Binge or control? : assessment of the validity, treatment and underlying mechanisms of Binge Eating Disorder

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Chapter 1

Introduction

Introduction

Binge eating disorder (BED) is one of the eating disorders besides anorexia nervosa (AN) and bulimia nervosa (BN). BED is part of the "eating disorders not otherwise specified" (EDNOS) category, which is reserved for patients who have significant eating disorder psychopathology but who do not meet full criteria of AN or BN. Binge eating disorder (BED) is characterized by recurrent episodes of binge eating (American Psychiatric Association, 1994). An eating episode is considered as a binge-eating episode when an amount of food is eaten that is definitely larger than what most people would eat during a similar period of time and under similar circumstances. Eating has to occur within a discrete period of time (two hours) and there has to be a sense of lack of control over eating during that period. Most people who engage in binge eating eat alone because of being embarrassed by how much one is eating. Most people describe eating much faster than usual during binge eating episodes and do not enjoy eating. Usually large amounts of food are eaten when not feeling physically hungry. Afterwards they feel disgusted with themselves, depressed, or very guilty. There must be a marked distress regarding the behaviour. Overweight and obesity are common comorbidities (Spitzer et al., 1991).

In 1991 Spitzer and others suggested that BED should be included in the DSM-IV. Their rationale for this proposal was that many individuals with marked distress about binge eating could not be diagnosed with bulimia nervosa (BN). People with the BED-syndrome have episodes of binge eating as do patients with bulimia nervosa but unlike the latter they do not engage in compensatory behaviours such as self-induced vomiting, the misuse of laxatives, diuretics or diet pills, fasting and excessive exercise. They indicated that such patients are common among the obese in weight control programs.

Binge eating disorder (BED) was introduced in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association (DSM-IV)(1994) as a new diagnostic category requiring further study and as an example of Eating Disorder Not Otherwise Specified (ED-NOS). The criteria are described in an appendix, indicating that BED requires further research before it can be incorporated as a fully accepted category in the DSM. There was some reluctance to admit BED as a distinct eating disorder in a next version of the DSM.

Some studies suggested that BED might be a transient and unstable condition in contrast with Anorexia Nervosa en Bulimia Nervosa, which are frequently chronic (Cachelin et al., 1999; Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Pope et al., 2006). Is BED distinguishable enough from BN and obesity? The addition of new and unproven diagnoses carries the risk of trivializing the construct of mental disorders and/or its misuse. This is relevant for BED since we would not wish normal gluttony to be classified as a psychiatric disorder (Fairburn, Welch, & Hay, 1993b).

Table 1. Research criteria for binge eating disorder (American Psychiatric Association, 1994)

A. Recurrent episodes of binge eating. An episode of binge eating is characterised 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 in a similar period of time under similar circumstances
2. A sense of lack of control over eating during the episodes (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 usual
2. Eating until feeling uncomfortably full
3. Eating large amounts of food when not feeling physically hungry
4. Eating alone because of being embarrassed by how much one is eating
5. Feeling disgusted with oneself, depressed, or very guilty after overeating
C. Marked distress regarding binge eating is present
D. Binge eating occurs, on average, at least 2 days a week for 6 months
E. The binge eating is not associated with regular use of inappropriate compensatory behaviours (e.g. purging, fasting, excessive exercise) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa.

In the two decades following the proposal of Spitzer et al. in 1991, researchers investigated many aspects of BED. Since the early nineties the number of studies in patients with BED increased enormously. In 1991 only 1 study was mentioned in Pubmed with BED in the title whereas in 2007 44 were mentioned. An up-to-date review of this expanding research field was needed. What are the results of a decade of research in the field of BED? Is BED distinguishable with respect to eating disorder symptoms, cognitions and maladaptive schemas from the other eating disorders? Several studies have demonstrated that patients with an eating disorder also have dysfunctional cognitions or maladaptive core beliefs that are not directly related to food, weight or shape (Cooper, 1997; Leung, Waller, & Thomas, 1999; Waller, Ohanian, Meyer, & Osman, 2000). Young (Young, 1999) states that maladaptive core beliefs represent the deepest level of cognition. These core beliefs reflect a person's unconditional negative beliefs and feelings in relation to the environment. What are the differences and similarities of these core beliefs between patients with BED and patients with AN, BN and healthy controls? What do we know about the etiology of binge eating in patients with BED? How widespread is this eating disorder? What kind of treatment is suitable for patients with BED? The main question is in what aspects patients with BED differ from patients with bulimia nervosa or anorexia nervosa and from healthy controls. Does it represent a distinct eating disorder?

Treatment approaches to BED

Although BED is not a formal diagnosis within the DSM-IV, in day-to-day clinical practice binge eating disorder is a generally accepted category. Nowadays practically every eating disorders centre or clinic has developed a distinct treatment program for BED. Various treatment studies have been preformed in patients with BED ranging from psychological (Grilo & Masheb, 2005; Carter & Fairburn, 1998; Eldredge et al., 1997; Agras et al., 1995; Nauta, Hospers, & Jansen, 2001; Agras, Telch, Arnow, Eldredge, & Marnell, 1997a; Nauta, Hospers, Kok, & Jansen, 2001; Wilfley et al., 2002a; Telch, Agras, & Linehan, 2002; Peterson et al., 1998; Peterson et al., 2001; Gorin, le Grange, & Stone, 2003), pharmacological (Hudson et al., 1998; McElroy et al., 2000; McElroy et al., 2003; Arnold et al., 2002; McElroy et al., 2004; Appolinario et al., 2003; Pearlstein et al., 2003), dietary (Raymond, Zwaan, Mithcell, Ackard, & Thuras, 2002a; Porzelius, Houston, Smith, Arfken, & Fisher, 1995a; Goodrick, Poston, Kimball, Reeves, & Foreyt, 1998a), surgical treatments (Busetto et al., 2005; Silva et al., 2007) to combinations from the previous (Grilo, Masheb, & Salant, 2005; Agras et al., 1994a; Grilo et al., 2005; Grilo, Masheb, & Wilson, 2005; Laederach-Hofmann et al., 1999; Munsch et al., 2007). Abstinence of binge eating is usually the primary goal of treatment. Secondary goals might be weight loss, reduction of eating disorder symptomatology, depressive and anxiety symptoms or improving quality of life or self-esteem.

Cognitive Behavioural Therapy (CBT)

Cognitive behavioural therapy is one of the treatments for BED. Cognitive therapy and behavioural therapy emerged independently. Nowadays cognitive therapy, behavioural therapy and cognitive behavioural therapy are used interchangeably. Behaviour therapy emerged partly from the behaviourist traditions, which state that psychological matters can be studied scientifically by observing overt behaviour without discussing internal mental states. Cognitive therapy is aimed at changing dysfunctional cognitions. Beck and Ellis both developed their own cognitive therapy independently of each other with basically the same underlying theory (Beck, 1976; Ellis, 2008). Its own specific schemas and automatic cognitions characterize each psychopathological disorder. An effective correction of dysfunctional cognitions and schemas is presumed to resolve the emotional disorder.

CBT is a semi-structured and problem-oriented therapy. It is concerned mainly with the patients' present and future rather than with their past (Fairburn, 1993). CBT is a psychotherapy based on modifying cognitions, assumptions, beliefs and behaviours, with the aim of influencing disturbed emotions and behaviours. The particular therapeutic techniques vary according to the particular kind of client or issue, but commonly include keeping a diary of significant events and associated feelings, thoughts and behaviours; questioning and testing cognitions, assumptions, evaluations and beliefs that might be unhelpful and unrealistic; gradually facing activities which may have been avoided; and trying out new ways of behaving and reacting.

The focus in CBT is on the factors and processes that maintain the eating problem rather than on those that operated earlier in its evolution. Fairburn (Fairburn, 1981) was the first to describe CBT as a useful therapy for bulimia nervosa. It was the first promising treatment for bulimia nervosa. The theoretical model underlying this cognitive-behavioural approach for binge eating is that the cultural expectations regarding body shape shifted in the 1960s, 1970s and 1980s toward thinness. Dieting became the way to achieve thinness (Agras, 1993). In some women dieting became very rigid accompanied by excessive caloric deprivation. This led to cycles of dieting followed by episodes of binge eating when food rules were broken. The criteria for BED are derived from the criteria for bulimia nervosa (BN). The characteristics of BED are partly similar to those of bulimia nervosa. The central criterion for BED is the occurrence of episodes of binge eating which is also an essential criterion for bulimia nervosa. Therefore, it is not surprising that the first generation of BED treatment research focused on examining the efficacy of those treatments that had been shown to be effective for BN: cognitive behavioural therapy (CBT), interpersonal psychotherapy (IPT) and antidepressant medication. CBT is the most widely investigated treatment for both BN and BED and has emerged as the treatment of choice for both disorders; it has become the golden standard to which other treatments are compared.

Three stages in the treatment can be distinguished (Fairburn, 1993). The first is to identify the rationale underlying the cognitive-behavioural treatment approach, and the second is to replace binge eating with a stable pattern of regular eating. In stage two there is continuing emphasis on regular eating and the use of alternative behaviour, but in addition the focus broadens to address all forms of dieting, concerns about shape and weight, and more general cognitive distortions. The aim of the third and final stage is to ensure that progress is maintained in the future.

Worldwide only a dozen randomized controlled trials were conducted in patients with BED. In the Netherlands no randomized controlled treatment trials are preformed in patients with Binge Eating Disorder. Also, little is known about possible predictors and mediators for change. Only two studies have investigated possible predictors for success (Wilfley et al., 2000a) and relapse (Safer, Lively, Telch, & Agras, 2002). No studies were done to investigate possible mediators for treatment success in patients with BED. Thus far, only one study in patients with bulimia nervosa investigated mediators of treatment success (Wilson, Walsh, Kraemer, Fairburn, & Agras, 2002). Consequently, the following questions remain unanswered. Is Cognitive Behavioural Therapy also effective in a group of Dutch patients with BED? What are the mechanisms of change? Are there predictors for treatment success? In other words, is it possible to predict in advance who will be successful in abstaining binge eating?

Theoretical models of Binge eating: Restraint versus emotion regulation

Restraint models

In the BN literature it has been suggested that binge eating develops in the context of dieting or restraint eating. According to this restraint theory (Polivy & Herman, 1985) dieting increases the likelihood of subsequent binging. Dieting seems to precede binging more often than the other way around. Polivy and Herman (1985) found that in clinically normal dieters almost any inhibitor (high-calorie preloads, alcohol, emotions like anxiety or depression) might disrupt the dieter's characteristic restraint and release suppressed eating which is not observed in non-dieters. They argue that the role of cognitions and situational pressures is crucial in the instigation of binge eating. As long as dieters think they are in control they will not overeat, but when they believe that their diets have been violated or that they are no longer capable of controlling their intake, they overeat or even binge.

In bulimia nervosa restrictive dieting is almost viewed as a 'precondition' for the development of binge eating, is central to most etiological and risk models and plays an important role in treatment approaches (Agras, 1993). However, the association between dieting/restraint and binge eating does not apply to a substantial number of individuals with BED. For example, measures of restraint of eating (Three-Factor Eating Questionnaire (TFEQ) (Stunkard & Messick, 1985) and Eating Disorder Examination (EDE) (Fairburn & Cooper, 1993a)) are usually significantly lower in obese BED patients compared to patients with BN and have consistently shown either no correlation or even a negative correlation with binge eating among obese patients (see for an overview de Zwaan, 2005b). Also, Ardovini et al. (1999) found a close relationship between restriction, disinhibition and BN that was not present in BED, where high disinhibition levels were associated with low caloric restriction levels. Further, in BED, in contrast with BN, there is evidence that the onset of binge eating precedes the onset of dieting in about 35% to 65% of the cases (Abbott et al., 1998; Grilo & Masheb, 2000; Mussell et al., 1995; Spurrell, Wilfley, Tanofsky, & Brownell, 1997; Marcus, Moulton, & Greeno, 1995).

Treatment studies comparing patients with and without BED in weight reduction programs do not find a significant differential outcome in weight loss. Patients with BED did not lose less weight than patients without BED. Moderate caloric restriction does not seem to exacerbate binge eating in obese BED patients (Agras et al., 1994a; Yanovski et al., 2000; Goodrick et al., 1998a; Porzelius et al., 1995a; Raymond et al., 2002a; de Zwaan, 2005b). Also, numerous studies have reported reductions in symptoms of depression and anxiety or, at minimum, no worsening in affect in obese patients treated by behaviour modification combined with moderate or severe caloric restriction or use of weight loss medications (Yanovski et al., 2000). For example, Goodrick et al. (1998a) investigated the effectiveness of a nondieting (NDT) and dieting treatment (DT) for obese patients with BED. Both treatments were weekly during 6 months and biweekly during an additional 12 months. They found a sustained reduction in binge eating in both groups (measured by the BES). Both treatments

were similarly ineffective in weight loss. Also Raymond et al. (2002a) found that a Very Low Calorie Diet (VLCD; 800 kcal per day) did not worsen BED symptoms. It may be that the encouragement of regular meals and snacks, stimulus control etc. are per se effective in reducing binge eating in BED patients. Chaotic eating patterns may play a larger role in triggering binge eating than the intention to restrict calories and actual efforts to restrain food intake (Nauta et al., 2001).

Butryn and Wadden (2005) reviewed the relationship between weight loss interventions and the risk for eating disorders in children and adolescents. They concluded that professionally administrated weight loss interventions pose minimal risks of precipitating eating disorders. Also in adults, studies consistently found that prescribed modest caloric restriction or VLCD did not precipitate binge eating (Butryn et al., 2005; Yanovski et al., 2000). Wadden et al. (Wadden et al., 2004) randomly assigned 123 overweight women (without a history of binge eating, major depression and other psychiatric conditions that significantly affected daily functioning) during 40 weeks to 1) a balanced-deficit diet (1200-1500 kcal), 2) meal replacement plan (1000 kcal) or 3) non-dieting approach. They found no evidence that a diet was associated with binge eating or other disordered eating. Moreover, neither of these diets was associated with increases in hunger, dietary disinhibition or symptoms of depression. However, Telch and Agras (1993) who examined the effects of a Very Low Calorie Diet (VLCD) on binge eating, did find that about 62% of the obese women identified as non-bingers at baseline, reported binge eating episodes at the termination of the behaviour therapy weight loss program. These findings must be interpreted cautiously, because all measurements of binge eating were based on patients' subjective assessments except baseline and final assessments. Between baseline assessment and beginning of the VLCD 30% already reported having binge eating episodes (Yanovski et al., 2000). In conclusion, caloric restriction does not seem to have the same effect on binge eating in overweight patients with BED as it is assumed in normal-weight patients with BN.

Emotion regulation models

Another model, which tries to explain the etiology of binge eating, is the affect regulation model. Affect regulation models posit that emotional disturbance and coping deficits increase the likelihood of binge eating (Grilo & Shiffman, 1994). Also Waters et al. (2001) found a clear link between negative emotional states and bulimic behaviour (in patients with BN), omitting the restrictive stage. Food cravings that led to a binge were associated with lower levels of mood, lower energy, higher levels of tension and lower levels of hunger than cravings that did not lead to a binge. Greeno et al. (2000) investigated binge antecedents in women with BED and obese women without BED and concluded that locally poor mood precedes binge episodes in women with BED. Although mood is worse overall for women with BED, mood is especially poor before binge episodes. Women with BED also experienced

less eating control and more food cravings although not more hunger than women without BED.

Stice (1994; 2001b) introduced the dual-pathway model. This model posits that problems with either (or both) dietary restraint or affective regulation may trigger binge eating in patients with bulimia nervosa. Stice et al. (2001b) also found evidence for a dietary subtype and a dietary-depressive subtype in patients with BED. The dietary-depressive subtype is a more severe variant of BED marked by increased psychopathology (greater eating, weight and shape concerns, more objective binge episodes), more impaired social functioning and a poorer treatment outcome (after dialectical behavioural therapy) compared to the dietary subtype. Grilo et al. (2001) replicated this study and found similar results. The one discrepancy was that they failed to find a statistically significant difference in the frequency of objective binge eating episodes between the dietary BED-subtype and a dietary-depressive BED-subtype.

Stice et al. (2000) found in a community sample of adolescents that dieting and negative affect were positively related to binge eating and that negative affect potentates the relation between dieting and binge eating (cross sectional data). Negative affect seems to distract people from their dietary vigilance. Also, chronic negative affect that occurs naturally may be different from acute negative affect inductions (as in an experiment).

In sum, several cross-sectional and therapy outcome studies suggest that depressive symptoms (trait), acute negative mood (state), and binge eating behaviour are related. However, little is known about the causality of mood and binge eating. What is the causal relationship between negative affect and binge eating in patients with BED? Only a few experimental studies have attempted to unravel the causal relationship between depression and binge eating in patients with BED. In the subsequent paragraph an overview is given of the relevant studies in patients with BED.

Experimental studies on binge eating disorder (BED)

Only a few experimental studies have been done in patients with BED. These studies focused on the effect of changes in mood, deprivation of food or a combination, on eating behaviour. All studies tried to find an answer to the question what are causal mechanism for binge eating in patients with BED.

Telch and Agras (1996b) addressed the question whether negative mood states influence the eating behaviour of individuals diagnosed with BED. They examined the effects of an induced neutral or negative mood on the caloric intake of participants with BED compared to weight-matched participants without BED. Their hypothesis was that participants receiving a negative mood induction would consume more calories than participants receiving a neutral mood induction, and that BED participants in the negative mood induction would evidence the greatest caloric intake. However, no statistically significant effects were found. Another finding of the study was that negative mood influenced the perception of BED

participants and therefore the labeling of the eating episode as a binge rather than the amount of food eaten.

Telch and Agras (1996a) tested in an experiment the hypothesis that caloric deprivation leads to disinhibited overeating in eating-disordered participants. They examined the effects of a 1-hour and 6-hour food deprivation manipulation on the caloric consumption of BED, BN and overweight non-eating disordered participants. Within each diagnostic group participants were randomly allocated to a 1-hour or 6-hour food deprivation. They hypothesized that 6-hour deprived eating disordered participants would respond to the restriction by binge eating and the eating disordered group who was not deprived, would not. However this was not the case, no statistically significant differences were found. Caloric deprivation did not lead to disinhibited eating.

In another experiment Agras and Telch (1998) extended on their previous discussed research regarding the influences of caloric deprivation and negative mood on caloric consumption, loss of control and binge eating in women with BED. They extended the deprivation period in one group to a minimum of 14 hours and in the other group to 2 hours. Negative mood led to more self-defined binges than neutral mood, whereas self-defined binge eating occurred equally frequently for the two levels of caloric deprivation. When binges were objectively defined, both caloric deprivation and negative mood led to binge eating. Caloric deprivation, but not negative mood, was associated with significantly increased caloric intake. Negative mood, and not deprivation, significantly increased loss of control over eating, which perhaps explains the mechanism underlying the triggering of binge eating by a negative mood. Baseline depression scores (BDI) were equal for those who binged and those who did not, suggesting that it is acute negative affect rather than a stable negative mood that leads to binge eating. A further interesting finding was the alleviation of anxiety over the course of the buffet, whereas depression declined but was still significantly higher than the level reported in the neutral mood condition post-buffet.

Chua et al. (2004) tested in their experimental study two hypotheses: 1) that induction of negative mood would increase food intake in obese binge eaters and 2) that food intake would be greatest in those who were highly restrained after induced negative mood. Forty patients with binge eating disorder (DSM-IV) were categorized (after calculating the median of the restraint scale of the Dutch Eating Behaviour Questionnaire (DEBQ)) to a high restrained and a low restrained group. Participants were randomly assigned to either a negative or a neutral film condition. After watching a negative or a neutral film fragment, they were asked to rate chocolate on taste. Participants in the negative condition consumed significantly more chocolate than participants in the neutral condition. However dietary restraint had no significant impact on food intake according to the authors. However, the film by restraint interaction was marginally significant ($p = 0.06$) which might give some indications of the influence of restraint on binge eating. High-restrained participants in the negative mood condition did eat significantly more than the high-restraint participants in the neutral mood

condition. Overall, food intake in the low-restrained groups did not differ significantly from the intakes of the high-restrained groups.

Munsch et al. (2008) investigated whether negative mood and unbalanced nutrition style synergistically trigger binge eating in overweight and obese patients with binge eating disorder. Sixty-nine patients with BED were randomly assigned to four groups: negative mood induction and balanced nutrition, negative mood induction and unbalanced nutrition (carbohydrate-low/fat-rich), neutral mood induction and balanced nutrition, neutral mood induction and unbalanced nutrition. After following their nutrition plan during three days, they were first subjected to a mood induction (neutral or negative) followed by a taste task in which their food intake was measured. Negative mood and nutrition style were not associated with a high food intake. No differences were found between the 4 groups. Negative mood increased in the negative mood induction conditions and decreased during the taste task, whereas this was not the case in the neutral mood induction conditions. The authors assumed that binge eating often occurs in negative mood because eating decreases negative mood rather than negative mood being a trigger for binge eating.

Thus to date, findings on the causal relationship between depressive symptoms, acute negative mood, and excessive eating are inconclusive. Also it is not clear what the effect of dieting and total food intake during the day is on binge eating. There are many indications that more severe binge eating is related to higher levels of depression (Telch & Agras, 1994a; Antony, Johnson, Carr-Nangle, & Abel, 1994; Mussell et al., 1996a; Stice et al., 2001b; Grilo et al., 2001; Peterson, Thuras, Crow, Mitchell, & Miller, 2005). Major depressive disorder (MDD) is by far the most common diagnosis (46% to 51% lifetime prevalence) associated with BED (Hudson, Hiripi, Pope, & Kessler, 2007; Wilfley et al., 2000a; Mussell et al., 1996a; Specker, Mitchell, de Zwaan, & Raymond, 1994; Telch & Stice, 1998; Yanovski, Nelson, Dubbert, & Spitzer, 1993). The pervasiveness however of the link between emotional distress and binge eating suggests that the question is not whether but how negative affect produces these effects (Tice, Bratslavsky, & Baumeister, 2001a). Many questions have not yet been addressed. For example, binge eating may be used as a mean to escape from these negative thoughts and worries and may in this way help to alleviate emotional stress (Heatherton & Baumeister, 1991). Studies on mental control in general and emotion suppression in particular have showed frequently and consistently that people fail when they try to suppress an emotion or negative mood (Wegner, White, Schneider, & Carter, 1987; Wegner, Erber, & Zanakos, 1994). When people want to stop a worry, escape bad moods or to stop thinking about food when on a diet, they fail again and again. Is loss of control over eating (binge eating) a result of attempts to regulate negative emotional reactions? On the other hand it might also be suggested that emotional distress shifts priorities to the immediate present (Tice et al., 2001a). When people feel acutely bad, they generally wish to feel better and this wish is often urgent. Certain impulses or self-indulgent behaviours are not always simply a sign of reduced control; rather,

they may be strategic efforts at affect-regulation. Is it possible that specific expectation of mood improvement by the consumption of high calorie food leads to overeating?

Goal and outline of the thesis

This thesis focuses on patients with Binge Eating Disorder. The thesis consists of three parts. In the first part the validity of the diagnosis of BED will be discussed. The results of two literature reviews and an empirical cross-sectional study will be discussed. In the second part results of a randomized controlled trial will be described and in the third and last part the results of two experimental studies will be presented.

The first part of this thesis will be a further examination of the validity of the diagnosis of BED. Is there evidence that BED is an eating disorder that can be clearly distinguished from other eating disorders? In chapter two an overview will be given of the epidemiology, characteristics, etiology, criteria, course and possible treatments for BED and their outcome. An overview will be given of the first decennium of research in the field of BED addressing questions, such as: Is there evidence that BED is a distinct eating disorder category and should it be admitted into the next version of the DSM?; Is there enough distinction between BED and bulimia nervosa?; In what respect are these two eating disorders alike?

The third chapter focuses on the question whether the existing evidence warrants assignment of BED as a distinct eating disorder category, which therefore should be included as such in the next version of the DSM

The fourth chapter addresses the question whether BED can be distinguished from the other eating disorder categories and the healthy controls with respect to maladaptive core beliefs. In a cross-sectional study differences and similarities between patients with Binge Eating Disorder, Bulimia Nervosa, Anorexia Nervosa (restrictive and binge/purge subtype) and healthy controls with respect to maladaptive core beliefs were investigated. Is there a difference in the level of maladaptive core beliefs in patients with BED, other eating disorder and healthy controls? Do specific core beliefs predict the occurrence of eating disorder behaviours like bingeing, vomiting, misuse of laxatives or fasting? Is there an association between core beliefs and BMI?

In the second part the results of a randomized controlled treatment trial in patients with BED are discussed. Only a few randomized controlled treatment trials were performed in patients with BED worldwide. The aims of this randomized controlled trial were to explore the effectiveness of cognitive behavioural therapy (CBT) compared to a waiting list (WL) and to identify possible predictors and mediators of CBT for patients with BED. Of main interest were not only the effects of CBT on reducing the frequency of binge eating but also the influence of treatment on other eating disorder psychopathology, as well as co-morbid psychopathology (such as general psychopathology and depressive symptoms), maladaptive core beliefs (as a measure for personality psychopathology), ineffective coping styles and body

weight loss. Our second aim was to investigate whether changes in coping styles, general eating disorder psychopathology, co-morbid psychopathology and body weight during treatment mediate treatment outcome. Our third aim was to investigate whether co-morbid psychopathology, maladaptive core beliefs, body weight and coping styles at baseline predict treatment outcome and whether these variables predict maintenance of treatment outcome during the 1-year follow-up period. The results will be presented in the fifth chapter.

What is the mechanism underlying binge eating? What is the causal relationship between negative affect and binge eating in patients with BED? In the third part of this thesis the results of two experimental studies will be presented. The aim of the first experimental study was to investigate the causal relation between the regulation of negative emotions, negative mood, and binge eating (chapter 6). Is there a causal relation between the regulation of negative emotions, negative mood, and binge eating? Participants were randomly assigned to either a condition in which they were instructed to suppress their emotional reactions during a sad film fragment or to a condition in which they had to show their natural reactions. Afterwards they were all subjected to a taste task. It was hypothesized that overeating is a consequence of an attempt to regulate negative emotions because the limited store of self-control resources has been depleted by a prior act of self-control (suppression of emotional reactions).

Is it possible that specific expectation of mood improvement by the consumption of high calorie food, leads to overeating? In the second experimental study it was investigated whether there is a causal relationship between expectancies regarding the effect of eating on mood, changes in mood (positive or negative) and actual caloric intake (chapter 7). Is there a causal relationship between the expectation that eating alleviates negative mood, mood changes and caloric intake? If this causal relationship exists than altering the expectations with regard to eating would cause a change in caloric intake.

Chapter 8 contains an overview and a general discussion of the findings. The results of the studies will be discussed and integrated. Clinical implications and future directions will be given.