affected by present obesity.

Psychology, behavior and obesity

The psychological risk factors for developing obesity and the psychological and behavioral consequences of having developed obesity partially overlap. For example, physical inactivity and sedentary behaviors, such as television viewing, among children predict adolescent overweight and obesity (Monasta et al., 2010). Children and adolescents with obesity may also develop difficulties in engaging in more physically active leisure activities than media consumption, thus exacerbating the risk of further weight gain. Generally, prolonged psychological stress as a response to environmental factors during childhood or adolescence seems to be a risk factor for developing obesity (Björntorp, 2001; Torres & Nowson, 2007). More specifically, a stress response with increased levels of cortisol in the body affects the accumulation of visceral fat and also the Leptin regulation system of energy balance. For adults, environmental risk factors that affect visceral adiposity through the stress response system include low education and financial pressures. The associations between psychosocial stress and weight gain seem to be partly gender-specific, since men tend to increase their caloric consumption (partly by drinking more alcohol) and gain weight as a consequence of stress more than women (Torres & Nowson, 2007). The association between psychosocial stress, physiological stress response, eating behaviors and obesity is complex (McLaren, 2007). Psychosocial stress is associated with low socioeconomic status which in turn is associated with unhealthy eating habits and physical inactivity as well as with poor health and psychiatric comorbidities. In adults, those with depression have an increased risk of developing obesity but the association is bidirectional so obesity also predicts future depression (Faith et al., 2011; Luppino et al., 2010). It has been suggested that both obesity and depression are associated with low-grade inflammation and also with changes in the endocrine system. A similar bidirectional association has been found between obesity and anxiety disorders, but has attracted less attention (Gariepy, Nitka, & Schmitz, 2009). Since most studies investigating psychosocial stress and weight gain have been cross sectional, it is difficult to establish the causal mechanisms behind these associations.

The overall prevalence of depression and anxiety disorders is higher among both men and women in the obese population than among the non-obese (D. Barry, Pietrzak, & Petry, 2008). For overweight however, there is no elevated risk for psychiatric comorbidity for men, only for women. It has been argued that this difference is due to divergent social consequences of overweight in men and women. While most people who develop obesity are not depressed, many report psychosocial stigma (Puhl & Heuer, 2009). People with obesity encounter prejudice and are perceived as lazy and less com-

petent by, for example, employers and clinicians in the healthcare system. Both men and women report low self-esteem due to overweight and may avoid social interactions and physical exercise because of negative social evaluation. These negative experiences may form part of a vicious circle of negative emotion, low activity levels and weight gain. Negative mood is associated with eating behaviors and may increase everyday consumption of sweet- and high-fat foods (Konttinen, Männistö, Sarlio-Lähteenkorva, Silventoinen, & Haukkala, 2010). This may, over time, lead to excess consumption of energy and thus to weight gain, especially since physical activity is inversely related to stress and depression (Ströhle, 2009). In conclusion, while weight gain and obesity can be largely explained by hereditary factors, psychological factors and everyday behaviors also play important roles (Marti, Martinez-González, & Martinez, 2008).

Eating behavior and obesity

Besides hereditary factors, eating behaviors and a sedentary lifestyle (e.g. much time spent watching TV) have been closely associated with the development of obesity (Berg et al., 2009; Hu, 2003). The energy expenditure side of energy-balancing behaviors has received relatively little attention as yet (Thorburn & Proietto, 2000). Concerning energy consumption, we know that eating behaviors are affected by many factors, both endogenous and exogenous. As described above, eating behaviors can be initiated by signals from several of the inner organs involved in the metabolism of the body. But negative feelings like stress and negative mood caused by environmental factors can also affect eating behaviors (Adam & Epel, 2007; Canetti, Bachar, & Berry, 2002). The patterns are complex and both the valence and intensity of emotions seem to affect eating. For example, intense fear may reduce both appetite and eating while anger may increase impulsive eating behaviors. These associations seem to be somewhat different in men and women and also contain individual differences. In general however, eating activates the same reward systems that are active in regulating emotional states. The exact underlying mechanisms are not well known but it has been hypothesized that both the dopamine and the serotonin systems play major roles (Wang et al., 2001; Volkow, et al., 2011). For example, some drugs that affect the dopamine system (e.g. amphetamine) effectively reduce appetite and eating.

When we start to eat, the neurological signals for initiating eating are counteracted and eating eventually stops. The transmitters used in these systems, mainly dopamine and serotonin, can produce pleasant feelings or relief from some negative experiences, such as mild pain (Schwartz, Woods, Porte, Seeley, & Baskin, 2000). Eating behavior can thus be either positively or negatively reinforced, depending on the situation. Negative reinforcement is often a very effective way of controlling behavior and this operant mechanism may be one of the reasons why changing eating behavior can be very

difficult (Volkow & Wise, 2005). Withholding reward, as in refraining from eating, will increase the reward value and thus, for example strengthen the potential reinforcement of eating when on a diet. At the same time, eating behavior can be classically conditioned on stimuli, both environmental and emotional (Berthoud & Morrison, 2008). When operant and classical conditioning coincide, behaviors very soon become habits that are less affected by changes in contingencies.

Since eating behavior can be subject to the control of both classic and operant conditioning, the processes can be difficult to investigate. This may help explain why experimental studies have had mixed results regarding the relationship between negative feelings and eating behaviors, especially when they are analyzed in more ecological studies (Tomiyama, Mann, & Comer, 2009; Yeomans & Coughlan, 2009). Paradoxically, such emotional eating appears to be more common in people who also report high levels of restraint over eating. It has been hypothesized that people with high levels of emotional eating engage in, or are cognitively occupied with, restraint in order to control their impulsive eating. Cognitive effort to inhibit a previously reinforced behavior is difficult, and people characterized by emotional eating typically experience that their efforts repeatedly fail. This pattern is a core feature of the eating disorders of Bulimia Nervosa and Binge Eating Disorder (BED). Emotional eating is also closely associated with other symptoms of disordered eating such as overeating and losing control over eating (Goossens, Braet, Van Vlierberghe, & Mels, 2009; Telch & Agras, 1996).

Disordered eating in obesity

Apart from the formal eating disorder diagnoses, there are many forms of problematic eating, such as constant snacking, that may not in themselves constitute disorders but may hinder healthy eating or lead to negative health consequences (Latner, Hildebrandt, Rosewall, Chisholm, & Hayashi, 2007; Tanofsky-Kraff & Yanovski, 2004). For example, these problems may lead to long term weight gain and also affect the ability to follow behavioral weight loss programs (Elfhag & Rössner, 2005; Hays et al., 2002). When it comes to weight maintenance, having a flexible control over eating and regular eating patterns seem to be beneficiary while the opposite is associated with weight cycling and weight gain.

An irregular eating pattern is not considered a symptom of disordered eating, but people with obesity tend to eat more irregularly than the nonobese (Ma et al., 2003). Specifically, people with obesity tend to skip breakfast, eat more in the latter part of the day and to have longer intervals between meals. While irregular eating is associated with obesity, it is also common among people with BED (Masheb & Grilo, 2006a) and is targeted in behavioral treatments of that disorder (see below). The suggested mechanism is that regular eating patterns reduce hunger and facilitate inhibition of eating with

triggers other than hunger. This is based on the finding that irregular eating may lead to both increased snacking and poor dietary choices (Kerver, Yang, Obayashi, Bianchi, & Song, 2006). For these reasons, daily monitoring of calorie intake and eating patterns is often a major component of behavioral treatments for obesity (Butryn, Webb, & Wadden, 2011).

Obesity treatments

Over the years there have been many attempts to develop effective behavioral treatments for obesity (Franz et al., 2007). Early on, it was discovered that behavioral programs can initiate weight loss but that few participants maintain their weight loss over time (Elfhag & Rössner, 2005; Stunkard & Penick, 1979). Adherence to treatment prescriptions is closely associated with weight loss in these programs. At the same time, adherence is often only moderate during the treatment and often decrease even further after the end of treatment (Moroshko, Brennan, & O'Brien, 2011). It has been difficult to find variables that consistently predict treatment adherence and drop out, partly because insufficient reporting in many studies. However, lower education, lower social support and symptoms of disordered eating seem to be associated with high risk of attrition from treatment (Moroshko, et al., 2011).

Maintaining changed behavior after the end of treatment is very difficult, owing to the specific contingencies associated with weight loss behavior (Stunkard & Penick, 1979). During weight loss treatment, obese participants seem to find new sources of positive reinforcement that are not related to food or eating but whether this effect diminishes over time is unknown (Buscemi, Murphy, Berlin, & Raynor, 2014). Unfortunately, the natural positive reinforcements of weight-loss behaviors are scarce while stimuli for incompatible behaviors are common. This can be seen in contrast to other problems, such as depression, where treatment often leads to a positive circle of activities that are naturally reinforced and thus more easily maintained. Because of the difficult set of contingences involved in losing weight, even obesity treatment programs specifically focusing on maintaining behavior change show no better long-term results than other interventions (Z. Cooper et al., 2010). Today, few standardized behavioral interventions for obesity have good empirical support (LeBlanc, O'Connor, Whitlock, Patnode, & Kapka, 2011) but there are a few interventions that show some promise despite modest results (Butryn, et al., 2011).

Irrespective of demographical variables, such as education, the presence of psychiatric problems is a risk for inferior weight loss after behavioral treatments. Both binge eating and depression seem to hinder weight loss and may be important to target specifically (Linde et al., 2004). Nonetheless, nearly half of all participants in an intensive intervention will return to their original weight within five years (Butryn, et al., 2011). The prevalence of eating or mood disorders alone, though high, cannot explain the relative

ineffectiveness of behavioral treatments for obesity. Rather, obesity seems to represent a condition where physiological, neurological and behavioral factors make it very difficult for most people to affect their weight by changing behavior. Today, the only treatment that can lead to substantial, long-term weight loss for most patients is bariatric surgery.

Bariatric surgery for obesity

Bariatric surgery includes several procedures where the capacity of the stomach and intestines to accommodate food and/or absorb energy is restricted, which also leads to reduced feelings of hunger (Elder & Wolfe, 2007; O'Brien, 2010). In Sweden, a common procedure for patients with a BMI between 30 and 50 is Roux-en-Y Gastric Bypass (RYGBP or GBP), where only a small pouch of the stomach remains and the small intestine is shortened. RYGBP typically leads to about 60% excess weight loss and is a relatively safe procedure with few medical complications (Buchwald et al., 2009). For these reasons, RYGBP has become the most common bariatric surgery procedure in Sweden.

Surgical reduction of the stomach and intestines reduces the ability to absorb energy but also affects food preferences and the amount and types of food that can be eaten (Stocker, 2003). After RYGBP, sugar-rich food and some fat-rich food may lead to a type of discomfort called "dumping syndrome". This syndrome often includes nausea, sweating and psychological discomfort. The fact that suboptimal food choices lead to aversive experiences is sometimes considered to enhance the effect of surgery. It is noteworthy that not all RYGBP patients experience dumping syndrome and that sensitivity to sugar and fat can change over time. Apart from dietary choices, patients are also required to change their eating habits in other ways.

After RYGBP, patients are instructed to eat small meals at regular intervals, typically every two to three hours (Stocker, 2003). They are also instructed to chew thoroughly, eat slowly and avoid drinking while eating. Dietary guidelines prescribe that proteins should be prioritized over carbohydrates, fats and fibers, a recommendation that is often a major readjustment for patients. Since RYGBP is in practice irreversible, they must follow these dietary regimens for the rest of their lives. At the same time, a large minority of patients show low adherence to dietary recommendations, which may affect weight loss results negatively (Sarwer et al., 2008).

Long-term results of bariatric surgery

Overall, health and psychosocial status improve for most patients after surgery, and a vast majority of patients are satisfied with having had the operation (Herpertz et al., 2003). Reduced levels of anxiety- and depressive symp-

toms are reported by many patients and disordered eating seems to decrease. Weight loss is initially rapid but levels out after one to two years. A weight-loss nadir typically occurs within 24 months after surgery, after which most patients slowly regain weight (Christou, Look, & MacLean, 2006). In the Swedish Obese Subjects study (which includes several forms of bariatric surgery), where patients were followed for up to 10 years, about 9% eventually regained almost all of their lost weight (Sjöström, et al., 2004). Weight loss seems to follow fairly stable trajectories so patients at risk of less than optimal weight loss can often be identified early after surgery based on their poor initial weight loss (Courcoulas, Christian, Belle, & et al., 2013). It is not known whether the weight regain is due mainly to physiological or behavioral factors. Repeat surgery due to medical complications is performed on 3-10% of patients, depending on surgery type. The search for psychological or behavioral risk factors for reduced weight loss or weight regain has been extensive but the results are inconclusive.

Risk factors for inferior weight loss after surgery

Both demographic variables and mental health before surgery have been found to be associated with weight outcome after surgery (Van Hout, Verschure, & Van Heck, 2005; Wimmelmann, Dela, & Mortensen, 2013). Younger patients show more weight loss after surgery than their elders, partly thanks to fewer complications and better mobility. Regarding gender, results have been mixed but men usually show greater weight loss than women. The reasons for this apparent gender difference are not known but it has been suggested that gender-specific metabolic differences may play a part, as may the higher prevalence of eating disorders among women. When it comes to mental health the results are even more contradictory, with few consistent risk factors identified. Nevertheless, psychiatric screening is often recommended as part of the presurgical evaluation procedure. Psychiatric disorders, including mood- and anxiety disorders, are common among bariatric surgery candidates (Kalarchian et al., 2007; Mühlhans, Horbach, & de Zwaan, 2009). Other diagnoses that are common are eating disorders, substance abuse disorders and personality disorders, all of which are potential risk factors for low weight loss after surgery. Multiple psychiatric diagnosis or more severe conditions seem to have a negative effect on weight loss (Kinzl et al., 2006; Livhits et al., 2012). For example, one study found that psychiatric patients with a history of inpatient care showed lower weight loss after surgery than psychiatric patients who had only received outpatient care (Wimmelmann, et al., 2013). This implies that the patient's overall functioning may be more important for surgery outcome than specific psychiatric diagnoses per se.

The two psychological variables that were identified as potential risk factors early on and have received the most attention over the years are binge eating and depression (e.g., Hsu et al., 1998). Binge eating, which is defined

as overeating while also losing control of one's eating, is common among people with obesity seeking surgical treatment (Niego, Kofman, Weiss, & Geliebter, 2007). Binge eating is still possible after bariatric surgery but may take other forms than prior to surgery. For example, patients may binge-eat small volumes of food frequently or binge-drink sweet liquids. These behaviors are more compatible with the postoperative physiological restrictions, while counteracting weight loss and leading to discomfort (Sarwer, Wadden, & Fabricatore, 2005). In many studies, but not all, binge eating has predicted inferior weight loss, especially if it occurs after surgery rather than before (Colles, Dixon, & O'Brien, 2008a). That binge eating has not persistently shown to predict reduced weight loss may be connected with how it is conceptualized and measured (Niego, et al., 2007). Disordered eating in this population is often conceptualized as full filling the BED diagnosis. The diagnosis includes a criterion that binge eating should involve an unusually large amount of food given the circumstances, a criterion that may be difficult to interpret after surgery, see Figure 1 for complete diagnostic criteria from the Diagnostic and Statistical Manual of Mental Disorders V (DSM-V; American Psychiatric Association, 2013) .

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

    1. Eating, in a discrete period of time (e.g. within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
    2. A sense of lack of control over eating during the episode (e.g. a feeling that one cannot stop eating or control what or how much one is eating).

B. The binge eating episodes are associated with three or more of the following:

    1. Eating much more rapidly than normal.
    2. Eating until feeling uncomfortably full.
    3. Eating large amounts of food when not feeling physically hungry.
    4. Eating alone because of feeling embarrassed by how much one is eating.
    5. Feeling disgusted with oneself, depressed or very guilty afterwards.

C. Marked distress regarding binge eating is present.

D. Binge eating occurs, on average, at least once a week for three months.

E. Binge eating not associated with the recurrent use of inappropriate compensatory behaviours as in Bulimia Nervosa and does not occur exclusively during the course of Bulimia Nervosa, or Anorexia Nervosa methods to compensate for overeating, such as self-induced vomiting.

Figure 1. DSM-V criteria for Binge Eating Disorder.

After bariatric surgery, most patients cannot eat large amounts of food but may still experience other symptoms of binge eating, such as eating rapidly, feeling a loss of control over eating and feeling ashamed after eating (Colles, et al., 2008a). Trying to assess and diagnose BED after bariatric surgery is thus difficult both for the patients themselves and for clinicians. Binge eating after surgery is associated with other forms of disordered eating such as emotional eating, night eating and constant nibbling (Niego, et al., 2007). It is unclear whether the various forms of disordered eating are equally important in terms of weight loss.

The other major potential risk factor, depression, can affect weight loss both directly and indirectly. Depression is common among people with obesity and even more so among people with comorbid BED (Stunkard, Faith, & Allison, 2003). Depression is most often associated with a decrease in appetite but some patients instead report the atypical symptom of increased appetite (Casper et al., 1985). Depression can affect eating behaviors but also weight loss, through a reduced level of physical activity typically seen in depression, or as a side effect of antidepressant medication (Blaine, 2008; Fogelholm & Kukkonen-Harjula, 2000).

Indirectly, depression can impair the patient's ability to follow medical prescriptions (DiMatteo, Lepper, & Croghan, 2000). The reasons for this are not well understood but, apart from impaired motivation, one pathway may be through reduced executive function. Depression often affects such aspects of executive function as working memory, while more severe depression is associated with more severe deficits in executive function (Paelecke-Habermann, Pohl, & Leplow, 2005). Depression may indirectly affect the ability to alter habitual behaviors and adherence to demanding treatment recommendations (Williams & Thayer, 2009). Like binge eating, depression has not consistently been associated with low weight loss after surgery (Van Hout, et al., 2005). However, when depression or BED is identified, adequate treatment can be initiated to reduce any potential risk but also to ameliorate the suffering of the patient. The standard treatment for depression is pharmalogical but for BED, psychological treatments have somewhat better empirical support and should be the first-line treatment option (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007).

Binge Eating Disorder

Binge Eating Disorder became a diagnosis in its own right with the new DSM-V in 2013 (American Psychiatric Association, 2013) but was previously included in DSM-IV as a research diagnosis (American Psychiatric Association, 1994). The main symptom criteria for BED are recurrent episodes of binge eating characterized by a sense of lack of control over eating. The second symptom criterion of BED, a feeling of loss of control over eat-

ing, seems to be the most central symptom of the disorder (Colles, Dixon, & O'Brien, 2008b).

The prevalence of BED is estimated to be 2-4% in the general population and up to 30% or more in obese patients seeking surgery (Hudson, Hiripi, Pope Jr, & Kessler, 2007; Niego, et al., 2007). The prevalence among bariatric surgery patients varies greatly and is probably affected to a large extent by means of assessment and sample characteristics. As mentioned above, binge eating may be difficult for both patients and clinicians to assess, especially during obesity treatment when patients are on diets or following other behavioral regimens. Before behavioral obesity treatment, BED is often assessed and targeted specifically since present BED may impede weight loss both due to uncontrolled eating and comorbid negative mood (Linde, et al., 2004; Sherwood, Jeffery, & Wing, 1999).

In an obesity treatment study by Sherwood et al. (1999) there was a clear indication that participants whose mood improved during treatment also improved in both binge eating and weight loss. In several subsequent studies, BED has often been ameliorated by weight-loss treatment, probably owing to more regular eating patterns and reduced obesity stigma (Grilo, Masheb, Wilson, Gueorguieva, & White, 2011; Vocks et al., 2010). Behavioral weight loss treatment does not target binge eating directly and has smaller effect sizes than psychotherapy. Thus, for optimal results, patients diagnosed with BED should preferably receive specialized treatment.

Treatment of Binge Eating Disorder

Cognitive Behavior Therapy (CBT), Interpersonal Therapy (IPT) and, to some extent, Dialectical Behavioral Therapy (DBT) are empirically supported treatments for BED. CBT has received most support, having shown to be effective in treating BED in individual format, in group format and in the form of self-help bibliotherapy (Brownley, et al., 2007). IPT seems to be as effective as CBT but has been assessed in fewer studies so far (Iacovino, Gredysa, Altman, & Wilfley, 2012; Wilson, Wilfley, Agras, & Bryson, 2010). Treatment with DBT for BED have received some attention but the amount of empirical support is still rather limited and DBT is not a recommended first-line treatment for eating disorders (Hay, 2013; Safer & Jo, 2010). Patients who are not helped by one treatment do not seem to benefit from receiving another type of treatment, indicating that the various treatments overlap to some extent or target the same mechanisms of the disorder. At the same time, the treatment rationales are quite different for the different treatments (Murphy, Straebler, Basden, Cooper, & Fairburn, 2012; Murphy, Straebler, Cooper, & Fairburn, 2010).

CBT for BED focuses on increasing regular eating and decreasing inflexible dietary rules while also challenging patients' overvaluation of shape and weight (Murphy, Cooper, Hollon, & Fairburn, 2009). In IPT for BED, the

patient's social networks and role function are in focus and patients are helped to examine their interpersonal relations and develop healthier ones if need be (Lipsitz & Markowitz, 2013). DBT conceptualizes binge eating as a dysfunctional affect regulation strategy and the treatment is focused on changing these strategies into more functional ones (Linehan & Chen, 2005). While the model used in CBT for BED has found some empirical support, the proposed mechanisms in treatments with IPT and DBT are still mostly untested (Rieger et al., 2010; Safer & Jo, 2010). The reason why several different treatments are effective for BED may be due partly to the existence of different subgroups of patients whose symptom profiles differ and who thus respond to different treatments (Peterson et al., 2013). However, as mentioned above, patients who show no improvement from one treatment do not seem to benefit from another and there seems to be no additive effect of providing different treatments in succession. This indicates that the treatments to some extent work through common mechanisms. A common feature of all three treatment models is negative social evaluation, including weight and shape concerns, and an association between the resulting negative mood state and disordered eating.

CBT and DBT focus mainly on the connection between anxiety and eating while IPT focuses more on depressed mood and eating. Patients with negative mood or low self-esteem may thus respond better to IPT than CBT, though this is far from clear (Iacovino, et al., 2012; Sysko, Hildebrandt, Wilson, Wilfley, & Agras, 2010; Wilson, et al., 2010). Apart from their comparable effects on binge-eating outcomes, CBT, IPT and DBT all affect depressive symptoms (Safer & Jo, 2010; Vocks, et al., 2010). This effect is expected for IPT, which was developed to treat depression, and the IPT treatment protocol for BED is similar to that used for treating depression. The effect is, however, somewhat surprising in CBT for BED, which does not explicitly target symptoms of depression. The effect of treatment on these symptoms may simply be due to ameliorated symptoms of eating disorder. Although the mechanisms remain unclear, this is a further indication that disordered eating and negative mood are closely connected. While CBT and IPT for BED are theoretically very different they share some features concerning affect regulation. In both treatments, efforts are made to help patients engage in positively reinforced behaviors. Such features are common in all treatments for depression and may help explain why patients with BED improve in terms of mood. Apart from being interesting theoretically, this may be clinically important since depressive symptoms are common among people with BED.

BED and negative mood

Depression is a common comorbid disorder in bariatric surgery patients with BED (Jones-Corneille et al., 2012). While the majority of patients with BED

are not depressed, negative mood may affect eating behaviors and longtime care after surgery. In general, people with BED report more eating as a response to negative emotions than people without BED (Eldredge & Agras, 1996; Masheb & Grilo, 2006b). Early on, it was hypothesized that emotional eating in patients with BED may be associated with Alexithymia, a difficulty in identifying and verbally expressing emotions (Taylor, 1984). Although inflexible restrain over eating, overvaluation of weight and shape are the main contributors to binge-eating symptomology, difficulties in affect regulation may play an important part (Whiteside et al., 2007).

However, experimental studies have shown that negative mood may be present both before and after binge eating, suggesting that more detailed study of the time frame and association between emotions and binge eating is essential (Hilbert & Tuschen-Caffier, 2007). From a learning theory standpoint, binge eating would be negatively reinforced by reduced distress directly after the binge episode. Showing that such contingencies exist is very difficult since mood often deteriorates after binge eating (Haedt-Matt et al., 2014). Several explanations for these difficulties are possible: that the positive effect on mood is too transient to be measured with the methods used in most studies, that behavior has become habitual and thus less sensitive to emotional consequences or that patients report their cognitive interpretation of the situation rather than their emotional or physical state.

Elevating negative mood in general does not seem to be an effective way of improving binge eating and antidepressant medication has only a limited effect on binge eating symptoms (Vocks, et al., 2010). Patients with BED and depressive symptoms may actually benefit more from CBT than from medication (Kalarchian, et al., 2007). This may be explained by the fact that CBT is quite comprehensive and teaches skills that can be used in many circumstances, to tackle various problems regarding both eating and mood. In contrast to treatments of BED, the therapeutic mechanisms in psychological treatments for depression are relatively well known.

Psychological treatment of depression

Cognitive Therapy (CT) for depression was the first psychological treatment for depression with good empirical support. CT (later Cognitive Behavior Therapy, CBT) for depression is fairly complex and comprises many components including cognitive restructuring and behavioral experiments (Beck, 2011). In a series of studies, Jacobsen and colleagues showed that one component of CBT, Behavioral Activation (BA), when provided on its own was at least as effective for treating depression as the full CBT treatment (Jacobson et al., 1996; Jacobson, Martell, & Dimidjian, 2001). Since then several studies have shown that BA is an effective treatment for depressive symptoms in many populations and also effective in group format (Mazzucchelli, Kane, & Rees, 2009). One small study has shown that BA

can mitigate depression in people with obesity and that it also affects eating habits (Pagoto et al., 2008). Unfortunately, symptoms of disordered eating were not assessed in that study.

Behavioral Activation

Behavioral Activation is based on a behavioral model of depression that states that mood and behavior are closely associated (Jacobson, et al., 2001; Martell, Dimidjian, & Herman-Dunn, 2013). The fundamental principle of BA is that freely chosen active behaviors are positively reinforced while passive behaviors are negatively reinforced (Dimidjian, Barrera Jr, Martell, Muñoz, & Lewinsohn, 2011). If a person's environment or everyday life is changed in some profound way, the behavioral contingencies may be altered. Previous active behaviors may become unavailable or no longer be positively reinforced; they may even be punished. Passive behaviors thereby become more adaptive, especially in the short term. This may initiate a vicious circle of negative mood with low levels of activity and a lack of positive reinforcement. Major symptoms of depression include loss of energy and initiative, both closely associated with the level of positive activity. BA is centered upon breaking the vicious circle by increasing behaviors that are positively reinforced (Dimidjian, et al., 2011).

Early forms of BA focused on scheduling activities to break patterns of inactivity and to practice social skills that may lead to positive reinforcement (Lewinsohn, Sullivan, & Grosscup, 1980). In later versions of BA a more functional way of analyzing each individual has been emphasized in planning activities (Martell, Addis, & Jacobson, 2001). Patients are encouraged to identify what behaviors they find positively reinforced, for example social activities or leisure activities that they find meaningful. The focus is not so much on increasing the general level of activity but to find each person's sources of intrinsic positive reinforcement. While BA was developed as a treatment for depression, BA has proven to affect psychological well being in non-depressed populations as well (Mazzucchelli, Kane, & Rees, 2010). Behavioral Activation has also been used in behavioral medicine and adapted to suit different clinical populations, for example smokers (MacPherson et al., 2010). Nonetheless, the fundamental aspect of BA is activity scheduling and thus acting according to planned behavior rather than emotional states or impulses (Kanter et al., 2010).

BED and impulsivity

The loss of control over eating in BED is initiated by stimuli, such as emotional states, often combined with environmental cues or circumstances. Overall, signs of elevated impulsivity are more common among people with BED, as well as those with other eating disorders, than in the normal popula-

tion (Stice, 2002). Impulsivity is also associated with risk behaviors like alcohol- and drug abuse (Dawe & Loxton, 2004). It has been suggested that impulsivity includes increased reward sensitivity, implying that impulsive eating (e.g. binge or emotional eating) may be more strongly reinforced in highly impulsive people. Experimental studies have indicated a weak inter-action effect between negative mood and impulsivity on emotional eating but further studies are needed to clarify the association (Bekker, van de Meerendonk, & Mollerus, 2004; Nasser, Gluck, & Geliebter, 2004). High impulsivity may thus increase the risk for emotional eating or binge eating when the person is in a negative mood state.

Impulsivity is a multifaceted phenomenon where the central feature may be defined as a tendency to act in response to immediate cues rather than planned goals (Evenden, 1999). Impulsivity is closely associated with inhibi-tory control, which is part of the larger construct of executive function. This construct is elusive but includes the ability to perform goal-oriented behav-ior, as well as flexibility (Jurado & Rosselli, 2007). Other important aspects are problem solving, self-monitoring and fluency. Executive function devel-ops during childhood and measures of executive function typically increase with advancing age, following the development of the brain, primarily the frontal lobes (Zelazo, Carlson, & Kesek, 2008). Exhibiting impulsive behav-ior is normal in infants, but sustained elevated impulsivity during school age and adolescence is associated with neuropsychological difficulties such as Attention Deficits/Hyperactivity Disorder (ADHD) (Sonuga-Barke, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

Adult ADHD

Attention Deficits/Hyperactivity Disorder (ADHD) is a developmental dis-order found in about 5% of all children (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). ADHD is more common in boys (with a preva-lence of up to 10%) than girls but since the disorder may present differently in boys and girls, girls may be underdiagnosed as a group (Gershon, 2002). Whether this gender difference is due to biological or environmental factors or to methodological issues and stereotypical assessment is debated. The diagnostic criteria of ADHD include symptoms both of attention deficits and of impulsivity/hyperactivity (see Figure 2 for full symptom criteria from DSM-IV). In both sexes, ADHD is associated with deficits in some, but not all, aspects of executive function (Sergeant, Geurts, & Oosterlaan, 2002). Executive function is difficult to measure and children with ADHD often have comorbid psychological disorders, such as anxiety disorders, that could affect measurement of ADHD symptoms as well as executive function. For some children, the ADHD symptoms are ameliorated when they grow into

adulthood but for many the symptoms prevail or present in a different form (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Kessler et al., 2010).

Adult ADHD is characterized more by attentions deficits than by hyperactivity but many with adult ADHD still show marked inhibition deficiencies or impulsivity (Boonstra, Kooij, Oosterlaan, Sergeant, & Buitelaar, 2010). While persons with adult ADHD may have deficiencies in some aspects of executive function, it is noteworthy that present adult ADHD is not

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A) Either (1) or (2):
 1) Inattention: six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

   (a) Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
   (b) Often has difficulty sustaining attention in tasks or play activities.
   (c) Often does not seem to listen when spoken to directly.
   (d) Often does not follow through on instructions and fails to finish school work, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions).
   (e) Often has difficulty organizing tasks and activities.
   (f) Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework).
   (g) Often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools).
   (h) Is often easily distracted by extraneous stimuli.
   (i) Is often forgetful in daily activities.

 2) Hyperactivity-Impulsivity: six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

   Hyperactivity
   (a) Often fidgets with hands or feet or squirms in seat.
   (b) Often leaves seat in classroom or in other situations in which remaining seated is expected.
   (c) Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness).
   (d) Often has difficulty playing or engaging in leisure activities quietly.
   (e) Is often "on the go" or often acts as if "driven by a motor".
   (f) Often talks excessively.

Figure continues on the next page.

Impulsivity
  (g) Often blurts out answers before questions have been completed.
  (h) Often has difficulty awaiting turn.
  (i) Often interrupts or intrudes on others (e.g., butts into conversations or games).

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorders, or a Personality Disorder).

Figure 2. DSM-IV criteria for Attention-Deficits/Hyperactivity Disorder.

in itself associated with general measures of intelligence (Bridgett & Walker, 2006). Nevertheless, adult ADHD may lead to disruptions in daily life and effect both health and quality of life (Harpin, 2005). Problems such as losing one's job, interpersonal conflicts and parenting difficulties are all more common among people with adult ADHD than in the normal population. Fortunately, ADHD can often be successfully treated with stimulant medication in both children and adults (Faraone & Glatt, 2010). But many adults with ADHD are probably undiagnosed and never offered treatment.

The prevalence of adult ADHD is estimated at about 2-3% in the general population, but the diagnosis is difficult to assess and the level is therefore uncertain (Simon, Czobor, Bálint, Mészáros, & Bitter, 2009). Furthermore, people with adult ADHD are probably overrepresented in the healthcare system, since adult ADHD is a disorder which is often associated with other psychosocial problems (Halmøy, Fasmer, Gillberg, & Haavik, 2009).

Adult ADHD comorbidities

Adults with ADHD often have one or more comorbid disorders with rates of depression, bipolar disorder, anxiety, eating and sleep disorders often double that of the normal population (Spencer, Biederman, & Mick, 2007; Wilens, Biederman, & Spencer, 2002). Drug abuse and conduct disorders are also

common with prevalence of up to 50% in untreated young adults with ADHD (Kalbag & Levin, 2005). ADHD symptoms like impulsivity are elevated in patients with Bulimia Nervosa and BED and may be an important factor in these disorders (Dawe & Loxton, 2004; Rosval et al., 2006; Waxman, 2009). Symptoms of ADHD may contribute to overeating, emotional eating and binge eating through different mechanisms (Cortese, Bernardina, & Mouren, 2007; Caroline Davis, Levitan, Smith, Tweed, & Curtis, 2006). Besides impulsivity, deficiencies in the dopamine reward system typically seen in individuals with ADHD may play a role in the difficulties of controlling eating behaviors. People with ADHD typically need stronger reinforcement to change behavior and are less sensitive to changing contingencies. It has been hypothesized that these reward deficiencies may promote behaviors that lead to strong reinforcement and thus contribute to maintaining hedonistic- or risk behaviors often seen in people with ADHD. Such deficiencies may also influence the ability to make adequate food choices and regulate eating behavior. However, the genetic and neurological factors that contribute to patterns of risk behaviors are far from clear (Sagvolden, Johansen, Aase, & Russell, 2005).

Risk behavior

Risk behavior include all behaviors that offers positive arousal or emotional numbing while having a comparably high risk of bodily, psychological or social harm, such as reckless driving, alcohol consumption and drug use (Roberti, 2004). Risk behaviors are more common among adolescents than in adults, more common among men than women and more common among people diagnosed with ADHD than others. The mechanisms underlying these differences are not well understood but it is hypothesized that two factors play a crucial role: sensation seeking and reduced inhibitory control (Rahman, Sahakian, Cardinal, Rogers, & Robbins, 2001; Roberti, 2004). Sensation seeking is largely driven by dopamine and serotonergic systems that promote exploratory behavior in new environments. People with high levels of risk behaviors seem to be neurologically less sensitive to stress or threat signals. In general, among those with high levels of impulsivity, men tend to drink more alcohol in response to stress than women. Alcohol consumption is important to study since it has a major effect both on health and on the impact of chronic illness (Rehm et al., 2003).

High alcohol consumption is associated with being overweight and obese but the associations are complex (Suter & Tremblay, 2005). First, since alcohol is energy-dense, consuming it can easily result in a large energy surplus. This effect is enhanced by the absence of a physiological control mechanism for this kind of energy. As discussed above, energy consumption is otherwise governed by metabolic systems that regulate feeding behavior. Alcohol does not seem to affect these systems much, making overconsump-

tion of energy from alcohol more likely than from foodstuffs. Second, alcohol consumption triggers appetite, especially for fatty foods, and reduces feelings of satiety. Third, it has been suggested that alcohol promotes abdominal fat deposition, an important risk factor for many diseases. Which of these effects is most important is still debated but alcohol consumption is evidently an important factor in obesity and its treatment.

Bariatric surgery patients report high levels of alcohol risk consumption and high prevalence of lifetime alcohol disturbance before surgery, especially when anonymous data collection is employed (Heinberg, Ashton, & Coughlin, 2012). There are signs that the risk of alcohol disturbance increases after bariatric surgery with RYGBP, in both the short and the long term (Ertelt et al., 2008; King et al., 2012). This is probably due both to pharmacokinetic changes, such as accelerated alcohol absorption, and to difficulties in changing drinking habits after surgery. It is important to note that the variance in alcohol consumption is large and that many patients abstain from alcohol after surgery. Nevertheless, a small group of individuals seem to be at risk for elevated alcohol consumption after surgery. It has been difficult to find consistent predictor variables to identify these individuals prior to surgery. Given the associations among impaired executive function, risk behaviors and alcohol consumption, all these variables may be important to study.

Impaired executive function

Impaired executive function may be seen in people with frontal lobe injuries but also in people with ADHD or depression, in all cases, changes in the prefrontal cortex are evident (Alvarez & Emory, 2006; Arnsten & Li, 2005; Paelecke-Habermann, et al., 2005). Perhaps more surprisingly, medical illnesses such as hypertension or diabetes also seem to affect executive function (Schillerstrom, Horton, & Royall, 2005). While most medical effects on executive function are probably mild, this finding could still have important implications for these patients. Chronic illness typically entails treatment regimens or recommendations, such as daily medication, that depend on planning skills and high functioning memory. Adherence to medication shows large variance and is sometimes very low in people with chronic diseases (Dunbar-Jacob & Mortimer-Stephens, 2001). The reasons for low adherence to prescriptions have been investigated in various populations and a number of factors such as side effects and complexity of medication schedules, may be involved. Two other factors that has been found to influence adherence are executive- and cognitive function (Stilley, Bender, Dunbar-Jacob, Sereika, & Ryan, 2010; Williams & Thayer, 2009).

Deficits in executive function are among the core features of adult ADHD and may hamper these patients' ability to follow medical prescriptions for these patients (Adler & Nierenberg, 2010). Even in people without cognitive difficulties, differences in executive function may partly explain why some

show low adherence to health recommendations such as engaging in physical exercise, (Hall, Fong, Epp, & Elias, 2008). Knowledge of healthy behaviors, as well as the intention to practice them, may be moderated by the ability to carry out such goal-directed behaviors effectively. Inferior executive function may also be related to consumption of unhealthy foodstuffs (Hall, 2012). Treatments for obesity, both surgical and behavioral, involve fairly strict behavioral prescriptions that require great efforts to follow. Whether people with executive dysfunction and/or ADHD may have more difficulties in following diet regimens such as those recommended after bariatric surgery has not yet been investigated empirically (Galioto, Gunstad, Heinberg, & Spitznagel, 2013). This may be important since patients seeking bariatric surgery show higher levels of executive dysfunction than healthy controls in terms of specific cognitive abilities such as planning skills (Lokken, Boeka, Yellumahanthi, Wesley, & Clements, 2010). Although ADHD in the obese population has been studied previously, ADHD symptoms among bariatric surgery candidates have only recently attracted any attention.

ADHD and obesity

Adult ADHD is more common among people with obesity than people of normal weight (Pagoto et al., 2009) and many also show symptoms of adult ADHD without meeting the diagnostic criteria (Altfas, 2002). Further, symptoms of ADHD are associated with inferior weight outcomes in behavioral treatment for obesity, although the reasons are unknown. It is also unclear how adult ADHD and obesity interact and whether there are common genetic or physiological risk factors (Cortese & Vincenzi, 2012). On a physiological level, it has been proposed that changes in the dopamine reward system may be a common factor in both ADHD and obesity. On a behavioral level, it has been hypothesized that abnormal eating behaviors or obesity-related sleeping problems may present as attention deficits. While ADHD and obesity are clearly associated, the exact mechanisms and possible causal relationships remain unclear. To further complicate the picture, ADHD and disordered eating often present differently in men and women at different ages.

Gender, age, BED and adult ADHD

Both BED and adult ADHD are diagnoses with marked gender differences. Concerning BED, the disorder is more common in women (3-4%) than in men (about 2%) (Hudson, et al., 2007). The symptom profiles are also slightly different, with women reporting higher levels of body image dissatisfaction and drive for thinness than men (D. T. Barry, Grilo, & Masheb, 2002). Men have been found to show the same levels of eating disorder symptoms as women while reporting less shame and guilt for these behaviors

(Striegel-Moore & Franko, 2003). If this is true, when men and women report similar behaviors, women, but not men, may be conceptualized as having an eating disorder (Grilo & Masheb, 2005). In the context of bariatric surgery this may be important, since it is unknown what aspects of disordered eating may constitute risk factors for weight loss after surgery: weight and shape concerns or binge eating. Ideally, both disordered eating behaviors and psychological symptoms of eating disorders, given the potential effect of gender, should be assessed among bariatric surgery patients to better analyze and understand these variables.

As mentioned above, adult ADHD is more common in men than women (Kessler et al., 2006). The gender difference in prevalence is much smaller in adult ADHD than childhood ADHD. This is largely an effect of declining hyperactive- and disruptive behaviors among men with advancing age. However, patterns of psychiatric comorbidity are somewhat different, with men more often having conduct problems and drug abuse and women more often having eating disorders (Biederman, Faraone, Monuteaux, Bober, & Cadogen, 2004; Rasmussen & Levander, 2009). The reasons for these differences are not known but they follow the overall gender pattern for the normal population.

While the effect of age on ADHD hyperactivity symptoms is well known, relatively little is known about how age affects eating disorders. It seems that the prevalence of BED rises with age (Johnson, Spitzer, & Williams, 2001). Some people reporting BED at a higher age have probably previously had Bulimia Nervosa, which typically has an earlier debut than BED. This would indicate that eating disorder symptoms may change over time, something that has not been studied to any degree (Patrick & Stahl, 2009). At all ages, women report more eating disorder symptoms than men but they may also improve in terms of such variables with rising age (Keel, Baxter, Heatherton, & Joiner Jr, 2007). Gender and age may thus both moderate binge eating and be important variables to consider in analyses of weight loss.

Methodological considerations

The psychological- or behavioral risk factors investigated in bariatric surgery patients are associated in complex patterns. Taken together, the results from previous studies of risk factors in bariatric surgery have been mixed, indicating the need for some methodological reconsideration. There may for example be confounding variables or unknown associations among variables. There may also be unknown moderation effects from demographic variables.

Analysis of moderation

Moderation occurs when the level of one independent variable influences the relationship between another independent variable and a dependent one (Hayes, 2009). Statistically, moderation can be viewed as a case of mediation and there has been some confusion how to specify and separate the two concepts (MacKinnon, 2008). There have been several suggestions how to refine the two concepts to make them meaningful research tools. McKinnon (2008) concludes that for a moderator variable to be usable and interpretable it must be independent of the independent variable studied (e.g. the intervention) and should ideally be on a categorical scale. To be independent, the moderator variable should be relatively stable over time and unaffected by the intervention. Demographic and personality variables such as age, gender or impulsivity are thus suitable moderator variables. As mentioned above, gender and age may moderate how eating disorders and adult ADHD present. While disordered eating and impulsivity may be important factors in bariatric surgery patients, the moderating effects of gender and age have not been investigated in this population to any extent.

Summary

Low control over eating behavior is an important problem in both bariatric surgery and eating disorders. Why some patients do not achieve weight loss as expected after bariatric surgery is intriguing. Several psychological risk factors have been proposed and investigated but results have been inconclusive. To further our understanding we could investigate new unstudied variables that may affect adherence and weight outcome. We could also apply new analytic methods that can potentially reveal unknown associations among the variables under study.

One way to find new, unstudied variables that could affect weight outcome after surgery is to reconceptualize potential risk factors in order to identify common features at a higher level. Risk factors such as depressive symptoms or binge eating may not have a specific effect on weight loss but rather affect general function and ability to control eating behavior and follow strict regimens. Although inferior weight loss is often associated with psychosocial variables, causal mechanisms have not been confirmed. Interestingly, this is true not only of disordered eating, which arguably is closely associated with eating and weight, but also of personality disorders which should only affect eating and weight indirectly. This would mean that weight loss can be affected through different mechanisms or that all variables affecting general function and the ability to follow strict behavioral recommendations are important to consider. The ability to follow recommendations is largely based on executive function, which is impaired both in patients with

depression and in patients with adult ADHD. While depression has been studied previously, ADHD symptoms among bariatric surgery patients have not been investigated to any extent.

The conflicting results regarding risk factors may be due partly to moderating variables that affect the association between risk factors and weight loss in some, but not all, patients. Disordered eating presents differently in men and women and gender is thus one such potential moderating variable. The association between risk factors and weight outcome may be gender-specific, but this has not yet been thoroughly investigated.

The association between binge eating and negative mood is well established but the temporal and causal mechanisms are largely unclear. While affect regulation seems to play a role in the association between mood and control over eating, it has been difficult to show the operant mechanisms that maintain binge eating. Treating binge eating is often associated with improved mood but the opposite is not as clearly the case. Solely focusing on improving patients' mood may thus not be a feasible way to affect binge eating. A common strategy, derived from learning theory, for trying to change persistent behavior is not to focus on the problematic behavior but instead positively reinforce other, incompatible, behaviors. This is known as discriminative reinforcement of other behavior (DRO). While the reinforcement used can be arbitrary, to promote sustained behavior change the reinforcement should be intrinsic to the new behavior. One treatment that focuses on increasing behaviors with intrinsic positive reinforcement is Behavioral Activation. A treatment program with BA could potentially help patients with BED to engage in new positively reinforced behaviors and reduce their binge eating. While mood may also be improved, the mechanism would be reinforcement of behaviors incompatible with binge eating. Though BA has been used in various patient groups, including obese patients, it has not yet been investigated as a treatment for BED.

Aims of the thesis

The overall aim of this thesis was to investigate prevalence of, and clarify the complex associations between, variables that may affect control over eating in bariatric surgery patients.

This thesis includes three studies that aimed to clarify relationships between symptoms of ADHD and negative mood on the one hand, and disordered eating and weight loss on the other. To this end, three samples were recruited from patients at an obesity clinic. The aim of the first study was to estimate the prevalence of symptoms of adult ADHD and assess the associations among symptoms of ADHD, disordered eating, anxiety and depression. The second study aimed at evaluating Behavioral Activation as a treatment for Binge Eating Disorder among the obese. This was done by investigating whether an increased level of activation would reduce binge eating and eating disorder symptoms as well as elevate mood in patients with obesity and BED. The third study was a prospective study with the aim of assessing whether symptoms of anxiety, depression, adult ADHD, disordered eating and alcohol consumption constitute risk factors for low weight loss after bariatric surgery. A further goal was to assess moderation effects of age and gender on these risk factors.

The Empirical Studies

Study I: Screening of Adult ADHD among Patients Presenting for Bariatric Surgery

Introduction and aims

After bariatric surgery, adherence to dietary and behavioral prescriptions, such as physical exercise, is often low and this may affect weight loss (Elkins et al., 2005; Toussi, et al., 2009). Whether psychiatric problems are directly associated with adherence has not been studied to any extent among bariatric surgery patients. From research on depression we know that depressive symptoms are associated with low compliance with medication and recommended health behaviors (DiMatteo, et al., 2000). This suggests a behavioral association between depressive symptoms that affect every day function and weight loss after surgery. Another psychiatric problem associated with low levels of treatment adherence and high levels of disruption in daily life is adult Attention Deficits/Hyperactivity Disorder (ADHD) (Goodman, 2007).

While the prevalence of adult ADHD seems to be elevated in the obese population, there has been no investigation among patients of bariatric surgery. ADHD is a developmental disorder characterized by concentration difficulties, impulsivity and restlessness. For some young people with ADHD the symptoms decline with advancing age, but for many the symptoms persist into adulthood. However, the symptom profile typically changes where the hyperactive or impulsive symptoms are mitigated while the deficits in attention remain (Davidson, 2008). Adult ADHD may thus be less noticeable and many adults affected probably go undiagnosed. At the same time, adult ADHD is associated with both obesity and with several negative health behaviors such as smoking and drug abuse (C Davis, 2009).

Since noncompliance with recommendations after bariatric surgery is common, it is important to find factors that may influence patients' ability to follow strict behavioral regimens. Adult ADHD is one such factor that may potentially affect adherence after surgery. The prevalence of adult ADHD and its associations with disordered eating and depressive symptoms have not yet been investigated among bariatric surgery patients. The aim of this study was to assess the prevalence of adult ADHD symptoms and elucidate the association with other common psychological risk factors in this population.

Methods

Participants

Over five months, 217 patients at an obesity clinic were approached and informed about the study. Of these, 187 chose to participate and completed a set of self-report instruments. The majority (73.3%) were women, their mean age was 41.0 ($SD = 6.02$) years and the mean BMI was 44.3 ($SD = 6.02$).

Procedure

The study protocol was approved by the regional ethics committee board. Patients were informed about the study on their second visit to the clinic, after initial medical screening. The instruments of this study were thus completed 12-16 weeks before surgery by patients eligible for surgery. All the instruments were mailed to each patient prior to the second visit with information that the results would not be used to exclude anyone from examination or future surgery. This was done to minimize the risk of any patient withholding information because of worry about being denied surgery.

Instruments

Symptoms of adult ADHD were measured with the ADHD Self-Report Scale (ASRS; Kessler et al., 2005). The ASRS has two subscales corresponding to the two main symptom criteria for ADHD, Attention deficits and Hyperactivity/Impulsivity. The first six items of the ASRS can be used as a screening instrument for adult ADHD (ASRS-S). Symptoms of anxiety and depression were measured with the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) which has two subscales, Anxiety and Depression. The score on each scale can be used as a screening for psychiatric relevant problems with a cut off score > 7 for low risk and a score > 10 for high risk for present problems. The Eating Disorders in Obesity Questionnaire (EDO; De Man, Ghaderi, Halvarsson-Edlund, & Norring, 2007) was used as a screening instrument for eating disorders. The EDO is short and well suited for screening in a clinical population where the prevalence of eating disorders may be low. The EDO was extended with an additional section, created for this study, where participants were asked to report all Binge Eating Disorder (BED) symptoms, regardless of whether they screened positive for an eating disorder or not. Finally, the General Food Cravings Questionnaire Trait version (GFCQT; Nijs, Franken, & Muris, 2007) was used as a general measure of disordered eating. Both the total score on the GFCQT and two of the subscales, Loss of control and Emotional eating, were analyzed.

Statistical analysis

Pearson's *r* was used to analyze correlations between continuous variables. Differences between nominal and ordinal variables were tested using χ^2 and Kruskal-Wallis tests. Differences in continuous variables between groups were analyzed with Analysis of variance (ANOVA) and stepwise regression was used to investigate predictors of disordered eating. Cohen's *d* was used as a measure of effect size.

Results

According to the ASRS-S, 10.2% of the participants screened positive for adult ADHD. There were no differences on demographic variables between participants screening positive and negative for adult ADHD. Further, 7.5% screened positive for an anxiety disorder and 7.5% for depression, according to the HADS. Finally, 8.2% screened positive for BED according to the EDO. In the additional section of the EDO, 42.2% reported objective over-eating and 36.8% reported eating large amounts of food when not physically hungry. Large minorities reported the typical BED symptoms of losing control over eating (31.0%) or feeling disgusted, depressed or guilty after over-eating (26.1%). See Table 1 for the prevalence of all BED symptoms. Women scored significantly higher ($F(1,185) = 4.60$, $p = .033$, $d = 0.35$) than men on the Emotional cravings subscale of the GFCQT and women also reported more frequently eating alone because of being embarrassed by how much one is eating, than men ($\chi^2(3) = 11.09$, $p = .011$).

Table 1. *Number of participants reporting at least weekly episodes of each criterion for BED in DSM-IV (N = 187).*

	n (%)
Eating, in a discrete period of time, an amount of food that is definitely larger than most people would eat in a similar period under similar circumstances	78 (42.2)
A sense of lack of control over eating during eating	57 (31.0)
Eating until feeling uncomfortably full	68 (26.3)
Eating much more rapidly than normal	28 (15.1)
Eating large amounts of food when not feeling physically hungry	68 (36.8)
Eating alone because of being embarrassed by how much one is eating	49 (26.5)
Feeling disgusted, depressed, or guilty after overeating	48 (26.1)
Constantly eating snacks between meals[a]	85 (46.2)
Night eating[a]	16 (8.6)

[a] Additional items not included in the research criteria for BED in DSM-IV.

Symptoms of anxiety and depression, symptoms of adult ADHD and symptoms of disordered eating were all significantly moderately intercorrelated (r = .33 - .58). Participants who screened positive for adult ADHD had significantly higher scores on the HADS Anxiety ($F(1,185) = 10.14$, $p = .002$, $d = 0.75$), HADS Depression ($F(1,185) = 16.00$, $p < .001$, $d = 0.93$) as well as the GFCQT ($F(1,185) = 16.00$, $p < .001$, $d = 0.93$), see Table 2. They also reported higher prevalence of lack of control over eating and other symptoms of disordered eating in the extended section of the EDO.

Table 2. *Comparison of participants screening negative with those screening positive for Adult ADHD.*

| | ASRS-S | | | | |
	Negative (*n* = 168) *m* (*SD*)	Positive (*n* = 19) *m* (*SD*)	*F*	*p*	Cohen's *d*
HADS Anxiety	4.38 (3.78)	7.26 (3.43)	10.14	.002	.75
HADS Depression	4.02 (3.54)	7.47 (3.88)	16.00	.001	.93
GFCQT	48.06 (21.10)	68.79 (23.58)	16.00	.001	.93
Loss of Control	2.64 (1.29)	3.63 (1.27)	15.54	.001	.75
Emotional Cravings	2.07 (1.47)	3.32 (1.66)	18.12	.002	.99

Note. ASRS-S = Adult ADHD Self-Report Scale Screener, HADS = Hospital Anxiety and Depression Scale, GFCQT = General Food Cravings Questionnaire Trait.

Discussion

The aim of this study was to investigate the prevalence and associations between symptoms of ADHD and other potential psychological risk factors among bariatric surgery patients. The results show that symptoms of adult ADHD are common and that they are positively correlated with symptoms of anxiety and depression as well as symptoms of disordered eating. However, participants who screened positive for adult ADHD did not screen positive for BED to any higher degree than participants who screened negative for adult ADHD. This indicates that some patients may have had problems with disordered eating, as well as comorbid psychiatric problems, without fulfilling the diagnostic criteria for a formal eating disorder.

The measured levels of anxiety and depressive symptoms in this study were comparable to those in other European studies indicating that self-report assessment is feasible in this population (Karlsson, Taft, Rydén, Sjöström, & Sullivan, 2007). In previous studies, the reported prevalence of adult ADHD range from about 4% in the general population to 27% in the obese population (Altfas, 2002; Simon, et al., 2009). In the present study, the prevalence of 10.2% is in the middle range but close to the prevalence (12%) reported in a recent German study of the same population (Gruss, Mueller, Horbach, Martin, & Zwaan, 2012). The prevalence of BED has ranged from 7% to 24% in different studies depending on population and methods of assessment (De Zwaan et al., 2003; Grucza, Przybeck, & Cloninger, 2007; Sarwer et al., 2004). Unfortunately, screening for BED seems to be highly

sensitive to which method of assessment is used and there is no gold standard for assessing BED in the general population. The sole use of self-report instruments may be a weakness of the current study in this respect. On the other hand, patients may feel more comfortable reporting psychiatric problems in a more anonymous way since there is a, largely uncalled for, worry among some patients that psychiatric problems may be a reason to be denied bariatric surgery. One strength of the current study was that several steps were taken to assure patients that results from the study would be used only in research.

The results show that a sizable minority of patients report disordered eating and symptoms of ADHD, anxiety and depression. While many patients may be helped by established treatment protocols with CBT for BED, it may be possible to target these problems more effectively with a treatment that focuses on establishing alternative behaviors to binge eating through structured planning. This may help patients with both negative mood and high impulsivity who find it difficult to control their eating.

It is noteworthy that participants in this study reported higher than typical levels of education which may partly explain the moderate levels of psychiatric problems found. Moreover, data were collected at patients' second visit to the clinic, when some patients had already been excluded from further preparation for surgery. While this may limit the generalizability of the results, it also makes the sample more representative of patients who actually undergo surgery. Nonetheless, large minorities reported one or more symptoms of disordered eating, showing that these are common problems during the preparation for surgery.

Previous studies regarding risk factors for inferior weight loss after bariatric surgery show mixed results. Why some patients do not lose as much weight as expected remains unknown, but one major risk factor may be difficulties in following the strict postoperative regimens. Prior to surgery, identifying patients who may have problems in following behavioral prescriptions in the long run seems difficult. Patients with psychiatric problems must overcome everyday difficulties and find new coping strategies after surgery. People with psychiatric comorbidities or functional handicaps such as neuropsychiatric disorders may thus need closer support and follow-up care after surgery than other patients.

This study shows that bariatric surgery patients have elevated levels of adult ADHD and that symptoms of ADHD are associated with symptoms of disordered eating, as well as symptoms of anxiety and depression. How these variables interact and affects the ability to follow postoperative regimens needs further study. Future prospective studies should continue to broadly investigate potential risk factors for low weight loss after surgery in order to develop both better assessment guidelines and follow-up care. Further, new treatments for binge eating that are suited for patients with high impulsivity as well as low mood may be effective alternatives to established protocols.

Study II: Group Behavioral Activation for Patients with Severe Obesity and Binge Eating Disorder: A Randomized Controlled Trial

Introduction and aims

There are two empirically supported treatments for BED, each based on a distinct theoretical model and treatment approach toward BED. In the model underlying Cognitive Behavior Therapy (CBT) for eating disorders, overvaluation of weight and shape is the core feature of the disorder (Goldschmidt et al., 2010). An overvaluation of weight and shape for self-worth is manifested as inflexible restraint on eating and strict dietary rules as well as excessive social comparisons and self-blame. Inflexible restraint on eating combined with negative affect ultimately results in behavioral disinhibition and binge eating, followed by renewed restraint and self-blame. CBT for BED consequently focuses on promoting more flexible dietary rules, regular eating patterns and also, to some extent, strategies for affect regulation (Blomquist & Grilo, 2011; Fairburn, 2008). This is in contrast to Interpersonal Therapy (IPT) which does not directly target eating habits at all.

In the IPT model for eating disorders, dysfunctional social strategies or interpersonal difficulties lead to binge eating and other psychological problems (Murphy, et al., 2012). According to this model, maladaptive social behaviors, such as exaggerated appeasement or self-deprecation, lead to low self-esteem and shame. These emotions in turn result to binge eating and treatment with IPT therefore focuses on improving social strategies and social role function (Rieger, et al., 2010).

IPT developed as a treatment for depression and is only to a limited extent adapted for the context of eating disorders. While Cognitive Therapy is the most empirically evaluated treatment for depression, its crucial component appears to be Behavioral Activation (BA). Several studies have confirmed BA as a standalone treatment for depression (Sturmey, 2009). The BA model for depression states that people with depression have lost contact with natural sources of intrinsic positive reinforcement and emphasize reconnection to such sources through increased behavioral activation (Martell, et al., 2001). Reducing avoidant behaviors and increasing positive reinforcement through engagement in behaviors that are incompatible with binge eating may, in both CBT and IPT, lead to reduced binge eating in patients with BED. The

mechanisms of IPT and CBT for BED probably overlap and a raised level of engagement in rewarding activities, social or other, may be one of the factors affecting binge eating in both treatments. There has been no study specifically investigating this possible mechanism in patients with BED.

The aim of this study was to examine whether treatment with BA can be effective in reducing binge eating in patients with obesity and BED. The main hypothesis was that participants receiving BA would report decreased binge eating and eating disorder symptomatology as well as enhanced levels of positive activity and improved mood.

Materials and methods

Design

This study was a randomized controlled trial with a wait-list control group conducted in a clinical context. With an expected attrition rate of 10%, a minimum of 50 participants were needed in each group to obtain a power of 80% with a p-threshold of .05 and between group effect sizes of Cohen's $d = 0.65$.

Participants

Participants for the study were recruited at an obesity clinic offering both behavioral treatments and obesity surgery. As part of the clinic's routine care, all patients underwent an initial medical examination and were screened for eating disorders. Every patient who met the DSM-V criteria for BED was eligible for participation (American Psychiatric Association, 2013). Exclusion criteria were severe mental disorder or current alcohol- or drug abuse. A total of 142 patients were assessed for participation and 100 proved eligible for inclusion. Thirty-two of the participants reported stable antidepressant medication and all participants were asked to report any changes in medication during the study period.

Procedure

Before randomization to either BA or control, participants were asked to complete all the instruments of the study. Participants randomized to BA started the treatment within two weeks after randomization and were assessed at the end of treatment and at six month follow-up. Participants in the control group were assessed after 12 weeks and then received BA. For ethical reasons all participants had to be offered active treatment, and follow-up data were thus unavailable for the control group. The study was approved by the regional ethics committee.

Treatment

The BA treatment consisted of group therapy with four to seven (*m*=5.0) participants in each group. Each group met for 10 weekly sessions of 90 minutes each. The treatment protocol was an adaptation of the BA-programs by Addis & Martell and Kanter, Busch & Rusch (Addis & Martell, 2004; Kanter, Busch, & Rusch, 2009). The BA program included psychoeducation, planned activation, identification of avoidance behaviors, thought-action diffusion, problem solving, social activation and goal setting. The overall aim was to increase participants' engagement in meaningful activities. This meant scheduling everyday activities they perceived as positive and meaningful in the long term. Homework assignments were given after every session. The treatment protocol was highly structured, with detailed instructions for all sessions. Dieting or eating habits were not discussed during treatment. The treatment was provided by two psychologists at the clinic but each participant also made one visit to a physician and one or two visits to a dietitian as part of the clinic's routine care.

Measurements

A structural clinical interview, the Eating Disorder Examination (EDE; Z. Cooper & Fairburn, 1987), was used to assess BED diagnosis at inclusion and at post treatment. Eating disorder symptomology was measured with the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994). The EDE-Q has four subscales; Restraint, Eating Concerns, Shape Concerns and Weight concerns and a total score. Number of binge eating episodes was also assessed with the EDE-Q.

Symptoms of anxiety and depression were measured with the HADS (Zigmond & Snaith, 1983). Social function was measured with the Modified Social Adjustment Scale (SAS-M; P. Cooper, Osborn, Gath, & Feggetter, 1982) comprising seven subscales; Work, Housework, Social and leisure activities, Extended family, Marital, Parental and Family unit. Activity levels were measured with the Environmental Reward Observation Scale (EROS; Armento & Hopko, 2007) and the Behavioral Activation in Depression Scale (BADS; Kanter, Mulick, Busch, Berlin, & Martell, 2007), two instruments specifically developed to measure the construct of positive activity in BA treatments. The BADS has four subscales; Activation, Avoidance/Rumination, Work/School impairment and Social impairment, while the EROS has only a total score. Finally, regular patterns of eating were measured with the Eating Patterns questionnaire, an instrument created for this study.

Statistical analysis

Missing data on single items were imputed using expectation-maximization estimates. Normality of data was investigated before further analysis and judged adequate. Chi^2 and one-way ANOVAs were used to investigate differences in background variables between the two groups at pretreatment measurement. A mixed-effects model for repeated measures (MMRM) was used to investigate treatment effects, using an intent to treat sample with two levels between (Treatment and Control) and two within (Pre and Post) levels. An MMRM with three levels (Post, 3 months and 6 months) was used to analyze long-term effects in the treatment group. The analyses were conducted using different covariance structures, starting with unstructured, followed by Toeplitz, first-order autoregressive AR(1), compound symmetry and heterogeneous compound symmetry covariant structures. The model with the fewest parameters was reported unless there was a significant difference in fit, determined by comparison of the restricted log-likelihood. A p-value of .05 was used as a threshold for significant difference in all analyses and Cohen's d was used as a measure of effect size.

Results

During the study, dropout was significantly ($p = .01$) larger from the BA group ($n = 16$, 16.7%) than from the control group ($n = 8$, 8.3%). Posttreatment data were available from 34 participants from the BA group and 38 participants from the control group. The mean BMI among all participants was 41.2 ($SD = 5.32$) and there was no significant change in BMI in either group during treatment. At pre treatment, there were no significant differences between the groups on any background variable. However, there was a difference on the Social and leisure activities subscale of the SAS-M, so this variable was entered as a covariate in each analysis. There were no differences on background variables between participants who dropped out from the study and the others. See Table 3 for all outcome variables at pre and post measurements.

   At the post treatment assessment, 10 (29%) participants in the BA group and 10 (26%) in the control group no longer fulfilled the criteria for BED. There was no time x group interaction effect on the Objective binge eating episodes items of either the EDE or the EDE-Q. The only treatment effect on eating disorder symptoms was a significant ($F(1,78.314) = 6.28$, $p = .014$, $d = 0.51$) time x group interaction effect on the Weight concerns subscale of the EDE-Q. There was however a significant ($F(1,77.441) = 13.90$, $p < .001$, $d = 0.76$) time x group interaction effect on the HADS Depression subscale. There were significant treatment effects on several of the secondary outcome

variables, including the EROS. See Table 4 for all comparisons on outcome variables.

Table 3. *Means and standard deviations on the dependent variables for the treatment and control groups at pre and post assessment (N = 96).*

	BA group		Control group	
	Pre (*n* = 50) *M* (*SD*)	Post (*n* = 34) *M* (*SD*)	Pre (*n* = 46) *M* (*SD*)	Post (*n* = 38) *M* (*SD*)
EDE				
OBE days	9.60 (7.06)	7.35 (7.03)	9.43 (7.11)	9.29 (9.06)
EDE-Q				
Restraint	1.64 (1.36)	1.96 (0.98)	1.47 (1.34)	1.15 (1.09)
Eating concern	2.91 (1.58)	2.49 (1.59)	2.76 (1.50)	2.40 (1.65)
Shape concern	4.69 (0.99)	4.26 (1.21)	4.69 (1.06)	4.41 (1.18)
Weight concern	3.96 (0.90)	3.45 (0.91)	3.78 (0.90)	3.84 (0.95)
Total score	3.30 (0.91)	3.04 (0.84)	3.17 (0.79)	2.95 (0.97)
OBE days	11.32 (9.28)	8.24 (8.86)	12.63 (10.24)	10.89 (10.16)
Eating Patterns	24.40 (6.48)	26.21 (6.00)	26.74 (6.78)	26.94 (5.23)
HADS				
Anxiety	7.88 (3.56)	6.91 (3.94)	6.78 (3.99)	7.00 (3.10)
Depression	8.67 (4.50)	5.71 (3.83)	7.72 (4.91)	8.05 (5.29)
SAS-M				
Work	4.00 (0.71)	4.06 (0.55)	3.71 (0.63)	3.88 (0.75)
Housework	3.35 (0.65)	3.53 (0.68)	3.52 (0.67)	3.43 (0.70)
Social	3.32 (0.51)	3.60 (0.67)	3.57 (0.55)	3.46 (0.65)
Extended fam.	3.67 (0.54)	3.99 (0.42)	3.72 (0.69)	3.65 (0.66)
Marital	3.21 (0.67)	3.40 (0.71)	3.35 (0.69)	3.29 (0.87)
Parental	4.14 (0.72)	4.08 (0.76)	3.85 (0.93)	4.11 (0.71)
Family unit	4.10 (0.77)	4.26 (0.71)	3.56 (1.76)	4.33 (0.46)
EROS	13.62 (5.27)	17.45 (5.33)	15.67 (6.21)	14.89 (4.63)

Note. EDE = Eating Disorder Examination, OBE = Objective Binge Eating, EDE-Q = Eating Disorder Examination Questionnaire, HADS = Hospital Anxiety and Depression Scale, SAS-M = Modified Social Adjustment Scale, Social = Social and leisure activities, Extended fam. = Extended family, EROS = Environmental Reward Observation Scale.

Table 4. *Pre- and post treatment results from the MMRM analysis displaying group, time, interaction effects and effect size (N = 96).*

	Group effect		Time effect		Group x Time effect		
	F	p	F	p	F	p	ES^5
EDE							
OBE days[b]	0.68	.41	3.21	.077	2.36	.13	0.31
EDE-Q							
Restraint[c]	3.92	.051	0.01	.91	3.65	.060	0.39
Eating concern[b]	0.01	.91	**6.48**	**.013**	0.51	.48	0.16
Shape concern[b]	0.27	.61	**6.11**	**.016**	0.58	.46	0.17
Weight concern[b]	0.58	.45	**4.47**	**.038**	**6.28**	**.014**	**0.51**
Total score[b]	0.14	.71	**6.38**	**.014**	0.40	.53	0.14
OBE days[a]	1.25	.27	**7.62**	**.007**	0.48	.49	0.16
Eating Patterns[c]	1.69	.18	2.07	.15	1.58	.21	0.28
HADS							
Anxiety[c]	0.16	.69	1.81	.18	**4.93**	**.029**	**0.45**
Depression[b]	0.57	.45	**14.24**	**.001**	**13.90**	**.001**	**0.76**
SAS-M							
Work[a]	3.67	.06	0.90	.35	0.24	.63	0.11
Housework[c]	0.04	.84	0.18	.68	2.09	.15	0.33
Social and leisure activities[b]	0.07	.77	1.84	.18	**6.46**	**.013**	**0.58**
Extended family[b]	1.36	.25	**5.16**	**.026**	**6.66**	**.012**	**0.53**
Marital[c]	0.06	.82	2.25	.14	**6.42**	**.017**	**0.52**
Parental[b]	0.10	.32	0.31	.59	0.16	.69	0.09
Family unit[b]	**4.87**	**.032**	0.58	.45	2.99	.09	0.39
EROS[a]	0.12	.73	**13.51**	**.001**	**15.45**	**.001**	**0.80**

Note. EDE = Eating Disorder Examination, OBE = Objective Binge Eating, EDE-Q = Eating Disorder Examination Questionnaire, HADS = Hospital Anxiety and Depression Scale, SAS-M = Social Adjustment Scale Modified, EROS = Environmental Reward Observation Scale. ES = Effect size (Cohen's *d*).
[a] = Unstructured, [b] = Toeplitz and [c] = Heterogeneous compound symmetry covariance structures.

The changes in outcome variables in the BA group were sustained at the 3- and 6 months follow-up measurements. There was a significant ($F(1,98.832) = 6.68$, $p < .001$, $d = 0.89$) main effect of time on Objective binge eating episodes from post treatment assessment to the two follow up measurements indicating that binge eating continued to decline after treatment. The participants in the BA group completed the BADS on three occasions during treatment and there was a significant effect on time on the total score as well as on three of the subscales, giving some support to treatment fidelity, see Table 5.

Table 5. *Mean score on the Behavioral Activation for Depression Scale in the BA group at pre- mid- and post measurement, results from the MMRM analysis and effects sizes (N=50).*

	Pre $n = 34$ m (SD)	Mid $n = 34$ m (SD)	Post $n = 34$ m (SD)	F	p	ES
Total score[b]	79.80 (22.24)	88.15 (19.59)	96.66 (20.15)	**10.51**	**.001**	**1.00**
Activation[b]	16.18 (8.69)	17.77 (7.54)	20.32 (6.79)	**6.24**	**.004**	**0.67**
Avoidance[c]	21.26 (9.28)	18.00 (9.38)	15.37 (9.23)	**9.01**	**.001**	**0.94**
Work/School Imp.[a]	15.08 (6.64)	12.92 (6.55)	10.45 (5.99)	**8.44**	**.001**	**0.87**
Social Imp.[c]	8.05 (6.20)	6.69 (5.62)	5.84 (5.55)	3.51	.067	0.54

Note. Work/School Imp. = Work School Impairment, Social Imp. = Social Impairment, ES = Effect size (Cohen's *d*).
[a] = Unstructured, [b] = Toeplitz and [c] = Heterogeneous compound symmetry covariance structures.

Discussion

In this study, participants in both groups reported fewer binge-eating episodes and improvement in other eating disorder symptoms at posttreatment measurement compared with premeasurement. There was thus no specific treatment effect and the results may be due to common factors for both groups, such as expectancy effects or the general clinical care each patient received. There was a specific treatment effect on the Weight concerns subscale of the EDE-Q which may have been due to the normalizing effect of

being part of a group, but this is purely speculative. The main conclusion must be that BA has no specific effect on binge eating symptomatology.

The BA group did, however, show improvements on the measures of depression and positive activation. The effect sizes were medium to large, confirming that BA is an effective treatment for increasing activation and improving mood. Participants in the BA group increased in activation and improved in mood but not in binge eating symptoms, indicating that the association among these factors is complex. That improved mood would not in itself ameliorate binge eating was expected. The alternative hypothesis, that increased activation would replace binge eating with incompatible behaviors, was not supported.

This study suffered from several limitations. There was a fairly large dropout from the study, especially in the treatment group. This dropout occurred mainly in the two last sessions of the treatment program for reasons that, unfortunately, were not investigated. Assessment of participants was conducted by nonblinded interviewers although this was ameliorated to some extent by the use of self-report questionnaires. The fidelity of the treatment was not possible to measure formally but the treatment effect on both instruments that intended to measure activation increased significantly, implying that BA was provided in accordance with the original protocol. Finally, there was no long-term follow-up of the control group. This was due to ethical reasons and the demands of the clinical setting in which the study was conducted. Patients at the clinic must be offered active treatment and delayed treatment was the only option possible for participants randomized to the control condition.

In summary, the main hypothesis of this study, that increased positive activation would improve both mood and binge eating symptomatology, was not supported. Though participants in the BA group reported increased activation and alleviation of depressive symptoms that were sustained over time, there was no treatment effect on binge-eating episodes. The associations between mood and binge eating and the mechanisms in behavioral treatments for BED are thus still elusive. Future studies need to be conducted in a more controlled setting and may investigate various treatment components, such as activation and flexible restraint, simultaneously to provide a better understanding of the mechanisms of change.

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Study III: Is age a better predictor of weight loss one year after Gastric bypass than symptoms of disordered eating, depression, adult ADHD, and alcohol consumption?

Introduction

Patients who report binge eating and other eating disorder symptoms after bariatric surgery often show relatively poor weight outcomes (White, Kalarchian, Masheb, Marcus, & Grilo, 2010). For many patients, disordered eating improves after surgery but why these problems persist for some patients remains unknown (Wadden et al., 2011).

Adult ADHD is a common problem in the obese population (Gruss, et al., 2012). In the context of bariatric surgery, the potential disruptive effect of ADHD on patients' ability to follow strict dietary regimens may be important. Patients with adult ADHD find following medical recommendations hard, but there has been no longitudinal study of adult ADHD as a risk factor for bariatric surgery patients (Adler & Nierenberg, 2010). Since adherence to behavioral recommendations after bariatric surgery is essential, patients with adult ADHD may be at increased risk of inferior weight-loss outcomes (Elkins, et al., 2005). In addition, symptoms of adult ADHD may also exacerbate the risk of harmful alcohol consumption, a risk that may already be elevated after bariatric surgery (King, et al., 2012; Svensson et al., 2013).

There have been signs that gender and age are important moderating variables for weight-loss outcome after bariatric surgery (Contreras, Santander, & Bravo, 2013; Wimmelmann, et al., 2013). Both gender and age are associated with such psychological problems as eating disorders and adult ADHD. How these variables interact and affect weight loss has not been thoroughly investigated. The aim of this study was to prospectively investigate several potential risk factors for inferior weight loss. The overall purpose was to investigate whether symptoms of eating disorder, of anxiety and depression and of adult ADHD can predict weight loss outcome. More specifically, moderation effects of demographical variables on these potential risk variables were investigated.

Methods

Participants and procedure

Potential participants were approached at an obesity clinic at a university hospital. Of 177 patients approached, 131 (74%) chose to participate in the study. Of these, two participants had a Body Mass Index (BMI) that, at >59, was outside the range of typical patients and they were therefore excluded from all analyses. Of the remaining 129 participants, 101 (78%) were women, the mean age was 42.8 ($SD = 10.52$) and the mean BMI was 42.9 ($SD = 3.98$). Data was collected five months before and 12 months after surgery. The study protocol was approved by the regional ethics committee.

Measures

Disordered eating was measured with the General Food Cravings Questionnaire Trait (GFCQT; Nijs, et al., 2007) which has four subscales; Loss of control, Emotional Eating, Positive expectancies and Occupation with food. Symptoms of anxiety and depression were measured with the HADS (Zigmond & Snaith, 1983) and symptoms of adult ADHD was measured with the ASRS (Kessler, et al., 2005). Alcohol risk consumption was measured with the Alcohol Use Disorder Identification Test (AUDIT; Reinert & Allen, 2007; Saunders, Aasland, Babor, & Grant, 1993). The AUDIT has two different cut off scores for women and men that can be used to detect alcohol risk consumption. Finally, weight was measured and Body Mass Index calculated by the medical staff at the clinic. Percent excess BMI loss (%EBMIL) was used as a measure of weight loss after surgery with normal BMI set at 25 kg/m^2.

Statistical analysis

Participants who had a %EBMIL below two standard deviations from the mean were considered to have minimal weight loss and participants in the lowest quartile of %EBMIL to have low weight loss. Square-root transformation was conducted on all outcome variables to make distributions adequate for parametric analyses. Pearson's r was used to analyze correlations between variables. Nominal and ordinal data were analyzed using Chi-2 and Kruskal-Wallis test. Differences among groups of participants were analyzed using One-way Analysis of Variance (ANOVA) and differences between pre- and follow-up measurement were analyzed with repeated measures ANOVA. Welch's t-test was used when comparing groups of different sizes. Multiple regression analysis was used for investigating the predictive value of variables at pre-measurement on BMI loss at post-measurement.

Cohen's *d* was calculated as a measure of effect size by using the pooled standard deviations (*SD*) for the groups and a *p*-value less than .05 was considered statistically significant in all analysis. Bonferroni correction was used when applicable. Missing data on individual items were imputed with Expectation Maximization simulation. SPSS 20.0 (IBM, 2011) was used to conduct all analyses.

Results

The mean reduction in BMI between pre- and post-measurement was 13.36 (*SD* = 3.86), corresponding to a mean %EBMIL of 75.65 (*SD* = 21.73), which was not significantly different (*p* = .071) between men and women. However, %EBMIL was correlated with age for both men (*r* = -.71, *p* < .001) and women (*r* = -.26, *p* = .010), but showed no significant correlation with other demographic variables. Since gender and age were associated with weight loss and psychological variables, each sex was analyzed separately and age was included as a covariate in all further analyses.

Between pre- and post measurement, there were significant reductions on the GFCQT Total score, the HADS Depression, the ASRS Hyperactivity/Impulsivity and the AUDIT. There were no significant changes in the ASRS Attention deficits or the HADS Anxiety subscales. All outcome variables are presented in Table 6.

Gender specific analysis showed a significant reduction on the ASRS Attention subscale for men (*F*(1,27) = 6.08, *p* = .02, *d* = 0.93), but not for women (*F*(1,100) = 0.007, *p* = .94, *d* = 0.01). Conversely, women reported a significant reduction on the AUDIT (*F*(1,100) = 5.68, *p* = .019, *d* = 0.47), while there was no such effect for men (*F*(1,27) = 3.62, *p* = .068, *d* = 0.72), which may be due to low statistical power. After controlling for age, no psychological variable at pre measurement could predict %EBMIL for either gender. At post measurement, there was a significant correlation (*r* = -.52, *p* = .006) between %EBMIL and the GFCQT Loss of control subscale, but only among men.

Three participants (2.3%) were considered to have minimal weight loss at post-measurement. One was 45 years old while two were notably older (60 and 65 years) than most other participants (*m* = 42.5). All three also scored higher on the GFCQT Loss of control at post-measurement than other participants but the small number of participants ruled out significance testing. The 32 participants in the lowest quartile of %EBMIL (i.e., having a %EBMIL < 65.08) whose weight loss was considered low were significantly (*F*(1,127) = 12.32, *p* < .001, *d* = 0.72) older than the other participants. After controlling for age, post-measurement values on the GFCQT Loss of control subscale were significantly higher among men (*F*(1,27) = 7.99, *p* = .002, *d* =

1.19) but not women ($F(1,100) = 1.49$, $p = .23$, $d = 0.31$) in this group than in the other participants.

Table 6. *Means and standard deviations of the studied variables at pre and post measurement and analyzed differences (N=129).*

	Pre M (SD)	Post M (SD)	F^a	p^a	d^a
ASRS					
Attention deficits	9.79 (4.88)	9.64 (6.51)	0.79	.38	0.16
Hyperactivity /Impulsivity	9.72 (4.51)	8.18 (5.92)	14.36	<.001	0.67
GFCQT					
Total score	2.42 (0.81)	1.52 (0.52)	169.34	<.001	2.31
Preoccupation with food	2.13 (0.87)	1.63 (0.65)	35.1	<.001	1.05
Loss of control over eating	2.77 (1.06)	1.38 (0.49)	254.26	<.001	2.83
Positive expectancies	2.53 (0.98)	1.68 (0.75)	109.86	<.001	1.86
Emotional cravings	2.27 (1.03)	1.39 (0.72)	93.80	<.001	1.72
AUDIT	3.31 (2.89)	2.42 (2.94)	9.17	.003	0.54
HADS					
Anxiety	4.46 (3.19)	3.95 (3.78)	3.80	.053	0.35
Depression	4.17 (3.09)	2.74 (3.30)	24.11	<.001	0.87

Note. ASRS= Adult ADHD Self-Rating Scale, GFCQT=General Food Cravings Questionnaire Trait, AUDIT=Alcohol Use Disorders Identification Test, HADS= Hospital Anxiety and Depression Scale.
[a] Analysis conducted on square-root transformed data.

The 10 participants screening positive for adult ADHD at post-measurement had significantly higher scores for Attention deficits and Hyperactivity/Impulsivity on the ASRS than other participants at both pre- and post-measurement. This did not apply to participants screening positive at pre-measurement only, indicating that pre-measurement symptoms of ADHD may be closely associated with anxiety or other psychological problems rather than with actual ADHD. Post-measurement symptoms of adult ADHD were associated not with weight loss but with disordered eating and

alcohol consumption ($r = .25 - .33$, $p < .01$), even after controlling for age. The number of participants who scored above the cutoff score of alcohol risk consumption on the AUDIT fell from 20 at pre-measurement to seven at post-measurement. However, at post-measurement three participants scored above the cutoff for alcohol disturbance compared with none at pre-measurement.
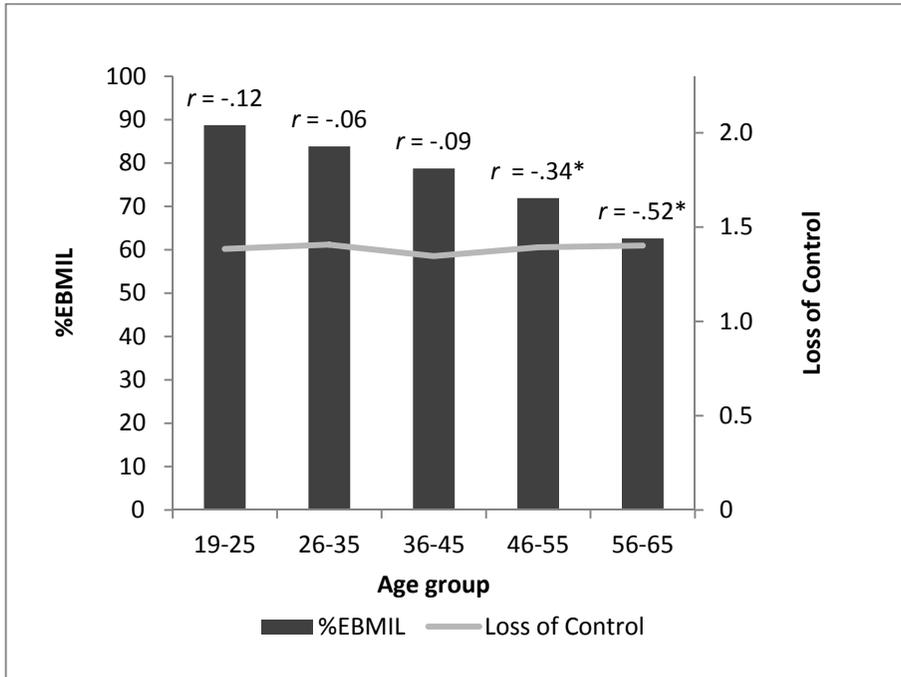

Figure 3. Percent excess BMI-loss, loss of control over eating and correlations between the two variables for the different age groups.
Note. * = $p < .05$, %EBMIL=Percent Excess BMI Loss, LOC= Loss of control over eating.

To analyze the moderating effect of age, participants were grouped into five different age groups and compared. Participants ($n = 17$) in the highest age group (55-65 years) had significantly lower ($F(1,124) = 12.86$, $p < .001$, $d = 0.94$) %EBMIL than other participants. Age also moderated the correlation between GFCQT Loss of control over eating subscale and %EBMIL so that this correlation was significant only in the two highest age groups ($p = .018$ and $p = .042$ respectively), see Figure 3.

Discussion

Most psychological variables investigated in this study improved after bariatric surgery and none of them could predict %EBMIL at post-measurement when controlling for age. At post-measurement, Loss of control over eating was correlated with %EBMIL among men and among the oldest women. Both age and gender thus moderated this association. While the reasons for these moderation effects are unknown, they may partly explain some of the inconsistent results found in previous studies. The results from this study show that disordered eating does not affect weight loss among young women which is surprising. The participants with low or minimal weight loss did report higher levels of disordered eating at post-measurement, indicating that this is still an important variable for many patients, at least for less than optimal weight loss, if not for weight-loss failure.

This is the first study to report surgery weight loss for patients with elevated symptoms of adult ADHD, but the results show that this group may not be at risk for inferior weight loss in the short term. However, post-measurement ADHD symptoms, which were deemed more reliable than those at pre-measurement, were correlated with alcohol consumption. Symptoms of adult ADHD may thus be a risk factor for alcohol disturbance after surgery and are thus important to assess in that respect rather than for inferior weight loss. Although alcohol consumption decreased overall after surgery, it appeared to increase in a subgroup of participants. One of the reasons why significant psychological risk factors were not found in this study may be the short follow-up time used. Twelve months may be too short a time after surgery to identify risk factors that predict weight loss. However, the participants with minimal weight-loss in this study had passed their weight loss nadir prior to the 12-month follow-up, showing that signs of weight-loss failure can appear early after surgery.

In conclusion, this study confirms the difficulty of finding consistent risk factors for inferior weight loss after surgery. However, the results shows that age and gender may have important moderating effects on risk factors and predict weight loss better the than the psychological variables measured. At post-measurement, symptoms of disordered eating were associated with weight loss for a subgroup of patients, further strengthening the argument that postsurgical assessment and support may be more important than presurgical assessment. While symptoms of adult ADHD were not associated with weight loss after surgery, they were associated with alcohol consumption and ADHD may, for this reason, be an important risk factor in this population. Future studies need to explore other risk factors for inferior weight loss as well as alcohol disturbance after surgery, and to use larger samples and longer follow-up times.

General discussion

Main results

The extent to which people can freely control their own behavior is a classic topic of discussion in behavioral and philosophical disciplines. In psychology, it is well established that our behavior is governed by many factors, both exogenous and endogenous, and both conscious and unconscious. In clinical psychology models, disorders are often maintained by nonfunctional behaviors: those that people know are problematic but find difficult to change. From studies of learning theory we know that behaviors are triggered by stimuli and maintained mainly by reinforcement contingencies, sometimes beyond conscious control. This is the overall reason why many people have problems in controlling their behavior, especially when it comes to health behaviors such as smoking or following prescriptions. To get better at helping patients to follow behavioral health recommendations we need an improved understanding of how such behaviors are governed and influenced by psychological variables.

Bariatric surgery is an effective treatment for obesity for most patients and vastly superior to behavioral or medical treatments. Nonetheless, a proportion of patients do not lose weight after surgery or start to regain weight soon after the operation. This weight regain has been associated with disordered eating but also with low adherence to behavioral prescriptions. Though different in many ways, both disordered eating and nonadherence to prescriptions may be viewed as difficulties in controlling behavior. Low adherence to prescriptions is common in traditional behavioral treatments for obesity and is one of the reasons behind the poor results of such treatments (Acharya et al., 2009; Wadden et al., 2013). Reasons for the patients' low adherence have been investigated and several factors, including low self-esteem, low social support and negative affect, seem to influence compliance (Moroshko, et al., 2011). The role of impaired executive function is still elusive but, in practice, many clinics are reluctant to operate on patients with memory deficits or with a diagnosis of adult ADHD. The prevalence of adult ADHD in this patient group and whether it is an important risk factor for low weight loss had not previously been investigated.

In Study I we found that symptoms of adult ADHD were common among bariatric surgery patients and associated with other problems, such as anxiety and disordered eating. Symptoms of ADHD have been shown to impede

behavioral weight-loss treatment and may be a risk factor after surgery as well (Altfas, 2002). Psychological problems such as anxiety and depression may to some extent be manifested as symptoms of ADHD or as concentration difficulties, so caution should be exercised in interpreting the results. At the same time, other studies have reported a prevalence of adult ADHD similar to that found in Study I. This indicates that levels of ADHD symptoms found in Study I were not simply a manifestation of other psychological problems or disorders. Previous studies have also shown that eating disorders are common among people with ADHD and the results from Study I are in line with these findings. Impulsivity moderates the association between stimuli and behavior and highly impulsive people may be more inclined to act in response to cues rather than according to long term goals. Consequently, since adult ADHD can disrupt people's daily function and ability to follow both behavioral and medical prescriptions, it may potentially hamper the ability to follow post bariatric recommendations.

Other researchers have noted that bariatric patients may underreport some problems that, in their view, might hinder or delay the surgery (Mitchell et al., 2012). This seems to be especially relevant for alcohol- and drug use, where the reported levels are markedly lower among patients seeking bariatric surgery than in the general obese population (Heinberg, et al., 2012). At the same time, several studies have reported an increased prevalence of alcohol abuse after surgery. Whether this is mainly due to underreporting alcohol use prior to surgery is unclear. If there is such an effect, the same may be true of disordered eating, depression and symptoms of adult ADHD. In Study I, data collection was designed in such a way as to minimize underreporting. When comparing the results from different studies, it is important to consider how data were collected and the incentives for patients to report problems. The exact timing of data collection during presurgical preparation may also be crucial since some patients with severe problems may be referred to other care early in the process. Prior to Study I, there was no published study that assessed symptoms of adult ADHD in the bariatric surgery population and its association with disordered eating. After Study I it was found that symptoms of ADHD may be an important factor in this population and it was decided to continue with an investigation of ADHD symptoms as well as related risk behaviors in a prospective study.

Besides low adherence to recommendations, binge eating after bariatric surgery has been shown to predict lower weight loss than expected. Most patients who binge-eat prior to surgery seem to improve after surgery, at least in the short term. But for a subgroup of patients, disordered eating continues to be a problem. It is possible that the improvement seen after surgery is analogue to the treatment effect observed after regular eating patterns are introduced in CBT for BED. As mentioned above, CBT treatment for BED starts with establishing regular eating patterns that are later developed into flexible dietary rules. It has been shown that many patients' binge eating is

reduced when they start to eat more regularly. After surgery, patients must eat at regular intervals and this may have a somewhat similar effect on binge eating as seen in CBT. But similar to CBT, some patients' binge-eating does not improve after surgery. There may be a subgroup of patients with a somewhat different disorder profile for example characterized by low self-esteem or negative affect (Stice et al., 2001).

Negative affect has long been associated with binge eating in patients with eating disorders although the exact mechanisms have been elusive. In terms of learning theory, it seems conceivable that negative affect may trigger binge eating which would then be negatively reinforced by the reduced distress. However, it has been difficult to show these mechanisms in action since people with BED typically report negative affect both before and after binge eating. Further, treatments such as IPT and DBT that specifically target negative affect and the strategies used to regulate affect have not succeeded in showing that these mechanisms explain the positive treatment effects.

Another way to conceptualize the treatments for BED is that they make patients engage in behaviors they find meaningful and that these are positively reinforced. Example may be seeing friends, engaging in hobbies or being outdoors. Such behaviors typically involve intrinsic positive reinforcement and are furthermore incompatible with binge eating. We thus hypothesized that another treatment, BA, which is characterized by helping patients find sources of positive reinforcement, might be effective in replacing binge eating behaviors. The results from Study II however, showed that while BA was effective in increasing the levels of positive engagement and elevating mood in obese patients with BED it was not effective in reducing binge eating. Interestingly, this further implies that negative affect and binge eating are not as closely associated as sometimes suggested. At least, the results indicate that a general improvement in mood does not substantially affect binge eating.

One weakness in the design of Study II was that patients both with and without negative mood were included, BA might possibly have had a better effect on patients with BED and negative mood. On the other hand, the hypothesis behind the chosen design was that the positive behavioral activation would replace binge eating directly rather than indirectly through elevated mood. A further limitation in the design was the absence of an active control group. For example, a control condition with a treatment that focused on improving flexible restraint over eating would have provided valuable additional data on the mechanisms at work in the treatment of BED. In conclusion, BA does not seem to be a feasible treatment option for patients with BED, at least in the short group format used in Study II.

After the data in Study I had been analyzed, Study III was designed to prospectively measure both symptoms of adult ADHD and alcohol consumption, in addition to the typical risk factors among bariatric patients. The re-

sults showed that for many patients, psychological variables measured prior to surgery had improved 12 months after surgery. None of the variables measured prior to surgery predicted weight loss after surgery but there was an association, moderated by sex and age, between post surgical disordered eating and weight loss. Symptoms of adult ADHD and alcohol consumption were not associated with weight loss but while the overall consumption of alcohol decreased, a small group of patients may have developed alcohol risk consumption. As mentioned above, this finding may be an effect of patients withholding information prior to surgery. There are however, strong indications that the risk of alcohol misuse increases after surgery with Gastric bypass procedures (Svensson, et al., 2013). It has previously been difficult to identify patients who may be at risk of alcohol risk consumption after surgery but in Study III we found that symptoms of adult ADHD were associated with alcohol consumption. Symptoms of ADHD may thus be a risk factor of importance for alcohol consumption and other risk behaviors rather than for inferior weight loss.

While eating behavior is regulated by a complex pattern of present contingencies and past experiences, mood, and especially negative affect, is a very strong determinant of behavior. However, impulsivity may moderate this association and may be important to investigate in the context of bariatric surgery.

Limitations

An important limitation in all three studies was the reliance on self-report questionnaires. While self-report questionnaires can be useful, for example for collecting sensitive data, the results regarding diagnoses or specific behaviors must be evaluated critically. For example, the ASRS should not be used to measure the prevalence of ADHD in a population but only to assess ADHD symptoms and to screen for possible cases. It is also difficult to interpret some results from self-report data since many symptoms of different disorders overlap. A person with an anxiety disorder may report restlessness and concentration difficulties that may mistakenly be interpreted as symptoms of adult ADHD. Structured clinical interviews might have been a valuable addition to the data collected by questionnaire but were not feasible owing to the limited resources at our disposal. In study III a self-report measure of compliance to recommendations after surgery would have been very valuable but no such instrument is currently available. Collection of additional data on executive function through computerized cognitive tests was assessed in a pilot study but unfortunately deemed unfeasible.

A further limitation that became evident primarily in Study II was that the clinical setting where the studies were conducted imposed restrictions on study design. These restrictions included prohibition of audio recording of

treatments and the obligation to provide adequate care to participants in the control condition. It was thus not possible to assess treatment fidelity properly or to use a pure control group. While measures were taken to ameliorate these limitations, such as highly structured therapist instructions, the internal validity of the study was affected negatively. The clinical context of low experimental control may not be the optimal choice for this kind of study. On the other hand, external validity benefited from conducting the studies and recruiting participants in a clinical setting.

Future studies

While the studies presented here provide some additional data, the associations between impulsivity, negative affect and binge eating are still elusive. The psychological variables studied, such as anxiety and symptoms of ADHD, are typically closely correlated and difficult to separate. Based on the results from Study I and Study III, disordered eating seems to be associated with symptoms of adult ADHD but it is unclear whether this association can be better explained by anxiety or other similar variables. Future studies need to clarify these associations and to distinguish between symptoms of ADHD and other psychological problems such as anxiety and depression in more detail. If it can be confirmed that symptoms of adult ADHD are independently associated with disordered eating, it would be very valuable to investigate whether treating ADHD may affect binge eating. It would also further substantiate the existence of subgroups of patients with BED with various psychological profiles that may not benefit from CBT for BED.

A better understanding of subgroups among people with BED may help to explain the mixed results from research on risk factors prior to bariatric surgery. Many patients with disordered eating report improved eating after surgery, at least in the short run. It seems that for a certain group of patients the symptoms improve, while for others they persist. Identifying such groups may improve routines for presurgical assessment and long-term follow-up and care. It may also lead to better understanding how bariatric surgery affects the mechanisms regulating eating and weight loss in these patients.

The effective components of psychotherapy for BED are still incompletely understood. Inflexible restraint over eating is still the only major factor identified that is important to target in treatment. Future studies may focus on improving treatments that emphasize flexible eating or combine such treatments with new components targeting negative mood or impulsivity.

Summary of findings

The three empirical studies of this thesis sought to clarify the prevalence and associations between negative mood, impulsivity and binge eating in people with obesity wishing to undergo bariatric surgery. In these people, prior to surgery, the prevalence of adult ADHD may be higher than in the normal population. Symptoms of impulsivity and hyperactivity were elevated and associated with levels of disordered eating as well as anxiety and depression. After surgery, impulsivity was associated with risk behaviors such as alcohol consumption but not with disordered eating and did not predict short-term weight loss after surgery. Symptoms of adult ADHD may thus be a risk factor for unhealthy behaviors after surgery rather than for low weight loss. Most patients' disordered eating improved after surgery but a small group reported sustained symptoms. Consistently with previous studies, disordered eating was found to be associated with weight loss only if present after surgery and only among some patient groups.

Increased levels of positively reinforced activities improved mood in patients with obesity and BED but did not ameliorate their binge eating. This suggests that binge eating is strongly influenced by such factors as dietary restraint and overvaluation of weight and shape rather than activity levels or mood.

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