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

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Binge or control?

Assessment of the validity, treatment and underlying mechanisms of Binge Eating Disorder

Binge or Control?
Assessment of the validity, treatment and underlying mechanisms of Binge Eating Disorder
Alexandra Dingemans
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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.

Chapter 2

Binge Eating Disorder: A review

Alexandra Dingemans
Tijs Bruna
Eric van Furth

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Summary

Binge eating disorder (BED) is a new proposed eating disorder in the DSM-IV. BED is not a formal diagnosis within the DSM-IV, but in day-to-day clinical practice the diagnosis seems to be generally accepted. People with the BED-syndrome have binge eating episodes as do subjects with bulimia nervosa, but unlike the latter they do not engage in compensatory behaviours. Although the diagnosis BED was created with the obese in mind, obesity is not a criterion. This paper gives an overview of its epidemiology, characteristics, aetiology, criteria, course and treatment. BED seems to be highly prevalent among subjects seeking weight loss treatment (1.3-30.1%). Studies with compared BED, BN and obesity indicated that individuals with BED exhibit levels of psychopathology that fall somewhere between the high levels reported by individuals with BN and the low levels reported by obese individuals. Characteristics of BED seemed to bear a closer resemblance to those of BN than to those of obesity.

A review of RCT´s showed that presently cognitive behavioural treatment is the treatment of choice but interpersonal psychotherapy, self-help and SSRI´s seem effective. The first aim of treatment should be the cessation of binge eating. Treatment of weight loss may be offered to those who are able to abstain from binge eating.

Introduction

In the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) binge eating disorder (BED) is proposed as a new diagnostic category within the spectrum of eating disorders (American Psychiatric Association, 1994). The disorder falls into the category 'eating disorders not otherwise specified' (EDNOS). BED is not a formal diagnosis, the criteria described in Appendix B of the manual require further research.

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. Although the diagnosis BED was created with the obese in mind, obesity is not a criterion for BED. An overview of the proposed diagnostic criteria can be found in table 1.

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.

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. However, amongst clinicians there is considerable controversy about if and how the treatment demands of these patients

should be met. Nevertheless, eating disorder clinics have reluctantly begun treating patients with binge eating disorder. One reason for this reluctance is that subjects with binge eating disorder presenting for treatment in at eating disorder services are in general obese. Consequently these subjects have two problems: obesity and binge eating. Eating disorder practitioners are trained to treat psychiatric eating disorders and are inclined to leave the treatment of obesity to other specialists. The inverse seems to be true in the field of obesity. The identification of BED within patients presenting at an obesity clinic is important, because of the implications for treatment.

This paper will provide an overview of the research that has been done on BED in the decade following the first proposal of the diagnostic criteria by Spitzer and others in 1991. First of all, the epidemiological studies will be reviewed. Secondly, the aetiology will be discussed and the characteristics of patients with binge eating disorder will be compared to those of patients with obesity and to those of patients with bulimia nervosa. The differences and similarities will be discussed. Recommendations for improving the classification of BED will be given. Finally, the treatment and course of binge eating disorder will be reviewed.

Methods

The relevant literature was identified by a search of computerised databases (including MEDLINE, EMBASE, PsycLIT, Science Citation Index Expanded (SCI-EXPANDED) and Social Science Citation Index (SSCI)) and cross-referencing.

Results

Epidemiology

Only a few epidemiological studies have investigated the prevalence of BED in different populations i.e. in general populations and in specific populations (e.g. male, female, adolescent and obese populations). Three studies have examined the prevalence of BED in the general population (Spitzer et al., 1992; Spitzer, Yanovski, Wadden, & Wing, 1993; Hay, 1998). Spitzer and others (1992; 1993) reported a prevalence of 3.3% in their first study and 2% in their second study. In an Australian community-based survey 1% of the population had BED (Hay, 1998).

Other studies examined random samples of women in France (Basdevant et al., 1995), Norway (Götestam & Agras, 1995) and Austria (Kinzl, Traweger, Trefalt, Mangweth, & Biebl, 1999) and found that 0.7%, 3.2% and 3.3% respectively met the criteria for BED. Two studies investigated the prevalence of eating disorders among young female students and found that 1.0% (Rosenvinge, Borgen, & Borresen, 1999) and 0.2% (Cotrufo, Barretta, Monteleone, & Maj, 1998) satisfied the BED criteria. Kinzl et al. (1999) reported a prevalence of 0.8% in an all male community sample. BED seems to be distributed equally among the sexes (Spitzer et al., 1993; Hay, 1998; Striegel-Moore, 1995).

The population that has been investigated most consists of obese adults seeking weight loss treatment. The reported prevalence rates vary greatly and range from 1.3% to 30.1% (Spitzer et al., 1992; Spitzer et al., 1993; Ramacciotti et al., 2000; Basdevant et al., 1995; Ricca et al., 2000; Varnado et al., 1997). BED is associated with obesity and unstable weight (Fairburn et al., 1998; Spitzer et al., 1993). Also, BED seems to be more prevalent as the degree of obesity increases (Telch, Agras, & Rossiter, 1988; Hay, 1998; Hay & Fairburn, 1998). Probably the degree of obesity is different in the populations that have been investigated.

Prevalence rates of eating disorders have also been investigated in patients with diabetes mellitus. Herpertz et al. (2000) found that 10% of the type II diabetic sample they studied, had an eating disorder, which was characterized by binge eating (5.9 to 7.8% had a lifetime diagnosis of BED). All these patients were overweight or obese. Mannucci et al. (1997) found a prevalence of 5.7% for BED in type II diabetics. BED seems to precede type II diabetes in most patients and could be one of the causes of obesity that often precedes type II diabetes (Herpertz et al., 1998). There does not seem to be an increase of eating disorders in type I diabetes mellitus and vice versa (Nielsen & Molbak, 1998).

In all studies but one, the diagnosis of BED was determined solely on the basis of answers to questionnaires. Furthermore, most samples were relatively small. The study by Hay (1998) was the only study with a reasonably large sample (N=3000) and was the only one, which used both a questionnaire and an interview.

Aetiology

In a community-based, retrospective case-control study Fairburn and others (1998) aimed to identify specific risk factors for BED. They compared subjects with BED with healthy controls, subjects with other psychiatric disorders and subjects with bulimia nervosa. The findings support the prediction that BED would be associated with exposure to risk factors that increase the risk for psychiatric disorder in general and with those that increase the risk for obesity.

Little is known about the family characteristics of BED patients. One study found that BED subjects rated their family environment as less supportive and cohesive, and less engendering of direct and open expression of feelings than healthy controls. The BED group scored worse than other eating disorder groups (Hodges, Cochrane, & Brewerton, 1998). One study investigated familial tendency for BED and the risk for other psychiatric disorders, but failed to show this (Lee et al., 1999b).

In BN most individuals start dieting prior to the onset of binge eating (Mussell et al., 1997; Marcus et al., 1995; Haiman & Devlin, 1999). However, a fairly large subgroup of the individuals with BED start binge eating prior to the onset of dieting (35-54%)(Grilo et al., 2000; Abbott et al., 1998; Spurrell et al., 1997; Mussell et al., 1995). Dieting seems to play a role in the aetiology of BED, but research does not indicate that dieting is always a key factor in BED, as it seems to do in BN (Howard & Porzelius, 1999). The binge-first group seems to diet

because they binge, not binge because they diet (Abbott et al., 1998). For subjects who start binge eating before dieting, binge eating seems to be the primary symptom that leads to weight gain. Obesity is found to develop several years after the onset of binge eating (Haiman et al., 1999; Mussell et al., 1995).

Characteristics of binge eating disorder

The characteristics of BED are similar to those of both obesity and bulimia nervosa. The central criterion for BED is the occurrence of episodes of binge eating which is also an essential criterion for bulimia nervosa.

Obesity with and without binge eating disorder - Obese patients with BED have less self-esteem and greater depressive symptomatology than obese persons without BED; they also have more comorbid psychiatric disorders, in particular affective disorders and personality disorders (de Zwaan & Mitchell, 1992; Mitchell & Mussell, 1995; Yanovski et al., 1993; Kuehnel & Wadden, 1994; Striegel-Moore, Wilson, Wilfley, Elder, & Brownell, 1998; Telch et al., 1998). Individuals with BED are more likely to report dietary disinhibition (Marcus, Wing, & Lamparski, 1985; Wadden, Foster, & Letizia, 1992), excessive concern with shape and thinness and difficulty in interpreting visceral sensations related to hunger and satiety (Marcus et al., 1990; Kuehnel et al., 1994). Furthermore, subjects with BED have a tendency to experience negative affect in response to perceived evaluation by others of weight-related behaviour (Eldredge & Agras, 1997). They are more likely than obese persons without BED to become overweight at a younger age (Spitzer et al., 1993), to start dieting at a younger age and to spend more time on fruitless attempts to lose weight (de Zwaan et al., 1994; Kuehnel et al., 1994; Grissett & Fitzgibbon, 1996; Howard et al., 1999). Overweight subjects with BED consider themselves more overweight and fatter than non-binge subjects with a comparable weight (Mussell et al., 1996a). Others (Masheb & Grilo, 2001) found that individuals with BED are accurate reporters of weight. There is even a tendency for underreporting weight.

Two studies have recorded food intake in a laboratory (Goldfein, Walsh, LaChaussee, & Kissileff, 1993; Yanovski et al., 1992). Subjects with BED and obesity had a higher energy intake than subjects with obesity alone. Also, the recorded energy intake of subjects with BED on non-binge days was greater than that recorded by subjects without the disorder. Subjects with BED consumed more dessert and more snack foods (more fat and less protein) than did obese control subjects. The total intake was larger and duration of the episodes was longer in subjects with BED. Furthermore, for both subjects with and without BED, it seems that energy intake increases with the degree of obesity (Mitchell, Crow, Peterson, Wonderlich, & Crosby, 1998; Telch et al., 1994a). The degree of the psychopathology seems to be related to the degree of binge eating and not to the degree of obesity (Hay et al., 1998). Also, obese BED patients seem to be significantly older than normal-weight BED patients which suggests that binge eating may represent a risk factor for weight gain or obesity (Masheb & Grilo, 2000).

Binge eating disorder compared to bulimia nervosa - The criteria for BED are derived from the criteria for bulimia nervosa (BN). Most studies that compare BED and BN use the criteria for BN in the DSM-III-R, which makes no distinction between the purging and non-purging subtypes of BN. The DSM-IV does make a distinction between these subtypes. Purging bulimics engage in self-induced vomiting, misuse of laxatives, diuretics or enemas to compensate for binge eating. Nonpurging bulimics use inappropriate compensatory behaviours like fasting or excessive exercise. Several studies have compared BED patients with purging bulimics (Marcus, Smith, Santelli, & Kaye, 1992; Kirkley, Kolotkin, Hernandez, & Gallagher, 1992; Fichter, Quadflieg, & Brandl, 1993; Raymond, Mussell, Mitchell, & de Zwaan, 1995; Tobin, Griffing, & Griffing, 1997; Mitchell et al., 1999; LaChaussee, Kissileff, Walsh, & Hadigan, 1992; Goldfein et al., 1993; Mussell et al., 1995), and a few studies have compared BED patients to non-purging BN patients (Tobin et al., 1997; Hay et al., 1998; Santonastaso, Ferrara, & Favaro, 1999). In order to interpret the results of these studies correctly it is important to know which version of the DSM was used.

Compared to subjects with BN (DSM-III-R) subjects with BED seem less anxious about their eating patterns and bodyweight, feel less guilty about being overweight, are less preoccupied with their eating behaviour, have a better overall opinion of themselves, are able to perceive internal states more accurately, are more socially adjusted, and are more comfortable in maintaining interpersonal relationships (Raymond et al., 1995). BED subjects have lower levels of dietary restraint than BN subjects (Masheb et al., 2000). BED patients seem to show fewer comorbid psychiatric symptoms than BN purging or non-purging patients (DSM-IV)(Tobin et al., 1997). However, Marcus et al. (1992) found that obese women seeking treatment for binge eating reported levels of eating disorder psychopathology that were comparable to those of normal weight bulimia nervosa patients (DSM-III-R). Nonpurging bulimics and BED subjects do not seem to differ in clinical and psychological characteristics, such as psychiatric symptoms, frequency of bingeing, and impulsiveness traits. However, on many of the variables, the BED group showed a significantly greater variance (Santonastaso et al., 1999).

Energy intake during an episode of binge eating seems to be different in BN (DSM-III-R) and BED. In a laboratory subjects were asked to binge on ice cream. Subjects with BN (DSM-III-R) consumed four times as much as normal weight healthy controls (LaChaussee et al., 1992). The same research group reported that subjects with BED ate only half the amount of ice cream eaten by subjects with BN (Goldfein et al., 1993). One study compared the quality and quantity of binges reported in individuals with BED and BN (Fitzgibbon & Blackman, 2000). Binges of subjects with BN were higher in carbohydrates and sugar content than those of subjects with BED. No difference was observed in the mean number of consumed calories.

Studies which compared BED, BN and obesity (Fichter et al., 1993; Kirkley et al., 1992; Howard et al., 1999) indicate that individuals with BED exhibited levels of psychopathology

that fell somewhere between the high levels reported by individuals with BN and the low levels reported by obese individuals without binge eating. However, the characteristics of subjects with BED seemed to bear a closer resemblance to those of subjects with BN than to those of obese individuals.

It has also been suggested that bulimic eating disorders exist on a continuum of clinical severity, which starts with bulimia nervosa purging type (most severe), passes through bulimia nervosa nonpurging type (intermediate severity), and finishes with binge eating disorder (least severe)(Hay et al., 1998).

Classification

The definition of binge eating (criterion A) is identical for BED and bulimia nervosa. The operationalisation of the binge eating criteria in BED poses several problems (see also table 1). By comparison, episodes of binge eating are easier to define in bulimia nervosa because the binge eating is followed by compensatory behaviour like vomiting. In BED both the beginning and ending of an episode of binge eating are less clear. Also, there is no consensus about the best method for assessing binge behaviour, i.e. is it better to count the number of binge days or the number of binge episodes. This is also reflected in the discrepancy between criterion A1 and D. There is still a considerable debate about how to classify a binge episode in general, regardless of the diagnosis. The criteria that are most salient in identifying binges are the amount of food eaten, the time frame of the eating episode and loss of control. Of these variables, the impact of quantity and loss of control criteria are more apparent than the temporal criterion (Johnson, Boutelle, Torgrud, Davig, & Turner, 2000). It is important that the criteria of these binges should be well described first since we would not wish normal gluttony to be classed as a psychiatric illness (Fairburn et al., 1993b). The EDE (Eating Disorder Examination) scheme for classifying episodes of overeating (Cooper & Fairburn, 1987; Fairburn & Wilson, 1993c), which distinguishes between objective and subjective bulimic episodes, can also be helpful (see table 2).

Table 2: The EDE scheme for classifying episodes of overeating (Cooper et al., 1987)

	Large (EDE definition)	Not 'large' but viewed by subject as excessive
'Loss of control'	Objective bulimic episodes	Subjective bulimic episodes
No 'loss of control'	Objective overeating	Subjective overeating

Criterion B does not seem to represent a distinct criterion since B1, B2 and B3 refer to aspects of binge eating behaviour already described in criterion A and B4 and B5 refer to

aspects of distress mentioned in criterion C. It might be more correct to discuss these features not as a separate criterion but included respectively in criteria A and C.

What is meant with 'marked distress regarding binge eating' in criterion C is also not entirely clear. Distress might refer to an emotional state or it might describe impairment of the patient's functioning in social situations or at work due to binge eating (de Zwaan, Mitchell, Specker, & Pyle, 1993).

According to criterion E, a person should not be diagnosed as having BED if he/she engages in regular inappropriate compensatory behaviour. However, it is unclear what kind of compensatory behaviour is regarded as inappropriate. For instance nonpurging compensatory behaviours, like excessive exercise and misuse of diet pills, have never been clearly defined (Fairburn et al., 1993b; de Zwaan, Mitchell, Raymond, & Spitzer, 1994). Furthermore, there is no definition of the term regular. The term regular implies that some compensatory behaviour is permitted for the diagnosis BED. In DSM-IV fasting has replaced the phrase dieting to facilitate the distinction between BED and nonpurging BN (de Zwaan, 1997).

Although these criteria obviously need further study, there is considerable evidence that BED represents a valid eating disorder category. Researchers still use slightly different criteria for BED in their separate studies. Future research should refine the criteria, which should eventually lead to a more reliable determination of the diagnosis BED.

Randomised controlled clinical trails for Binge Eating Disorder

The treatment of BN has been researched extensively and there have also been numerous controlled treatment studies (Schmidt, 1998), however far less attention has been paid to BED (Wilfley & Cohen, 1997). Because BED is more similar to BN than to obesity without binge eating, 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 gold standard to which other treatments are compared (Wilfley et al., 1997). Many individuals with BED seek help for overweight. Treatment of obesity focuses on the reduction of caloric intake, encourages a shift to a low fat diet, addresses any medical contribution to the condition and initiates exercise. However, the underlying behavioural disturbances or the social and psychological consequences of obesity are often neglected. If the treatment of obese subjects with BED focuses only on reduction of bodyweight and does not address binge eating or underlying problems, binge eating continues or even worsens (Romano & Quinn, 1995; Howard et al., 1999). Weight-loss programmes seem to have little effect on the reduction of binge eating in obese subjects with BED (Kirkley et al., 1992).

The studies under discussion in this review are those in which BED is classified by means of the DSM-IV. These treatment studies have focused only on overweight women with

BED. Unfortunately, short-term data are very limited and there are almost no long-term data. Finally, the studies are inconsistent in their definitions of the disorder and do not use the same outcome measures for success.

Psychological clinical trials

To date there have been seven controlled clinical trials in which CBT was evaluated in BED patients (Telch, Agras, Rossiter, Wilfley, & Kenardy, 1990; Wilfley et al., 1993; Agras et al., 1994a; Agras et al., 1995; Eldredge et al., 1997; Peterson et al., 1998; Carter et al., 1998). CBT has been compared to interpersonal therapy, weight loss therapy and antidepressant medication. These studies are summarized in table 3.

Table 3: Controlled clinical psychological trials for binge eating disorder

Authors	Subjects & Design	Primary outcome measures	Outcome
Telch et al., 1990	44 obese women with nonpurging bulimia nervosa (DSM-III-R) 1. Group CBT, 10 sessions, (N=23) 2. Waiting list (N=21)	frequency binge of eating episodes	CBT > waiting list (CBT: 94% reduction in binge eating episodes, 79% abstinent; waiting list no reduction)
Wilfley et al., 1993	56 obese women with nonpurging bulimia nervosa (DSM-III-R) 1. Group CBT, 16 sessions (N=18) 2. Group IPT, 16 sessions (N=18) 3. Waiting list (N=20)	frequency of binge days	CBT = IPT > waiting list Abstinence: CBT:28%; IPT: 44%; Reduction inbinge eating episodes: CBT: 48%; IPT:71% → neither is statistically significant At 1 year follow-up 50% reduction in binge eating in both CBT-treated and IPT-treated patients. No significant weight loss
Telch et al., 1994a	108 obese women with binge eating disorder (DSM-IV) 1. WLT, 30 sessions, (N = 37) 2. Group CBT, 12 sessions, followed by WLT, 18 sessions (N=36) 3. Group CBT, 12 sessions, followed by WLT, 18 sessions, plus desipramine, 6 months (N=36)	1.weight 2. frequency binge eating episodes	WLT = CBT/WLT = CBT/WLT/desipramine Abstinence: respectively 19%, 37%, 41% no statistical significance Weight loss res. 6.0 kg, 1.6 kg and 3,7 kg

Authors	Subjects & Design	Primary outcome measures	Outcome
Agras et al., 1995	50 obese subjects (43 women/7 men) with binge eating disorder (DSM-IV) 1. Group CBT 12 sessions (N=39) plus weekly weighings, an exercise programme and information about low fat food If successful: WLT, 12 sessions If not successful: group IPT, 12 sessions 2. Waiting list (N=11)	1. Frequency of binge days 2. Weight	Reduction in binge days: CBT 77%; waiting list 22%. IPT did not add to the effect of CBT Weight: significant weight loss in CBT/WLT, no differences in weight in CBT/IPT
Eldredge et al., 1997	46 obese subjects (44 women / 2 men) with BED (DSM-IV) 1. Group CBT, 12 weeks (N=36) If successful: WLT, 12 sessions (N=18) If not successful: group CBT, 12 sessions (N=18) 2. Waiting list (N=10)	Frequency of binge eating episodes	After 12 weeks: CBT: 50% abstinent and 68.2% reduction in binge eating; Waiting list: 19.8% reduction in binge eating. No reduction in weight After 24 weeks: CBT: 67% abstinent
Peterson et al., 1998	61 women with BED (DSM-IV), self-help based on CBT techniques. Each session: psycho-education and group discussion (14 sessions). 1. Therapist-led (TL) (N=16) 2. Partial self-help (PSH) (N=19) 3. Self-help (SH) (N=15) 4. Waiting list (WL) (N=11)	1. Frequency of binge eating episodes 2. Duration of binge eating episode	TL=PSH=SH>WL (abstinence 69%, 68%, 87%, 13% respectively) Significant reduction in frequency and duration of binge eating episodes
Carter et al., 1998	72 subjects with BED (DSM-IV), self-help based on CBT techniques (12 weeks). 1. Pure self-help (PSH) (N=24) 2. Guided self-help (GSH) (N=24) 3. Waiting list (WL) (N=24) (after 12 weeks these subjects were randomly assigned to one of the two conditions)	Frequency of binge eating episodes	Abstinence form binge eating: GSH>PSH>WL (50%, 43% and 8% respectively) 6 months follow-up: abstinence form binge eating: GSH>PSH (50% and 40% respectively)

CBT=Cognitive Behavioural Therapy; IPT=Interpersonal Psychotherapy; WLT=Weight Loss Treatment; WL=Waiting list

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). The focus is on the factors and processes that maintain the eating problem rather than on those that operated earlier in its evolution. Fairburn (1981) was the first to describe CBT as a useful therapy for bulimia nervosa. Three stages in the treatment can be distinguished. 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.

Klerman and colleagues were responsible for developing IPT (Klerman, Weissman, Rounsaville, & Chevron, 1984). It was designed for the treatment of depressed outpatients and is based on an interpersonal view of the maintenance of depression. The treatment was modified to suit patients with bulimia nervosa (Fairburn et al., 1991). Later Wilfley and colleagues (1993; 2000) modified IPT to a group format so that it could be used to treat groups of persons with BED. IPT uses techniques derived from psycho-dynamically oriented therapies, but the focus is on the patient's current circumstances and relationships. It is based on the assumption that psychiatric disorders are intimately related to disturbances in social functioning, which, in turn, may be associated with the onset and/or maintenance of the disorder. IPT involves well-defined treatment strategies, techniques, and a therapeutic approach to the resolution of problems within four social domains: grief, interpersonal disputes, role transitions, and interpersonal deficits. IPT is especially well-suited for binge eating disorder patients because it teaches: 1) the requisite skills for developing and sustaining satisfying relationships and 2) more effective strategies (than binge eating) for coping with social and interpersonal problems (Wilfley, Frank, Welch, Borman Spurrell, & Rounsaville, 1998).

Psychological treatment in general seems to cause a statistically significant reduction in binge eating compared to no treatment (reduction in number of episodes: 68-77%; abstinence from binge eating: 40-87%). Two studies combined psychological treatment with a weight loss program (Agras et al., 1995; Eldredge et al., 1997). Treatment of obese subjects with BED seems to be more successful if binge eating is treated before any attempts are made to lose weight. Agras and others (1995) investigated the effectiveness of IPT in treating overweight patients with binge eating disorder who did not stop binge eating after 12 weeks of CBT. IPT did not lead to further improvement in those who did not improve with CBT.

Two studies (Peterson et al., 1998; Carter et al., 1998) examined the effectiveness of a self-help format (CBT) in the treatment of BED. Self-help formats seem to be effective. However, some caution is needed with the interpretation of the high abstinence rates, because the participants in these 'self-help' studies were probably less severely ill than those in the other studies (for example, subjects in the study by Carter and others (1998) had not received any prior treatment).

Pharmacological clinical trials

The number of double-blind placebo-controlled pharmacological trials is also small (see table 4). Drugs which have been examined are three selective serotonine reuptake inhibitors

(SSRI´s; fluoxetine, fluvoxamine, sertraline)(Greeno & Wing, 1996; Hudson et al., 1998; McElroy et al., 2000) and an appetite suppressant with serotonin-enhancing properties (d-fenfluramine)(Stunkard, Berkowitz, Tanrikut, Reiss, & Young, 1996).

Fluoxetine seemed to reduce dietary intake but did not affect the frequency of binge episodes. This finding suggests that fluoxetine affects satiety, not hunger (Greeno et al., 1996). Fluvoxamine was found to be effective in reducing the frequency of binge episodes and in lowering Clinical Global Impression (CGI) severity scores (Hudson et al., 1998). Finally, sertraline seemed to be effective and well tolerated, although the number of participants in that study was low (McElroy et al., 2000).

D-Fenfluramine reduced the frequency of binge eating in obese women with BED, but failed to reduce their bodyweight (Stunkard et al., 1996).

A striking finding in these pharmacological studies is a high placebo-effect. All studies had a single-blind lead-in period from 1 to 4 weeks. After this lead-in period 42 to 44% of the participants no longer met the DSM-IV-criteria for BED.

In all studies the drugs under investigation seemed to be more effective than placebo with regard to the primary outcome measures. However, no long-term effects were found. Further, drugs did not seem to bring about a reduction in bodyweight. Disadvantages of these studies were the small number of participants and the short duration of the trials.

Course of binge eating disorder

Two studies investigated the natural course of BED in the general population. Fairburn et al. (2000) followed 102 subjects with BED for five years. After five years only 10% of these subjects still fulfilled the criteria for BED (1 subject (3%) fulfilled the criteria for bulimia nervosa and 2 subjects (5%) for 'eating disorders, not otherwise specified'. In total 18% had an eating disorder of clinical severity. At the 5 year follow-up 77% of the group was abstinent (i.e. no objective bulimic episodes). However, the group as a whole became heavier during the five years and a large proportion tended to have a BMI over 30 (obesity) (22% at recruitment compared to 39% at follow-up). Striking was the finding that only 8% had been treated for an eating disorder during these five years.

Cachelin and others (1999) examined women with BED in the general population for a period of six months. At the six-month follow-up 52% of these women suffered from full-syndrome BED, whereas 48% appeared to be in partial remission. Treatment seeking in general did not appear to be associated with improvement in BED over a relatively short time period.

Table 4: Double- blind placebo-controlled clinical trials

Authors	Subjects & Design	Primary outcome measures	Outcome
Greeno et al., 1996	38 overweight women with BED (DSM-IV) and 32 without BED (6 weeks) 1. Fluoxetine 60 mg / day (N=34) 2. placebo (N=36)	dietary intake	Fluoxetine > placebo for both groups Fluoxetine did not affect frequency of binge episodes or mood
Stunkard et al., 1996	24 subjects with BED (DSM-IV) (8 weeks) 1. d-Fenfluramine 30 mg/day (N=12) 2. placebo (N=12)	frequency of binge episodes	d-Fenfluramine > placebo no effects left after follow-up at 1 and 4 months no reduction in weight
Hudson et al., 1998	67 subjects with BED (DSM-IV) (9 weeks) 1. Fluvoxamine 50-300 mg/day (N=29) 2. placebo (N-38)	1. frequency of binge episodes 2. Clinical Global Impression (CGI)	Fluvoxamine > placebo (also BMI) more drop-outs in Fluvoxamine-group because of adverse medical event
McElroy et al., 2000	34 subjects with BED (DSM-IV) (6 weeks) 1. Sertraline 50-200 mg/day (N=18) 2. Placebo (N=16)	1. frequency of binge episodes 2. severity 3. global improvement 4. BMI	Sertraline > placebo

Fichter and others (1998) assessed the course and outcome of 68 women with BED over a period of six years after intense inpatient treatment. In general, the majority of these patients showed substantial improvement during treatment, a slight (in most cases non-significant) decline during the first 3 years after treatment ended and further improvement and stabilization in the 4, 5 and 6 years following treatment. At the six year follow-up only 6% fulfilled the criteria for BED. In total 20% met the criteria for some eating disorder according to the DSM-IV.

The studies mentioned above could be taken to indicate that treatment worsens the course of BED, since a higher percentage of subjects improved without treatment. However, subjects seeking help for BED seem to have more severe problems than subjects with BED in the general population (Fairburn, Welch, Norman, O'Connor, & Doll, 1996).

Discussion

Binge eating disorder and obesity are obviously associated with each other. The prevalence of BED seems to increase with the degree of obesity. In almost half of the obese subjects with BED the onset of binge eating is prior to the onset of obesity. This implicates

that binge eating might be a primary symptom that leads to weight gain and obesity. How overweight plays a part in the development and maintenance of BED needs to be investigated more precisely.

Most studies have investigated BED in populations of obese women. However, the majority of the subjects with BED identified in community samples are not obese. Further, BED seems to be equally distributed between the sexes. Perhaps women with BED and obesity seek help more readily than men. Another possible explanation is that women are more easily diagnosed with BED than men are. More men should be included in future research studies. Also practitioners should be more aware of the possible existence of binge eating in men.

A striking finding is the improvement of BED in untreated subjects in the general population. However, these patients showed a considerable increase in (over)weight. Binge eating engenders an increase in bodyweight in normal weight subjects. Both the shame associated with binge eating and the increase in bodyweight may lead to a negative self-evaluation. Subsequently, a vicious spiral may develop. The development of obesity with its personal, social and health consequences may be a better predictor for help-seeking behaviour than the occurrence of binge eating.

BED holds an intermediate position between bulimia nervosa and obesity with regard to the severity of the psychopathology. However, the characteristics of obese BED patients seem to be more similar to the characteristics of BN patients than to those of obese subjects.

Patients with BED often suffer from two problems: binge eating and obesity. The identification of BED amongst patients presenting at obesity services is important. The first aim of treatment should be the cessation of binge eating. Treatment directed at weight loss may be offered to those patients who are able to abstain from binge eating.
Cognitive behavioural psychotherapy is currently the treatment of choice for binge eating. However, interpersonal psychotherapy, self-help based on techniques of CBT and selective serotin reuptake inhibitors also seem to be promising forms of treatment.

Chapter 3

The Empirical Status of Binge Eating Disorder

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Introduction

In the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association (DSM-IV) Binge Eating Disorder (BED) is proposed as a new diagnostic category requiring further study and as an example of Eating Disorder Not Otherwise Specified (ED-NOS)(American Psychiatric Association, 1994). 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. No comparable diagnostic category exists in ICD-10 (World Health Organization, 1992).

BED was introduced as a new eating disorder about ten years ago. The aim of this chapter is to give an overview of the results and to discuss the empirical status of BED after this decade of research. What is the empirical evidence for and against the state currently defined as BED in the DSM-IV? In this chapter I will focus on the present status of BED as an eating disorder. The issue of the relationship between BED and obesity will be discussed elsewhere in the book.

The Diagnostic and Statistical Manual of Mental Disorders (DSM)

Classifying mental disorders by means of the DSM is one of the many methods of classification. Since the publication of the third edition of the DSM the taxonomy proposed by the American Psychiatric Association has become more dominant than anyone would have believed possible in the light of the limited impact of the first and second editions (American Psychiatric Association, 1952; American Psychiatric Association, 1968; Follette & Houts, 1996). In its proposal for the DSM-III the American Psychiatric Association considered mental disorders as medical disorders (American Psychiatric Association, 1980). Although there is not yet an agreement about the diagnosis and the criteria, in actual practice binge eating disorder is already accepted as an eating disorder. However, there is considerable debate about how much effort should be made to treat these patients in an eating disorder clinic. If patients with a binge eating disorder are treated within an eating disorder clinic there is also discussion about the kind of therapy, which is suited for these patients (Dingemans, Bruna, & van Furth, 2002).

Objections to this viewpoint came from the American Psychological Association. The DSM reflects the underlying model of traditional medicine. In order to gain wide acceptance of the system the task force of the DSM decided to abandon the theoretical (medical) view and to cease referring to mental disorders as a subset of medical disorders. This decision largely explains the syndrome-based and non-theoretical nature of the DSM (Follette et al., 1996).

According to the DSM-IV a mental disorder is defined as *'a clinically significant behavioural or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g. a painful symptom) or disability (i.e. impairment in one or more important areas of functioning) or with a significant increased risk of suffering death, pain, disability, or an important loss of freedom. In addition,*

this syndrome or pattern must not be merely an expectable and culturally sanctioned response to a particular event, for example, the death of a loved one. Whatever its original cause, it must currently be considered a manifestation of a behavioural, psychological, or biological dysfunction in the individual' (page xxi). The definition of dysfunction is crucial to the definition because something is not a disorder unless something has gone amiss in the person concerned (Follette et al., 1996).

The proposal of Spitzer and others

In 1991 Spitzer and others suggested that BED should be included in the DSM-IV. The rationale for their proposal was that many individuals with marked distress about binge eating could not be diagnosed as having 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. The authors indicated that such patients are common among the obese involved in weight control programmes and/or belonging to overeaters anonymous (Spitzer et al., 1992; Spitzer et al., 1993). Although the diagnosis BED was formulated with the obese in mind, obesity is not a criterion for BED.

For inclusion in a new version of the DSM, the 'new' diagnostic is required to describe a pattern of symptoms not captured in the existing categories (Pincus, Frances, Davis, First, & Widiger, 1992). Pincus and others (1992), who served on the DSM-IV Task Force, stated that for a new category to be considered for inclusion in the DSM-IV "there must be solid evidence that the diagnosis is useful in predicting prognosis, treatment selection or outcome".

In 1993 Fairburn and others considered whether Pincus' arguments would apply to the proposed addition of BED to the DSM. One argument against new diagnoses is that if they are rare they may add unnecessary complexity to the already cumbersome system of classification and be irrelevant for clinical use. Furthermore, incorporation of new categories is likely to increase the overall prevalence of mental disorders. The addition of new and unproven diagnoses carries the risk of trivializing the construct mental disorder and/or its misuse. This is relevant for BED since we would not wish normal gluttony to be classed as a psychiatric disorder (Fairburn et al., 1993b). The criteria proposed by Spitzer et al. (1992) have been designed to minimize this risk.

However, Fairburn and others (1993b) argued that adding BED to the section of eating disorders does not make it very complex because the present scheme is relatively simple.

A second argument against adding new diagnostic categories is that new diagnoses are generally proposed by experts in the field concerned and are subsequently used by less expert assessors who may identify more false positives. These inaccurate diagnoses may lead to faulty treatments.

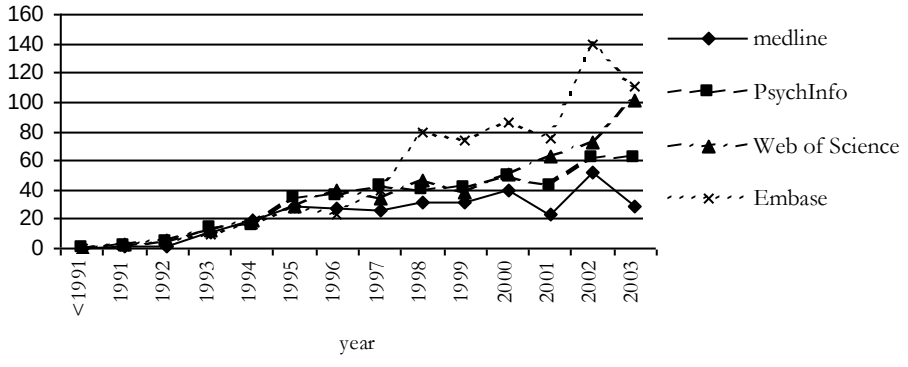
A third argument put forward by Fairburn and others is that 'adding unproven diagnostic categories may confer upon such categories an approval that they do not merit yet'.

They argue that there is no evidence to suggest that delineating BED from ED-NOS is a useful or valid approach. This delineation "may impede efforts to devise better classificatory schemes since investigators will inevitably tend to define their samples along the new lines".

A fourth argument concerns the risk of definitional overlap across related categories. Fairburn and others (1993b) argue that the delineation of BED could cause definitional overlap between the non-purging type of bulimia nervosa and BED. Bulimia nervosa non-purging type is distinguished from BED by the presence of compensatory behaviours (fasting and excessive exercise) and/or undue emphasis on self-evaluation of body shape or body weight. Although the existence of substantial differences between bulimia nervosa non-purging type and binge eating disorder has been confirmed (Santonastaso et al., 1999), it is difficult to draw clear boundaries between these two categories. It is unclear when these distinguishing behaviours are severe enough to warrant the diagnosis of bulimia nervosa non-purging type rather than binge eating disorder.

Another reason for adding a new diagnosis to the DSM, is to initiate research in the field. The DSM-III and DSM-III-R have been facilitators of research in areas that would have remained unresearched if they had not been included in an official nomenclature. Some see this as one of the most important goals of the DSM. Others, however, think that research should drive DSM and not the other way around. It makes no sense to include a category for which there is no empirical support (Pincus et al., 1992). Once categories are included in the DSM they are not easily deleted (Blashfield, Sprock, & Fuller, 1990). The insertion of BED in the DSM-IV has led to a considerable increase in the amount of research into BED over the last decade. The term binge eating disorder was first used in a paper that appeared in 1991. The number of papers in which BED has been investigated has grown immensely over the past ten years (see overview figure 1).

Figure 1: Overview of papers with 'binge eating disorder' as keyword



Classification criteria

The Eating Disorders Work Group of the DSM-IV Task Force in conjunction with Spitzer et al. (1992) developed preliminary criteria for the new eating disorder diagnosis designed to identify "the many people who have problems with recurrent binge-eating, but who do not engage in compensatory behaviours of bulimia nervosa, vomiting or the abuse of laxatives" The criteria for the diagnosis of binge eating disorder were adapted from those for BN , extra criteria being added to define the differences between the two disorders.

The definition of binge eating (criterion A) is identical for BN and BED. However, in practice, binge eating among BED patients does not always conform to the requirement of the consumption of "a large amount" of food during a "discrete period of time", such as two hours. Rossiter and others (1992) reported that many obese patients who overeat with a sense of loss of control consume quantities of food that would not be described as 'large' during any 'discrete period of time' but would be considered excessive over the course of the day. This pattern of overeating has been termed 'grazing' and is a frequent occurrence within the obese binge eating population (Marcus et al., 1992).

Binge eating episodes are required to be associated with behavioural symptoms of loss of control (criterion B). Spitzer and others (1992; 1993) felt that criterion B (symptoms of loss of control) should be included so as to set a high threshold for the diagnosis of binge eating disorder and to ensure that normal gluttony would not be classed as binge eating disorder. However, this criterion does not seem to be fully distinct from criterion A (2) (a sense of loss of control) or criterion C (feeling of distress regarding binge eating). Criteria B1, B2, and B3 refer to aspects of loss of control whilst eating a large amount of food in a discrete period of time (criterion A). Criteria B4 and B5 are related to characteristics of distress described in criterion C.

The distress criterion was included in the DSM-IV proposed criteria for BED in order to minimize false positives. Removal of this criterion would have increased the number of individuals meeting criteria for the disorder by 10% in a weight control sample and by more than 100% in a community sample in Spitzer's field trail (1992). However, it is not clear what is meant by 'marked distress'. Distress may refer to emotional distress or impaired social or occupational functioning as a consequence of binge eating behaviour. Does distress reflect the patient's self-report of his/her emotional state or does it also require social and/or occupational impairment due to binge eating? Furthermore, de Zwaan (1997) stated that by including the distress criterion one may identify individuals with high levels of general distress which is not directly related to binge eating. For a diagnosis of BED, binge eating is required to occur on two days a week (criterion D) whereas patients with BN are required to have two episodes per week. This definition is based on the rationale that BED patients may have more difficulty in recalling and labelling binge eating episodes than bulimia nervosa patients (Rossiter et al., 1992). The end of a binge-eating episode in BN is often characterized by purging behaviour, whereas in BED the termination of a binge episode is not punctuated by such

behaviour. However, defining the frequency of binge eating in terms of the number of days on which the binge occurs seems to allow binges to be non-episodic, lasting as long as an entire day.

The twice-weekly frequency criterion for binge eating episodes is arbitrary and has no empirical support in cases of BN or BED. Various authors have found that raising the frequency criteria of binge eating episodes from once or twice a week did not change the pattern on measures of psychopathology or treatment outcome in binge eaters (Wilson, Nonas, & Rosenblum, 1993; Striegel-Moore et al., 2000; Striegel-Moore et al., 1998; Garfinkel et al., 1995).

Two binge eating days per week are required to occur during a six-month period in BED whereas a three-month period is specified for BN The minimum duration of six months is required in order to ensure that transitory episodes of binge eating are not diagnosed as BED. Nevertheless, BED has been found to have a natural course that has a tendency to remit (Hay, Fairburn, & Doll, 1996; Cachelin et al., 1999; Fairburn et al., 2000).

DSM-IV proposed diagnostic criterion E (which states that the diagnosis of binge eating disorder should not be made if the patient engages in regular inappropriate compensatory behaviours also seen in BN (self-induced vomiting, laxative abuse, fasting, excessive exercise) or if he/she suffers from anorexia nervosa. However, there is no clear definition of what is meant by 'inappropriate' compensatory behaviours. Some compensatory behaviour in the obese is not necessarily inappropriate. Moreover, there is no definition for the term regular. The term regular implies that some compensatory behaviour could be compatible with the diagnosis of BED.

Unlike BN, the DSM-IV research diagnostic criteria for BED do not include 'unduly influenced self-evaluation by body weight and body shape'. It is not clear why this criterion has been excluded from the proposed diagnostic criteria. However, Eldredge and Agras (Eldredge & Agras, 1996) suggest that it may be due to uncertainty regarding the impact of the level of obesity on such concerns. Although obesity is not among the diagnostic criteria for either disorder, fewer BN patients are overweight than BED patients. The strong association between BED and obesity may be related to a patient's failure to compensate for the increased calories consumed during the binge eating episodes. Despite omission of this criterion various authors have observed a significant association between binge eating disorder and an overconcern about body weight and body shape (Wilson et al., 1993; Cachelin et al., 1999; Marcus et al., 1992; Masheb et al., 2000; Striegel-Moore et al., 2000; Wilfley, Schwartz, Spurrell, & Fairburn, 2000b; van Hanswijck de Jonge, van Furth, Lacey, & Waller, 2003).

A comparison of characteristics of binge eating disorder and bulimia nervosa

Most studies that compared BED and BN used the criteria for BN in the DSM-III-R, which made no distinction between the purging and non-purging subtypes of BN. The DSM-IV does make such a distinction between these subtypes. Purging bulimics engage in self-

induced vomiting, misuse of laxatives, diuretics or enemas. Non-purging bulimics do not purge but do use other inappropriate compensatory behaviours (i.e. fasting or excessive exercise). Several studies have compared BED patients with bulimics (with no distinction between purging and non-purging subtype) (Marcus et al., 1992; Kirkley et al., 1992; Tobin et al., 1997; LaChaussee et al., 1992; Goldfein et al., 1993; Mussell et al., 1995), and a few studies have compared BED patients to purging (Fichter et al., 1993; Mitchell et al., 1999; Masheb et al., 2000) and non-purging BN patients (Tobin et al., 1997; Hay et al., 1998; Santonastaso et al., 1999). In order to interpret the results of these studies correctly it is important to know which version of the DSM was used.

In a study in which normal weight subjects with BN (DSM-III-R) were compared to obese subjects with BED, the latter seemed less anxious about their eating patterns and bodyweight, felt less guilty about being overweight, were less preoccupied with their eating behaviour, had a better overall opinion of themselves, were able to perceive internal states more accurately, were more socially adjusted, and were more comfortable in maintaining interpersonal relationships (Raymond et al., 1995). Both obese and non-obese BED subjects have lower levels of dietary restraint than subjects with BN purging type (Masheb et al., 2000). However, Marcus and others (1992) found that obese women seeking treatment for binge eating reported levels of eating disorder psychopathology that were comparable to those of normal weight BN patients (DSM-III-R). Similarly, a cross-sectional study (van Hanswijck de Jonge, 2002) comparing BED to BN and obesity reported no significant difference between the two eating disorders on overall restraint psychopathology, eating concern psychopathology, body weight and body shape psychopathology. In all instances both BED and BN patients scored significantly higher in eating disorder psychopathology than the non-binge eating obese population.

Energy intake during an episode of binge eating seems to be different in BN (DSM-III-R) and BED. In a laboratory subjects were asked to binge on ice cream. Subjects with BN (DSM-III-R) consumed four times as much as normal weight healthy controls (LaChaussee et al., 1992). The same research group reported that subjects with BED ate only half the amount of ice cream eaten by subjects with BN (Goldfein et al., 1993). One study compared the quality and quantity of binges reported in individuals with BED and BN (Fitzgibbon et al., 2000). Binges of subjects with BN included food that was higher in carbohydrates and sugar content than the binges of subjects with BED. No difference was observed in the mean number of calories consumed.

BED patients seem to show fewer comorbid psychiatric symptoms than BN patients with either purging or non-purging subtype (Tobin et al., 1997). Schmidt and Telch (1998) have documented higher levels of depression, impulsivity, self-defeating tendencies and lower levels of self-esteem in bulimia nervosa than in binge eating disorder. Similarly, Raymond and others (1995) reported higher levels of depression and anxiety in bulimia nervosa patients than in binge eating disorder patients. Another study (Tobin et al., 1997) compared purging BN to

non-purging BN, BED and Eating Disorder Not Otherwise Specified (ED-NOS) on the Hopkins Symptom checklist and a measure of borderline syndrome and depression. The BED patients were reported to display significantly less anxiety, paranoia and psychoticism than the other three groups. No other differences were found between the groups on general psychopathology on the remaining measures. Unlike previous studies, no significant differences were found between BED and the two BN subtypes. Another study (van Hanswijck de Jonge, 2002) could not distinguish between BN and BED on general psychopathology as measured by the SCL-90-R. Furthermore, no distinction was reported between the two disorders on total levels of impulsivity. However, both BN and BED patients scored significantly higher on both general psychopathology and levels of impulsivity than did a group of non-binge eating obese patients. A study by Santonastaso and others (1999) showed no difference between nonpurging bulimics and BED subjects on clinical and psychological characteristics, such as psychiatric symptoms, frequency of bingeing, and impulsiveness traits. However, on many of the variables, the BED group showed a significantly greater variance. Webber (1994) does not document any significant differences between bulimia nervosa and binge eating disorder.

Aetiology

In a community-based, retrospective case-control study, Fairburn and others (Fairbu1998) aimed to identify specific risk factors for BED. They compared subjects with BED with healthy controls, subjects with other psychiatric disorders and subjects with bulimia nervosa. Their findings support the prediction that BED is associated with exposure to risk factors that increase the risk of psychiatric disorder in general and that increase the risk of obesity.

Little is known about the family characteristics of BED patients. One study found that BED subjects rated their family environment as less supportive and cohesive, and less engendering of direct and open expression of feelings than healthy controls. The BED group scored worse than other eating disorder groups (Hodges et al., 1998). One study investigated familial tendency for BED and the risk of other psychiatric disorders, but failed to show this (Lee et al., 1999b).

In BN most individuals start dieting prior to the onset of binge eating (Mussell et al., 1997; Marcus et al., 1995; Haiman et al., 1999). However, a fairly large subgroup of the individuals with BED start binge eating prior to the onset of dieting (35-54%)(Grilo et al., 2000; Abbott et al., 1998; Spurrell et al., 1997; Mussell et al., 1995). Dieting seems to play a role in the etiology of BED, but research does not indicate that dieting is always a key factor in BED, as it seems to do in BN (Howard et al., 1999). The binge-first group seem to diet because they binge, not binge because they diet (Abbott et al., 1998). For subjects who start binge eating before dieting, binge eating seems to be the primary symptom that leads to weight

gain. Obesity is found to develop several years after the onset of binge eating (Haiman et al., 1999; Mussell et al., 1995).

Course of binge eating disorder

Two studies have investigated the natural course of BED in the general population. Fairburn and others (2000) followed 102 subjects with BED for five years. After five years only 10% of these subjects still fulfilled the criteria for BED (1 subject (3%) fulfilled the criteria for BN and 2 subjects (5%) for EDNOS . In total 18% had an eating disorder of clinical severity. At the 5-year follow-up 77% of the group was abstinent (i.e. no objective bulimic episodes). However, the group as a whole became heavier during the five years and a large proportion tended to have a BMI over 30 (obesity) (22% at recruitment compared to 39% at follow-up). It was striking that only 8% had been treated for an eating disorder during these five years.

Cachelin and others (1999) examined women with BED in the general population for a period of six months. At the six-month follow-up 52% of these women suffered from full-syndrome BED, whereas 48% appeared to be in partial remission. Treatment seeking in general did not appear to be associated with improvement in BED over a relatively short time period.

Fichter and others (1998) assessed the course and outcome of 68 women with BED over a period of six years after intense inpatient treatment. In general, the majority of these patients showed substantial improvement during treatment, a slight (in most cases non-significant) decline during the first 3 years after treatment ended and further improvement and stabilization in the 4, 5 and 6 years following treatment. At the six-year follow-up only 6% fulfilled the criteria for BED. In total 20% met the criteria for some eating disorder according to the DSM-IV.

The studies mentioned above could be taken to indicate that treatment worsens the course of BED, since a higher percentage of subjects improved without treatment. However, subjects seeking help for BED seem to have more severe problems than subjects with BED in the general population (Fairburn et al., 1996; Wilfley, Pike, Dohm, Striegel-Moore, & Fairburn, 2001). Furthermore, it is unclear from these studies whether objective binge eating behaviour is replaced by overeating or subjective binge eating behaviour (explaining weight increase) in those patients reporting abstinence from bingeing behaviour at follow-up.

Treatment

The treatment of BN has been researched extensively and there have also been numerous controlled treatment studies (Schmidt, 1998); however, far less attention has been paid to BED (Wilfley et al., 1997). Because BED is more similar to BN than to obesity without binge eating, 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. Many individuals with BED however seek help for overweight. Treatment of obesity focuses on the reduction of caloric intake, encourages a shift to a low fat diet, addresses any medical contribution to the condition and initiates exercise. The underlying behavioural disturbances or the social and psychological consequences of obesity are often neglected. If the treatment of obese subjects with BED focuses only on the reduction of bodyweight and does not address binge eating or underlying problems, binge eating continues or even worsens (Romano et al., 1995; Howard et al., 1999). Weight-loss programmes seem to have little effect on the reduction of binge eating in obese subjects with BED (Kirkley et al., 1992).

The studies that will be discussed here are those in which BED is classified by means of the DSM-IV. To date there have been seven randomized controlled clinical trials conducted in which the psychological treatment of BED has been evaluated (Agras et al., 1994a; Agras et al., 1995; Eldredge et al., 1997; Peterson et al., 1998; Carter et al., 1998; Peterson et al., 2001; Wilfley et al., 2002a).

Cognitive Behavioural Therapy (CBT) seems to cause a statistically significant reduction in binge eating compared to no treatment (reduction in number of episodes after treatment: 68-90%; abstinence from binge eating after treatment: 40-87%; reduction in number of episodes after waiting list: 8-22%).

Two studies combined cognitive behavioural therapy (CBT) with weight loss treatment (Agras et al., 1995; Eldredge et al., 1997). Treatment of obese subjects with BED seems to be more successful if binge eating is treated before any attempts are made to lose weight.

Two studies compared the efficacy of CBT versus Interpersonal Psychotherapy (IPT). Agras and others (Agras et al., 1995) investigated the efficacy of IPT in treating overweight patients with binge eating disorder who did not stop binge eating after 12 weeks of CBT. Subjects who were successful after 12 weeks CBT received weight loss treatment. IPT did not lead to further improvement in those who did not improve with CBT. Wilfley and others (Wilfley et al., 2002a) randomized 162 overweight patients with BED to either CBT or IPT. The frequency of binge eating dropped significantly in both groups after 20 weeks of treatment (abstinence: CBT= 79% versus IPT = 73%) and at one-year follow-up (abstinence: CBT = 59% versus IPT = 62%). No differences were found between the two groups.

Nauta and others (2001) investigated the effectiveness of cognitive therapy and behavioural therapy in a group of obese subjects with and without BED, who were recruited from an obese community sample. Cognitive therapy appeared to be more effective than behavioural therapy with regard to abstinence from binge eating at six months follow up (86% and 44% respectively). At the end of treatment no differences were found in the abstinence rates (67% and 44% respectively).

Two studies (Peterson et al., 1998; Carter et al., 1998; Peterson et al., 2001) examined the efficacy of a self-help format (CBT) in the treatment of BED. Self-help formats seem to be

effective (abstinence varied between 50% and 87%). However, some caution is needed with the interpretation of the high abstinence rates, because the participants in these 'self-help' studies were probably less severely ill than those in the other studies (for example, subjects in the study by Carter (1998) and others had not received any prior treatment).

Also a few double-blind placebo-controlled pharmacological trials have been conducted in patients with BED. Drugs which have been examined are selective serotonine reuptake inhibitors (SSRI´s; fluoxetine, fluvoxamine, sertraline)(Greeno et al., 1996; Arnold et al., 2002; Hudson et al., 1998; McElroy et al., 2000), appetite suppressants (d-fenfluramine (Stunkard et al., 1996) and sibutramine (Appolinario et al., 2003) and an anticonvulsant (topiramate)(McElroy et al., 2003). Fluoxetine seemed to reduce dietary intake but did not affect the frequency of binge episodes. This finding suggests that fluoxetine affects satiety, not hunger (Greeno et al., 1996). In another study (Arnold et al., 2002) fluoxetine reduced binge frequency significantly compared to placebo. Fluvoxamine was found to be effective in reducing the frequency of binge episodes and in lowering Clinical Global Impression (CGI) severity scores (Hudson et al., 1998). Sertraline seemed to be effective and well tolerated, although the number of participants in that study was low (McElroy et al., 2000). D-Fenfluramine reduced the frequency of binge eating in obese women with BED, but failed to reduce their bodyweight (Stunkard et al., 1996). A significant reduction of binge eating and weight was found in the sibutramine group compared to the placebo group (Appolinario et al., 2003). Topiramate was associated with significantly greater reductions in binge frequency compared to placebo after 14 weeks (94% versus 46% repectively).

A striking finding in these pharmacological studies is a high placebo-effect. All studies had a single-blind lead-in period from 1 to 4 weeks. After this lead-in period 42 to 44% of the participants no longer met the DSM-IV-criteria for BED.

In all studies the drugs under investigation seemed to be more effective than placebo with regard to the primary outcome measures. However, no long-term effects were found. Further, drugs did not seem to bring about a reduction in bodyweight. Disadvantages of these studies were the small number of participants and the short duration of the trials.

Cognitive behavioural psychotherapy is currently the most investigated treatment for BED and consequently the treatment of choice for binge eating disorder.

Category or continuum?

The main question treated in this chapter is whether binge eating disorder can be distinguished as a separate mental disorder. In order to define a distinct eating disorder, the disorder must have well described characteristics. Is BED distinct from obesity? Another important question is whether bulimic disorders are dimensional or categorical in nature?

In a few cross-sectional studies (Fichter et al., 1993; Kirkley et al., 1992; Howard et al., 1999) patients with BED were compared to matched samples of patients with BN purging

type and of patients with obesity (BMI > 30). The scores of patients with BED had an intermediate position between BN and obesity but were closer to BN than to obesity. In a series of cross-sectional studies (van Hanswijck de Jonge, 2002) patients with binge eating disorder were compared to patients with bulimia nervosa and non-binge eating obesity on eating disorder psychopathology, general psychopathology, personality pathology (categorical and dimensional). The study revealed a dichotomy between binge eaters (bulimia nervosa and binge eating disorder) and non-binge eaters (obesity) rather than a continuum of severity between the groups on all measures of psychopathology.

Williamson and others (1992) identified three relatively homogeneous subgroups of subjects who had been diagnosed with EDNOS using two cluster analytic procedures. The three atypical subgroups were contrasted with two groups of subjects with anorexia nervosa and bulimia nervosa. These groups were very similar to the descriptions of sub threshold anorexia nervosa, non-purging BN and BED. Subjects in the 'BED' group were morbidly obese but did not report extreme motivation for thinness. They reported significant problems with binge eating, including significant concern about loss of control over eating. These subjects did not resort to extreme weight control methods such as purging or extremely restrictive eating. Estimations of current body size were closer to norms and ideal body weight preferences were larger than those predicted from norms. Members of the group also were less biased in their assessment of actual and ideal weight than those of the other clinical groups. They expressed a more realistic dissatisfaction with obesity.

In another study Williamson and others (2002) found further empirical support for conceptualizing BN and BED as discrete syndromes. Three factors were found to account for 66% of the variance in eating disorder symptoms: binge eating, fear of fatness/compensatory behaviours and drive for extreme thinness. The bulimia nervosa group scored high on the features of binge eating and fear of fatness/compensatory behaviours but not on drive for thinness. The binge eating disorder group scored high on binge eating but not on the other two features. Furthermore, persons with a diagnosis of an eating disorder appeared to differ (at least partly) from persons with no pathological eating behaviours in kind rather than simply in degree.

Hay and others (1996) investigated the presence of clinically meaningful subgroups among subjects with recurrent binge eating recruited from the general population. They identified four subgroups by means of a cluster analysis. The results supported the concept of bulimia nervosa and its division into purging and non-purging subtypes. The study failed to provide evidence to support the construct "BED". A possible explanation is that the population under investigation was too young (16-35 years). Patients with BED seem to present themselves for therapy in a later stage, when they are in their thirties and forties, whereas subjects with the other eating disorders seek help when they are generally much younger. The same population was re-analyzed by classifying the subjects according the DSM-IV (Hay et al., 1998). A number of subjects were excluded from the analysis because they did

not meet the DSM-IV criteria for any eating disorder. It was found that subjects with BN purging type did not differ from those with BN non-purging type and the latter did not differ from those with BED. There was a significant difference between subjects with BN purging type and BED.

There seems to be some evidence that subjects who binge without purging are different from subjects who binge and purge. Much research has focused on binge eating as the core psychopathological feature of bulimic disorders. Some state that compensatory behaviour ought to be the focal clinical feature of bulimia nervosa rather than binge eating (Tobin et al., 1997). Few studies (O´Kearney, Gertler, Conti, & Duff, 1998; Garfinkel et al., 1996; Walters et al., 1993; McCann, Rossiter, King, & Agras, 1991) have investigated the differences between BN purging type and BN non-purging type. The overall evidence is that there is a difference between these two subtypes. So far, no study has found evidence for the division of bulimic eating disorders into the three distinct DSM categories such as BN purging type, BN non-purging type and BED.

Others assume that bulimic disorders differ in degree rather than in kind. There is some support for the notion that bulimic eating disorders exist on a continuum of clinical severity, which starts with BN purging type (most severe), passes through BN non-purging type (intermediate severity), and finishes with BED (least severe).

Discussion

The aim of this chapter has been to evaluate the empirical status of binge eating disorder as defined by the DSM-IV. Ten years ago Spitzer and others (1991) reported that there was no classification taxonomy in the DSM for the many individuals who engage in binge eating but do not engage in inappropriate compensatory behaviours. The present discussion considers Pincus' arguments for and against the inclusion of a new diagnostic category in the DSM following a decade of research in the field of binge eating disorder.

Firstly, Pincus argued that rare diagnostic categories might add unnecessary complexity to the already cumbersome system of classification. However, epidemiological studies have shown that 1 to 3% (Hay, 1998; Spitzer et al., 1993; Spitzer et al., 1992) of the general population has binge eating episodes but does not engage in inappropriate behaviours. The prevalence is higher in obese populations (1.3 - 70%). Furthermore, BED seems to be more prevalent as the degree of obesity increases (Spitzer et al., 1992; Spitzer et al., 1993; Ramacciotti et al., 2000; Basdevant et al., 1995; Ricca et al., 2000; Varnado et al., 1997). Therefore, BED does not rank as a rare diagnostic disorder.

Secondly, Pincus argued that new diagnoses are generally proposed by experts and are subsequently used by less expert assessors who may identify more false positives. Clinical practice has indeed shown difficulties can arise in attempting to differentiate between binge eating and emotional overeating in obese patients; such difficulties can lead to high false positive diagnoses. A clear operationalisation of the criteria of a binge eating episode is needed.

Thirdly, Pincus argued that 'adding unproven diagnostic categories may confer upon such categories an approval that they do not merit yet'. Many studies have indicated that BED does represent a distinct diagnostic entity. The characteristics of subjects with BED differ significantly from those of subjects with bulimia nervosa and from those of obese subjects without binge eating. Furthermore, taxonomic studies have shown that there is a distinct category of BED, which differs from other clinical eating disorder categories. In day-to-day clinical practice BED is a generally accepted category and various eating disorder clinics have developed programmes for the treatment of BED.

Pincus' fourth and last argument concerned the definitional overlap across related categories. To date it has been difficult to distinguish BED from non-purging BN. Various studies have shown that there is a significant difference between the BN purging subtype and BED. Non-purging BN seems to occupy an intermediate position between these two categories, not differing significantly from either of them (Hay et al., 1998). It is not yet clear whether non-purging BN bears a closer resemblance to purging BN (supporting continued classification under BN) or a closer resemblance to BED (supporting a merger of BED and non-purging BN)(Striegel-Moore et al., 2000). Research is needed to clarify this issue.

Although there is evidence to suggest that BED represents a distinct eating disorder category, the criteria as currently described would benefit from some major revision. Criterion A as currently defined should be maintained. Binge eating episodes should be characterized by the consumption of a large amount of food within a discrete period with a sense of lack of control over eating. Criterion B seems to be superfluous. Criterion B measures binge eating characteristics, which overlaps with criterion A (binge eating characteristics) and criterion C (feelings of distress regarding binge eating). It is not clear what is meant by the term distress (criterion C) as currently described. Distress may refer to an emotional state with regard to binge eating or it may describe impairment in social or occupational functioning due to binge eating. I suggest that both types of distress should be operationalised in the revised version of criterion C. The DSM guidelines state that impairment in functioning is crucial in the definition of any mental disorder.

For a diagnosis of binge eating disorder, binge eating is required to occur on two days a week (criterion D) rather than in the form of two episodes per week. This is based on the rationale that binge eating disorder patients may have more difficulties in recalling and labelling binge eating episodes due to the absence of purging behaviours punctuating the termination of an episode. However, counting the number of days allows binges to last an entire day (in theory). In a population characterized by a high percentage of compulsive overeaters (without loss of control), this may complicate the separation of the diagnoses of binge eating episodes and compulsive overeating. Therefore, I suggest counting the number of binge eating episodes rather than counting the number of days.

I suggest eliminating the term 'regular' as mentioned in criterion E. The elimination of this term ensures clearer boundaries between bulimia nervosa and binge eating disorder.

Existing studies support the need for cognitive criteria in addition to the existing behavioural diagnostic criteria for binge eating disorder. Hitherto, various existing studies have argued for the inclusion of overconcern with body weight and body shape in self-evaluation (Eldredge et al., 1996; Wilson et al., 1993; Wilfley et al., 2000b).

Furthermore, future research needs to clarify the impact of obesity on the psychopathology of BED and vice versa. Although obesity is not a criterion for the diagnosis of BED, the classification for BED was created with the obese in mind (Spitzer et al., 1991). Future research will show whether obesity should be admitted as a criterion for BED in the same way as underweight was included as a criterion for anorexia nervosa.

In summary, I believe that BED represents a distinct eating disorder category and suggest that it be admitted into the next version of the DSM.

Chapter 4

Maladaptive core beliefs and eating disorder symptoms

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Summary

This study compared maladaptive core beliefs of eating-disordered groups (full and sub-threshold syndrome) and healthy controls and investigated the association between eating disorder symptoms and core beliefs. Participants were compared on self-report measures of core beliefs (YSQ) and eating disorder psychopathology (BITE). Anorexia nervosa (AN; both subtypes) and bulimia nervosa (BN) patients had significantly more core beliefs than healthy controls. Binge eating disorder (BED) patients had intermediate scores between AN and BN on the one hand and healthy controls on the other hand. No correlation was found between core beliefs and frequency of binge eating. Frequency of vomiting, laxative misuse and fasting was positively associated with all domains of core beliefs. Patients with eating disorders have some core beliefs, which are not directly related to eating, weight or shape. Frequency of purging and fasting behaviours is associated with more severe maladaptive core beliefs. Our data demonstrate the importance of identifying purging and fasting as significant clinical markers.

Introduction

According to cognitive-behavioural models, people with eating disorders judge themselves largely in terms of their eating habits, shape or weight (and often all three) and their ability to control these (Fairburn, Cooper, & Shafran, 2003). While cognitive behavioural models focus on maladaptive thinking patterns that are specific to eating disorders (negative automatic thoughts and dysfunctional assumptions), several studies have demonstrated that such patients also have dysfunctional cognitions or maladaptive core beliefs that are not directly related to food, weight or shape (Cooper, 1997; Leung et al., 1999; Waller et al., 2000; Waller, 2003; Waller et al., 2003). Such maladaptive core beliefs are considered in the schema-focused model devised by Young (1999). Young 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. They refer to stable and enduring themes that develop during childhood. During childhood a belief is a means for the child to comprehend and manage the environment. Core beliefs are a priori truths that are implicit and taken for granted and are central to the organization of personality. In adulthood, these core beliefs are usually activated by events in the environment relevant to the particular belief (Schmidt, Joiner, Young, & Telch, 1995). For example the abandonment belief is activated during real or perceived separations. Core beliefs can be assessed with the Young Schema Questionnaire (Schmidt et al., 1995; Young, 1999).

Several studies found that patients with an eating disorder have more maladaptive core beliefs than healthy controls. The clinical eating-disordered subgroups, however, did not differ much in the extent of maladaptive core beliefs. Only a few differences were found between Anorexia Nervosa restrictive subtype (ANR), Anorexia Nervosa binge/purge subtype (ANB/P), Bulimia Nervosa (BN) and Binge Eating Disorder (BED). Waller et al. (2000) showed that the three bulimic groups could be differentiated by three core beliefs, namely 'defectiveness/shame', 'insufficient self-control' and 'failure-to-achieve'. Another study (Leung et al., 1999) which also assessed patients with ANR, but no patients with BED, found that subjects with BN showed a higher level of entitlement beliefs (beliefs that people should be able to do, say or have whatever they want immediately regardless of whether it hurts others or seems reasonable to them) than subjects with ANR. Finally, differences were found between BED and BN patients (Waller, 2003). Patients with BED had higher scores on the YSQ on three subscales (failure-to-achieve, dependence/incompetence and entitlement) than patients with BN.

At the symptomatic level Waller et al. (2000) found that the frequency of bingeing seems to be positively associated with the core belief 'emotional inhibition' and the frequency of vomiting with the belief 'defectiveness/shame'. They hypothesized that vomiting results from a need to escape from intolerable cognitions especially from beliefs of being internally flawed and inadequate which often leads to shame (defectiveness/shame), whereas bingeing reduces the experience of intolerable emotions (emotional inhibition). When such emotions

arise bingeing serves as a mechanism to reduce the experience of these emotions. Leung et al. (1999) did not find a predictor for binging and compensatory behaviours independent of the kind of eating disorder, but showed that in BN the frequency of bingeing was negatively associated with the core belief social undesirability which is the belief that one is unattractive to and disliked by others, and that in ANB/P the frequency of vomiting was positively correlated with failure-to-achieve which is the belief that one is incapable of performing as well as one's peers. Finally, high levels of abandonment beliefs might account for the presence of purging behaviours i.e. vomiting and laxatives misuse (Waller, 2003).

The first aim of this study is to enlarge upon the previous studies. This study is the first to compare directly the level of maladaptive core beliefs in four eating disordered groups (ANR, ANB/P, BNP and BED) and a healthy control group.

The second aim is to investigate whether specific core beliefs are associated with eating disorder psychopathology. 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 this study all patients with an eating disorder according to the DSM-IV were included so that differences and similarities between the separate eating disorders could be studied in a comprehensive way. Thus, besides including patients with anorexia nervosa (AN) and bulimia nervosa (BN) we also included patients with an eating disorder not otherwise specified or EDNOS. Patients with EDNOS have an eating disorder of clinical severity and meet all but one criteria of AN and BN (Fairburn et al., 2003; American Psychiatric Association, 1994). If the range of severity of eating disorder symptoms is expanded by the addition of EDNOS it should be possible to obtain a better understanding of the associations with core beliefs.

Method

Participants and Procedure

The research sample consisted of 106 patients (100 women and 6 men[1]) with a DSM-IV eating disorder and 27 healthy female controls.

All eligible patients who met criteria for an eating disorder according to the DSM-IV were sent the Young Schema Questionnaire (YSQ) (Young, 1999) and the Bulimic Investigatory Test Edinburgh (BITE) (Henderson & Freeman, 1987) and were asked in an accompanying letter to participate in the study.

As stated above, the study encompassed all patients with a DSM-IV eating disorder, including patients with EDNOS. EDNOS patients represent the largest diagnostic category in

[1] No differences in demographic characteristics, eating disorder symptoms and core beliefs were found between men and women. Men and women were also not different with regard to the association of severity of symptoms with core beliefs.

eating disorders; furthermore, patients with EDNOS closely resemble patients with AN and BN (Fairburn & Harrison, 2003). We hypothesized that patients with the same symptoms had the same underlying core beliefs.

To investigate the first question of this study all 106 patients were subdivided into four DSM-IV categories. For example, we combined patients who met all four criteria of AN, with patients who lacked one criteria of AN, thus for example patients with a BMI between 17.5 and 20 or patients with only a mild fear of getting fat (see table 1 for all EDNOS criteria). Combining full-syndrome and their EDNOS equivalent led to the following group sizes: ANR-extended (*N*= 16; ANR (full): *N*=7 and EDNOS ANR: *N*=9); ANB/P-extended (*N*=31; ANBP (full): *N*=12 and EDNOS ANBP: *N*=19); BNP-extended (*N*= 23; BN (full): *N*=16 and EDNOS BN: *N*=7) and BED (*N*= 36).

Table 1: EDNOS criteria

EDNOS ANR EDNOS ANBP	3 out of 4 AN criteria and: or or or	BMI between 17.5 and 20 (irregular) menstruation mild disturbance of body image mild fear of fatness
EDNOS BN	2 out of 3 BN criteria and: or or	binge eating inadequate compensatory behaviours once a week mild concern with body weight and shape
BED	See DSM-IV appendix B	

The healthy control group consisted of 27 subjects not suffering from eating disorders who were screened to exclude any present or past history of an eating disorder. Subjects were excluded from the control group if they had a score above 10 on the symptom scale of the BITE or a score above 5 on the severity scale of the BITE (Bulimic Investigatory Test Edinburgh)(Henderson et al., 1987).

Measures

At the first assessment all patients participated in a standardized semi-structured clinical interview conducted by a psychiatrist or psychologist specialized in eating disorders. Patients were classified according to DSM-IV criteria. Patients who had eating problems but did not fulfill the DSM-IV criteria for an eating disorder (AN, BN or EDNOS) were excluded from the study.

Each participant completed a Dutch version of the Young Schema Questionnaire (YSQ) (Young, 1999) and the Bulimic Investigatory Test Edinburgh (BITE) (Henderson et al., 1987).

The YSQ is a 205-item self-report questionnaire developed to measure 16 core beliefs or early maladaptive schemas. The items are answered on a 6-point Likert scale ranging from 1 ('totally inapplicable to me') to 6 ('describes me perfectly'). The core beliefs are grouped in four domains or higher order factors according to Lee et al. (1999a). In their study Lee et al. undertook a higher-order factor analysis to investigate the psychometric properties of the YSQ in a clinical sample (N=433). Fourteen of the 16 core beliefs hypothesized by Young (1999) emerged as independent factors. The only core belief that did not emerge was 'Social Undesirability (the belief that one is unattractive to and disliked by others). The other core belief that did not emerge as a separate independent factor was 'Emotional Inhibition (the belief that one must inhibit emotions and impulses because any expression of feelings would harm others or lead to loss of self-esteem, embarrassment, retaliation or abandonment)'. This core belief was found to consist of two independent factors, 'Emotional Inhibition' and 'Fear of Loss of Control'. Of the original 205 items 21 items were deleted because they failed to load above 0.40 on any scale. In our study we used the division into core beliefs and domains according to Lee et al. (1999). The four higher order factors (a to d) and the sixteen core beliefs (1 to 16) are:

a) Disconnection (the sense of being unlikable or unlovable. Individuals who score high on this factor appear to expect that their emotional needs will not be met, believe that there is something wrong with them and hence try to hide their true self and/or to mistrust others). This domain includes the following core beliefs:

1. Abandonment/instability (the belief that close relationships will end imminently).
2. Defectiveness/shame (the belief that one is internally flawed and inadequate, which often leads to shame).
3. Emotional deprivation (the belief that one's primary emotional needs will never be met by others).
4. Mistrust/misuse (belief that others will intentionally take advantage in some way).
5. Social isolation (the belief that one is isolated from the world, different from other people and/or not part of any community).
6. Emotional constriction (the belief that one must inhibit emotions and impulses because any expression of feelings would harm others or lead to loss of self-esteem, embarrassment, retaliation or abandonment).

b) Impaired autonomy (the expectation about oneself and the environment that interferes with one's perceived ability to separate, survive, function independently or perform successfully. It includes the sense of perceiving oneself as a failure, having to depend on or to defer to others, of being vulnerable, and of being unable to have sufficient self-control or self-discipline to perform effectively). This factor includes the following core beliefs.

7. Dependence/incompetence (the belief that one is not capable of handling day-to-day responsibilities competently and independently).

8. Vulnerability to harm and illness (the belief that one is always on the verge of experiencing a major catastrophe).
9. Enmeshment (the lack of individual identity due to emotional over-involvement with others).
10. Failure to achieve (the belief that one is incapable of performing as well as one's peers).
11. Subjugation (the belief that one must submit to the control of others in order to avoid negative consequences),
12. Insufficient self-control/self-discipline (the belief that one cannot control one's impulses or feelings).

c) Impaired limits (Deficiency in internal limits, responsibility to others or long-term goal-orientation; leads to difficulty in respecting the rights of others, cooperating with others, making commitments or setting and meeting realistic personal goals. People with high scores on these scales have difficulty in respecting the rights of others, making commitments, setting and meeting personal goals and tolerating unpleasant emotional experiences.). This factor includes the following core beliefs.

13. Entitlement/grandiosity (the belief that people should be able to do, say or have whatever they want immediately regardless of whether it hurts others or seems reasonable to them)
14. Fear of loss of control (the belief that one must inhibit emotions and impulses; the reason for this inhibition is a fear of loss of control)

The third core belief that loaded on this factor, also loaded on the factor 'Impaired autonomy', namely, insufficient self-control/self-discipline (the belief that one cannot control one's impulses or feelings).

d) Over control (represents an emphasis on over-controlling one's feelings and choices, an emphasis on performance, duty, perfectionism and following rules). The factor includes the following core beliefs.

15. Self-sacrifice (the belief that one has to sacrifice one's own needs in order to help others)
16. Unrelenting standards / hypercriticalness (the belief that one should strive for unrealistic standards).

Item mean scores are calculated for each scale. A higher score on a scale indicates a more dysfunctional level of that core belief. In a study by Waller et al. (2000) the YSQ was found to be valid and reliable for eating disorders.

The BITE (Henderson et al., 1987) is a 33-item self-report questionnaire developed to assess the presence and severity of bulimic symptoms. We used the BITE to obtain a frequency index for the eating disorder symptoms in the patient groups. Frequency of vomiting, fasting and misuse of laxatives was measured on a seven-point scale (never, occasionally, once a month, once a week, two or three times a week, once a day, two or three

times a day and five times a day or more). Frequency of bingeing was measured on a six-point scale (never, once a month, once a week, two or three times a week, daily and two or three times daily). Frequency of fasting was measured on a five-point scale (once, occasionally, once a week, two or three times a week, every second day). The BITE has a symptom scale and a severity scale. These scales were used primarily to assess the healthy control group. A higher score on the symptom scale reflects a more disordered eating pattern. A symptom score below 10 falls within normal limits. A higher score on the severity scale reflects a higher severity of bingeing and purging as defined by their frequency. A high score on the severity scale alone may confirm the occurrence of psychogenic vomiting or laxative misuse in the absence of binge eating. A score below 5 on this scale is considered to be clinically not significant. The BITE has been found to be both valid and reliable (Henderson et al., 1987). In addition, the BITE seems to be effective in assessing levels of eating disorder symptomatology within nonclinical samples (Meyer, Leung, Feary, & Mann, 2001).

Statistical analysis

ANOVA tests and post-hoc Tukey's HSD tests were used to compare demographical variables and the YSQ scores of the four eating-disordered groups (ANR, ANB/P, BN and BED) and the healthy control group. In the case of nominal and ordinal variables Kruskall-Wallis and post-hoc Mann-Whitney *U* tests were used.

To determine the transdiagnostic dimensional associations, the core beliefs (in ANR, ANBP, BN and BED combined) were correlated (Pearson's *r*) with the severity of eating disorder symptoms (binging, vomiting, fasting and misuse of laxatives) (BITE). The healthy controls were excluded from this analysis.

Results

Characteristics of the sample

A significant difference was found between the mean BMI (ANOVA $F(4, 133) = 70.3$; $p < 0.01$) and age (ANOVA; $F(4, 133) = 3.20$, $p = 0.015$) of the groups. Post-hoc tests revealed that the restricted and binge/purge anorexics had a lower mean BMI (M=15.5, S.D.=2.16 and M= 15.8, S.D. = 2.16, respectively) than the bulimics and the normal controls (M= 25.6, S.D. = 7.43 and M=23.4, S.D.=4.08, respectively). The subjects with BED had the highest BMI, which differed significantly from all other groups (M= 38.9; S.D.= 9.67). The mean ages for ANR, ANB/P, BN, BED and healthy controls were 26.5 years (S.D.= 8.7), 28.9 years (S.D. = 8.9), 30.3 years (S.D.= 9.4), 35.0 (S.D.= 10.3) and 33.9 years (S.D. = 11.1) respectively. Post-hoc analysis revealed that the only significant difference was between patients with BED and patients with ANR. The former were significantly older than the latter.

To provide information on the clinical severity of the sample age of onset and duration of illness were investigated. No differences were found between the (eating disorder) groups regarding age of onset (M=18.2, S.D. =7.3; ANOVA, $F(3, 105) = 0.065$; p=0.978). A

significant overall difference was found between the groups regarding duration of illness (ANOVA, $F(3, 105) = 5{,}1$; $p < 0.01$). Post hoc tests revealed that for patients with BED (M=17.2, S.D.= 11.0) the duration of illness was twice as long as for patients with ANR (M= 7.8, S.D. = 5.6) or ANBP (M=10.7, S.D.=8.4).

A Kruskall-Wallis test revealed no differences between the frequency of bingeing of patients with ANB/P, BN and BED ($X^2 = 2.82$, df=2, p= 0.24). By definition patients with ANR and healthy controls do not binge. No differences were found between the frequency of vomiting and misuse of laxatives in patients with ANB/P and BN (Mann-Whitney U, df=1, p=0.65, and $p = 0.96$ respectively). By definition, patients with ANR, BED and healthy controls do not vomit and misuse laxatives. No differences were found in the frequency of fasting in patients with ANR, ANB/P and BN ($X^2 = 11.4$, $df = 3$, $p = 0.33$). By definition BED patients do not fast.

Patterns of core beliefs

Table 2 shows a significant overall difference on all four YSQ higher factors (ANOVA; $p< 0.01$) between the four eating-disordered groups and the healthy control group. Post-hoc Tukey HSD tests showed that the patients with an eating disorder had significantly more pathological core beliefs about themselves than the healthy controls on all four factors. Post-hoc Tukey tests showed significant differences between some eating-disordered subgroups (see table 2 for details). Overall, patients with BED had significantly fewer maladaptive core beliefs than patients with ANB/P.

Table 2 : Young Schema Questionnaire domains (according Lee et al. 1999) of patients with anorexia nervosa, restricting type (ANR), anorexia nervosa, binge-purge type (ANBP), bulimia nervosa (BN), binge eating disorder (BED) and control group (C).

	Group					Post-hoc	
	ANR Mean (SD) N=16	ANBP Mean (SD) N=31	BN Mean (SD) N=23	BED Mean (SD) N=36	Control group Mean (SD) N=27	ANOVA F (df)	Tukey HSD
Disconnection	3,14 (1,23)	3,59 (0,80)	3,07 (0,91)	2,49 (0,87)	1,43 (0,37)	26,7* (4, 127)	ED-groups > C; ANBP > BED
Impaired Autonomy	3,01 (1,06)	3,45 (0,84)	3,10 (0,87)	2,48 (0,78)	1,53 (0,34)	24,5* (4, 128)	ED-groups >C; ANBP > BED
Impaired Limits	3,07 (1,02)	3,40 (0,73)	3,23 (0,86)	2,73 (0,81)	1,66 (0,37)	22,2* (4, 128)	ED-groups > C; ANBP > BED
Overcontrol	3,52 (0,94)	3,90 (0,90)	3,70 (0,79)	2,95 (0,86)	2,07 (0,46)	22,8* (4, 127)	ED-groups > C; ANBP = BN > BED

* $p < 0.001$

Correlations between specific eating disorder psychopathology and core beliefs

The healthy controls were excluded from this analysis. In the following analysis BMI and age are used as covariates given the significant differences that were found between AN patients and BED patients. Another reason to use BMI as a covariate was a significant negative correlation that has been found between BMI and maladaptive core beliefs. A low BMI is associated with more severe maladaptive core beliefs (disconnection: $r = -0.30$; $p<0.001$); impaired autonomy $r = -0.29$; $p<0.01$; over-control $r = -0.31$; $p<0.01$). Other studies found some evidence that a low BMI induces cognitive dysfunctioning (Lena, Fiocco, & Leyenaar, 2004).

The results of the correlation analyses (Pearson's *r*) were as follows. No significant correlations were found between the frequency of binge eating (all eating disorders) and any of the four factors.

Significant correlations were found between frequency of vomiting, laxatives misuse and fasting and all four factors (see table 3). A higher frequency of compensatory behaviours is associated with more severe maladaptive core beliefs.

Table 3: Associations (Pearson's *r*) in ANR, ANBP, BN and BED combined between YSQ higher order factors (according to Lee et al., (1999a) and frequency of binge eating, vomiting, laxative abuse and fasting controlling for BMI and age.

	Binge eating *r* (CI)	Vomiting *r* (CI)	Laxative abuse *r* (CI)	Fasting *r* (CI)
Disconnection	0,04 (CI: -0.16 – 0.24)	0,24* (CI: 0.04 – 0.42)	0,36** (CI: 0.22 – 0.56)	0,30** (CI: 0.18 – 0.53)
Impaired autonomy	0,17 (CI: -0.17 – 0.23)	0,26* (CI: 0.14 – 0.50)	0,36** (CI: 0.22 – 0.56)	0,28** (CI: 0.17 – 0.52)
Impaired limits	0,14 (CI: -0.06 – 0.33)	0,27* (CI: 0.12 – 0.48)	0,32** (CI: 0.15 – 0.51)	0,27** (CI: 0.10 – 0.47)
Overcontrol	0,08 (CI: -0.12 – 0.28)	0,19 (CI: 0.05 – 0.43)	0,34** (CI: 0.18 – 0.53)	0,25** (CI: 0.16 – 0.50)

** $p < 0.01$* $p < 0.05$

Discussion

The study compared core beliefs in patients with ANR, ANB/P, BN and BED and in a group of healthy controls. In this way differences and similarities between all types of eating disorders could be investigated directly. Patients with an eating disorder (AN (both subtypes), BN and BED) had significantly unhealthier core beliefs than healthy controls on all the four factors of maladaptive core beliefs. Patients with AN (both subtypes) and BN did not differ in the degree of unhealthy core beliefs. With regard to the nature and severity of the core beliefs,

patients with BED had an intermediate score between AN and BN on the one hand and the healthy controls on the other hand. However, the core beliefs of BED patients seem to be more similar to the core beliefs of BN or AN patients than to those of healthy subjects. This confirms the results of previous studies (Waller et al., 2000; Leung et al., 1999; Dingemans et al., 2002).

Our results show that the frequency of inappropriate compensatory behaviours like vomiting, laxative misuse and fasting, are positively associated with all four higher order factors of maladaptive core beliefs unrelated to BMI and age. Patients who engage (more frequently) in compensatory behaviours have more severe unhealthy beliefs about themselves and their environment than patients who do not. Frequency of binge eating does not seem to be associated with severity of maladaptive core beliefs. This suggests that engaging in inappropriate compensatory behaviours is linked to more disturbed thinking patterns. This might also explain the lower scores of BED patients on the Young Schema Questionnaire (YSQ), since BED patients do not engage in inappropriate compensatory behaviours, by definition. Beumont et al. (1995) found that patients who purged were more disturbed on all psychopathological characteristics measured by the Eating Disorder Examination (EDE)(Cooper et al., 1987) than were those who did not purge. Also, O'Kearney et al. (1998) found that purgers were more disturbed than nonpurgers on measures of specific eating disorder psychopathology, for example sense of ineffectiveness, bulimic tendencies drive for thinness and body dissatisfaction, which was unrelated to weight, level of anxiety or general distress. Our data showed that cognitions or core beliefs that are not related to eating disorder psychopathology are distorted in patients with an eating disorder and even more in patients who engage in purging behaviours and fasting. These data demonstrate the importance of identifying purging and fasting as significant clinical markers. The frequency of these behaviours can be regarded as an indicator of the severity of the illness (Keel et al., 2004; Tobin, Johnson, & Dennis, 1992).

According to cognitive behavioural models, an over-evaluation of eating, shape and weight is the 'core psychopathology' of eating disorders (Fairburn et al., 2003). This leads to strict dieting which leads to binge eating. Purging behaviours are seen as a way to minimize weight gain (Fairburn et al., 2003; Jansen, Vandenhout, & Griez, 1990; Heatherton, Herman, & Polivy, 1992; Heatherton et al., 1991). However, our results suggest that purging and fasting behaviours in eating disorders might also serve another role besides that of simply minimizing weight gain caused by binge eating. This result concurs with the results of several other studies on the function of purging. In a small study Jeppson et al. (2003) found that purging behaviours also contributed to a marked sense of empowerment or control or an expression of opposition or rebellion. Furthermore, some patients reported that purging was a punishment or self-defeating behaviour. Also, most patients described the feeling of well-being (i.e. relief, relaxation, numbness) immediately after completing the purge.

In this study we also found that BMI is negatively associated with maladaptive core beliefs. A lower BMI is associated with a higher number of maladaptive core beliefs. However, in this cross-sectional study we cannot say anything about the direction of the causal relation between these two variables. It cannot be ruled out that a low BMI results in more disturbed core beliefs, because previous studies produced some evidence that a low BMI induces cognitive dysfunctioning (Lena et al., 2004; Polivy, 1996; Jones, Duncan, Brouwers, & Mirsky, 1991)

One of the strengths of our study is the inclusion of patients with a DSM-IV EDNOS diagnosis. By adding EDNOS patients we extended the range of clinical severity to include subthreshold cases. On a behavioural level this allowed us to examine the relationship between core beliefs and eating disorder psychopathology in an extended dimensional fashion. Another strength is the grouping of the sixteen core beliefs into four higher order factors as proposed by Lee et al. (1999). In this way we reduced type I errors and minimized chance findings. The variety in the results of studies investigating core beliefs in eating disorders suggests that we should use these higher order domains or the overall mean score of the YSQ as a general indication of core beliefs and /or dysfunctional thinking style which leads to unhealthy life patterns. The sixteen core beliefs and also the four higher-order factors are highly correlated. Because of the small number of subjects used in other studies that investigated core beliefs in eating disorder, correlations might be found by chance.

The study has some limitations. First of all, the group sizes were not equal and were small. Secondly, we did not assess co-morbid axis I disorders (such as mood or anxiety disorders). Thirdly, the study is based on cross-sectional data only. This implies that no statements can be made about the causal relationships between core beliefs and eating disorder psychopathology. Finally, large differences were found in the duration of illness especially between patients with AN and BED. However, no differences were found in the age of onset in the four diagnostic groups, which means that eating disorders in general tend to develop during adolescence. An explanation for the large differences in the duration of illness might be that BED, when compared to AN and BN, is a less severe eating disorder (Dingemans et al. 2002). Persons with more severe disorders are more likely to perceive a need for treatment (Sareen, Hassard, Menec, Stein, & Campbell, 2005). Patients with AN and BN will therefore seek help sooner than patients with BED.

Longitudinal studies are needed in order to investigate whether core beliefs constitute a vulnerability factor for the development of eating disorder pathology or can be better conceptualized as a concomitant or consequence of having an eating disorder. This study showed that there are some indications that the degree of inappropriate compensatory behaviours is linked to severity of domains of core beliefs. Future studies should be done in larger samples in which the association between the separate core beliefs can investigated and should address comorbid disorders and symptoms.

Chapter 5

Predictors and mediators of treatment outcome in patients with Binge Eating Disorder

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Summary
A randomized controlled trial (*N*=52) was conducted comparing cognitive behavioural therapy with a waiting list control group to identify mediators and predictors of treatment outcome. Reduction of weight concerns mediated abstinence of binge eating at post-treatment. Abstinence was marginally mediated by changes in eating and shape concerns, depressive symptoms and global severity of general psychopathology. Neither treatment outcome nor status at 1-year follow-up could be predicted by severity of eating disorder, comorbid psychopathology or maladaptive core beliefs at baseline or at post-treatment. The only predictor for abstinence at both post-treatment and 1-year follow-up was the coping style palliative reacting: Higher scores predicted less favourable outcomes. Lower expression of emotions at post-treatment predicted more reduction of eating disorder psychopathology at follow-up. No other patient characteristics allowing treatment-patient matching could be identified.

Introduction

Several controlled clinical trials indicate that psychological treatment leads to a significant reduction in binge eating in patients with Binge Eating Disorder (BED) compared to a waiting list control group. Reductions in the number of binge episodes after treatment reported in these studies ranged from 68 to 90% and rates of abstinence post treatment range from 40 to 87%. Reductions in the number of binge episodes in a waiting list control group reported in the studies above ranged from 8 to 22% and abstinence rates range from 0% to 19% (Dingemans et al., 2002).

In all of these controlled clinical trials, the frequency of binge eating and/or abstinence of binge eating wer the primary outcome measures, though most studies also assessed comorbid symptoms or disorders such as depressive symptoms or depressive disorder by means of self-report questionnaires or structured interviews as well. However, the previously mentioned controlled clinical trials primarily looked at differences in pre- and post-treatment scores on the primary and secondary outcome measures and barely addressed these variables as possible predictors or mediators of outcome. Treatment predictors specify for whom the treatment works. Mediators on the other hand define how or why effects occur (Baron & Kenny, 1986). They identify possible mechanisms by which a treatment might achieve its effect. These mechanisms are causal links between treatment and outcome (Kraemer, Agras, Wilson, & Fairburn, 2002).

To our knowledge, only two studies have investigated possible treatment predictors in patients with BED. Wilfley et al. (2000a) examined the association of baseline comorbid axis I and II disorders with eating disorder severity at baseline, at the end of treatment, and at 1-year follow-up. Axis I psychopathology was not associated with binge eating or global eating disorder severity, whether at baseline, post treatment or 1-year follow-up. Contrary to their expectations, comorbid axis I disorders did not predict treatment outcome. However, axis II psychopathology was associated with higher levels of binge eating and overall eating pathology at baseline. The presence of cluster B personality disorders predicted significantly higher levels of binge eating at 1-year follow-up. Safer et al. (2002) found that predictors for relapse were early age of binge eating onset and greater overall dietary restraint at post treatment. To our knowledge, mediators of change in the treatment for BED have never been studied.

The aims of the present randomized controlled trial were to explore the effectiveness and to identify possible predictors and mediators of CBT for patients with BED. First, we attempted to replicate findings in previous randomized controlled trials and investigated the effectiveness of CBT versus a waiting list control group (WLC) in patients with BED. We were not only interested in the effects of treatment on reducing the frequency of binge eating but also in treatment influence on other eating disorder psychopathology, as well as comorbid psychopathology (such as general psychopathology and depressive symptoms), maladaptive core beliefs (as a measure for personality psychopathology), ineffective coping styles and body weight loss. We expected that CBT results would be superior to the WLC.

Our second aim was to investigate whether changes in coping styles, general eating disorder psychopathology, comorbid psychopathology and body weight during treatment mediate treatment outcome. During CBT patients learn to identify and correct dysfunctional cognitions (with respect to eating, weight and shape) and to avoid behaviours associated with binge eating and to replace these behaviours with healthier coping styles. Since negative affect and binge eating seem to be related (Grilo et al., 1994) we were also interested whether changes in negative affect mediate treatment outcome.

Our third aim was to investigate whether comorbid 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. We expected that patients with more severe eating and more general psychopathology, more severe maladaptive core beliefs and more ineffective coping styles would benefit less from treatment and would be less able to maintain their treatment gains during the follow-up period.

Methods

Participants and recruitment

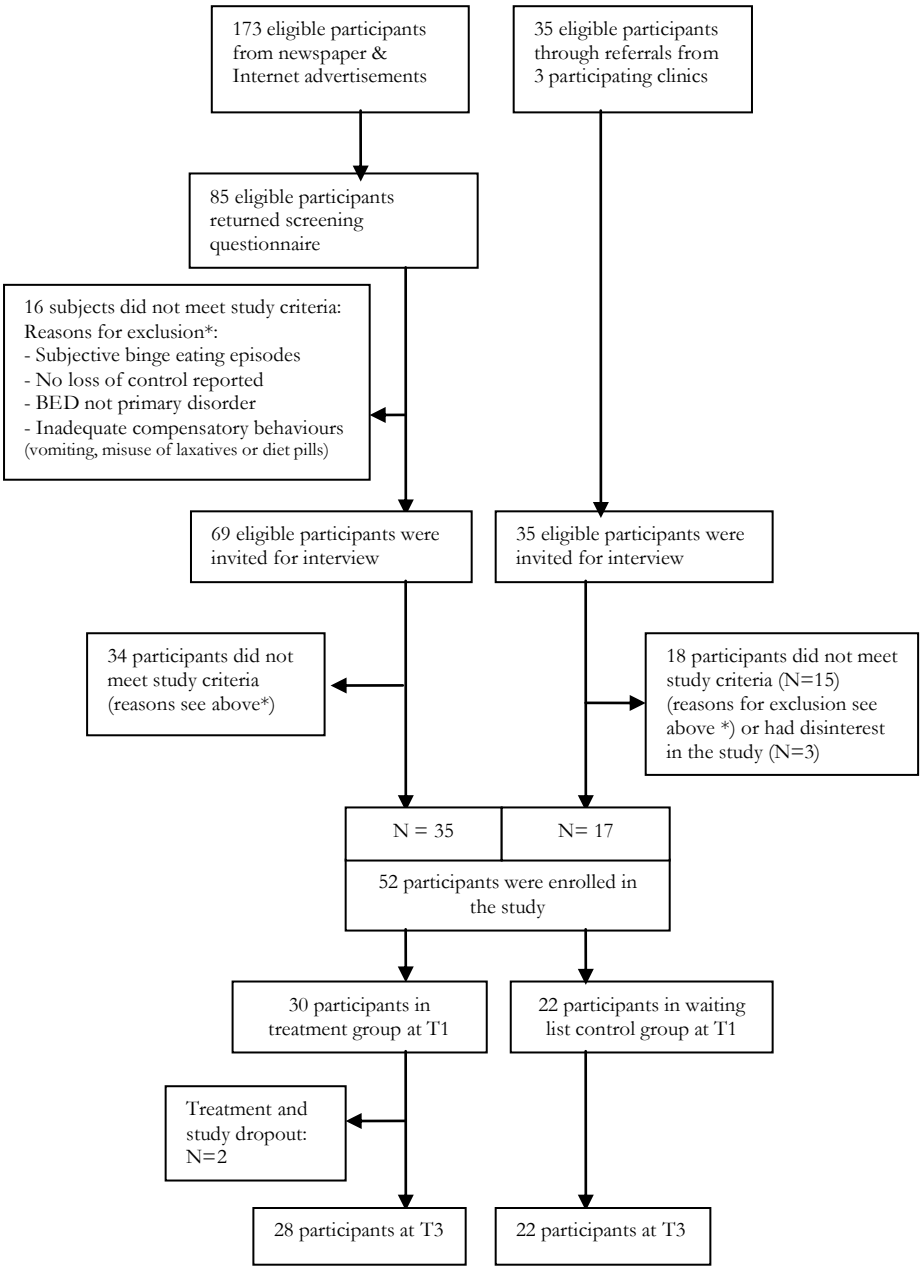
The patients included in the study had a primary diagnosis of binge eating disorder (BED) according to the DSM-IV (American Psychiatric Association, 1994). Comorbid psychiatric disorders were accepted. Patients were not admitted to the study if one of the following exclusion criteria was met: a) a current history of self-induced vomiting, misuse of laxatives, diuretics, enemas, diet pills or other weight controlling medications, fasting, or excessive exercise within the last 24 weeks, b) concurrent psychological or weight loss treatment, c) a comorbid diagnosis of psychotic disorder, self-damaging behaviours or mental deficiency, or d) pregnancy. We did not restrict our sample to those of any particular body weight.

Participants were recruited from three eating disorder centers, by advertisements in a local newspaper and via four Internet websites (see Figure 1). In total, 52 individuals (49 women and 3 men) appeared suitable and were enrolled in the study. The Medical Ethics Committee for Mental Health Institutions approved the study.

Design and procedure

The study was designed as a randomized controlled treatment trial with four assessments (baseline (T1), after ten weeks (mid-point, T2) by mail only, the end of treatment (T3) and 1 year after the end of treatment (T4)). The assessors had no therapeutic relationship with any of the participants. The assessors were blind to the group assignment.

Figure 1: Flow chart of patients in the study



Participants were recruited in four equal phases. A neutral person performed random allocation to the CBT or WLC GROUP blindly after the first assessment. After the third assessment (T3), participants in the waiting list control condition were all offered CBT.

Measures

The study used a number of assessment instruments to measure the primary and secondary outcome variables.

Eating psychopathology

The Dutch Eating Disorder Examination (Jansen, 2000) is an investigator-based, semi-structured interview format for the assessment of eating disorder specific psychopathologies. It provides a comprehensive profile of individual psychopathology based on scores from four subscales: restraint, eating concern, shape concern and weight concern. A global scale of eating pathology (mean of the four subscales) was also computed to assess overall eating psychopathology. Items are rated on 7-point forced-choice scales (0-6), with higher scores reflecting greater severity or frequency. Additionally, the EDE assesses frequency of overeating and use of extreme methods of weight control. The EDE provides frequency ratings for their occurrence in the past 28 days. The Eating Disorder Examination Questionnaire (EDE-Q) is a self-report version of the EDE interview and generates the same data and subscales (Fairburn et al., 1993a).

General Psychopathology

The Structured Clinical Interview for DSM IV axis I disorders (SCID-I) (First, Spitzer, Gibbon, & Williams, 1997; van Groenestijn, Akkerhuis, Kupka, Schneider, & Nolen, 1999) is a semi-structured interview for diagnosing the major DSM-IV axis I disorders (American Psychiatric Association, 1994). It is divided into six relatively self-contained modules: Mood episodes, psychotic symptoms, psychotic disorders, mood disorders, substance use disorders, anxiety and other disorders.

The Dutch Symptom Checklist-90 (SCL-90)(Arrindell & Ettema, 1986) presents respondents with a series of 90 physical and psychological complaints and asks them to rate each for the degree of distress associated with these complaints on a 5-point scale ranging from 0 (not at all) to 4 (extremely). The standard time frame that respondents use to rate the symptoms is the preceding 7 days. The Global Severity Index (GSI) is the average rating on all 90 items.

Depression

The Dutch version of the Beck Depression Inventory-II (BDI-II-NL) (Van der Does, 2002) contains 21items, each with four self-evaluative statements rated on severity. The BDI-

II-NL measures severity of depressive symptomatology. The score is a simple sum of values from 0 to 3 responses. The range of possible scores is 0 to 63.

Coping

The Utrecht Coping List (UCL) (Schreurs, van de Willige, Tellegen, & Brosschot, 1993) is a Dutch questionnaire that measures coping behaviour when confronted with problems. The UCL consists of 47 items and of 7 subscales: active tackling; palliative reacting; avoiding, waiting; seeking social support; passive reacting; expression of emotions; reassuring thoughts. Participants are asked to rate their usual reaction to a range of problems or unpleasant incidents on a 4-point scale ranging from 1 (not at all) to 4 (extremely).

Maladaptive core beliefs

The Young Schema Questionnaire (YSQ) is a 205-item self-report questionnaire developed to measure 16 maladaptive core beliefs. Young (1999) hypothesized that maladaptive core beliefs are at the core of personality disorders. These core beliefs are thought to underlie all personality pathology. The items are answered on a 6-point Likert scale ranging from 1 ('totally inapplicable to me') to 6 ('describes me perfectly'). The core beliefs are grouped into four domains, according to Lee et al. (1999a). Item mean scores are calculated for each domain. A higher score indicates a more dysfunctional level of that core belief domain.

1) Disconnection (the sense of being unlikable or unlovable).
2) Impaired autonomy (expectations about oneself and the environment that interfere with one's perceived ability to separate, survive, function independently or perform successfully).
3) Impaired limits (deficiency in internal limits, responsibility to others or long-term goal orientation which lead to difficulty in respecting the rights of others, cooperating with others, making commitments or setting and meeting realistic personal goals).
4) Over-control (an emphasis on over-controlling one's feelings and choices, an emphasis on performance, duty, perfectionism and following rules).

Body mass index (BMI)

Body weight and length were measured in clothes without shoes. Body Mass Index (BMI) was calculated (body weight (kg)/ height $(m)^2$).

Suitability of and confidence in cognitive behavioural therapy (CBT)

Suitability of and confidence in cognitive behavioural therapy was measured on a 100 mm Visual Analogue Scale (VAS). A higher score indicates reflects more suitability and more confidence.

Treatment

The cognitive-behavioural therapy (CBT) was conducted on an outpatient basis. It consisted of 15 group sessions conducted over a 20-week period. The first 10 sessions were weekly and the last five sessions were biweekly. Each session lasted two hours. Two therapists trained in cognitive-behaviour therapy and with ample experience in treating eating disorders conducted all the 15 sessions. A treatment manual from the Neuropsychiatric Instiute Fargo, USA was used.

CBT is semi-structured, problem-oriented, and mainly concerned with the patients' present and future rather than their past. The group CBT for BED consisted of three phases. In the first phase (sessions 1 to 7), the main goal was to develop a regular eating pattern and to resist the urge to binge eat. Patients learned to identify and correct dysfunctional cognitions and avoidance behaviours related to eating. Another goal was to replace these behaviours with healthier, self-enhancing responses. In the second phase (sessions 8 to 13) underlying problems such as body image, self-esteem, stress management, problem solving, assertiveness and weight loss issues were addressed. The third and last phase of the treatment (sessions 14 and 15) was concerned with relapse prevention after the end of treatment. Homework assignments were part of all sessions. Feedback was given on the food diaries and homework assignments. The elements, which were included in this treatment, were also present in the treatment given to participants in the study by Wilfley et al. (2002).

Statistical analysis

Differences in pre-treatment demographics and clinical variables between the two conditions (CBT and WLC) were analyzed by means of independent sample t-test or chi-square tests if appropriate

The analyses were performed on the following outcome measures: abstinence of binge eating, global eating disorder psychopathology (EDE), four subscales of the EDE, depressive symptoms (BDI), general psychopathology (SCL-90), four domains of core beliefs (YSQ), seven coping styles (UCL) and BMI.

Multilevel analysis (MLA) was used to analyze the development of each outcome measure over time. MLA is especially suitable to analyze repeated measure data because it takes into account the dependencies among observations nested within individuals. Another advantage to the methodology is its ability to handle missing data. Random coefficient models were fitted for all outcome measures, allowing for individual variation of intercepts and regression slopes. Fixed effects of Time and the interaction between Time and Condition were tested using two-tailed z-tests. We corrected for outcome variable at baseline in each analysis (Busing, Meijer, & Van der Leeden, 2005)

For the mediation analyses, the three-step process as described by Baron and Kenny was used (1986). All analyses were performed with linear or logistic regression. In the first step the criterion (outcome as measured by abstinence and global eating disorder psychopathology)

is regressed on the predictor (treatment). In the second step, the mediator (changes in coping styles, depressive symptoms, eating disorder psychopathology and general psychopathology) is regressed on the predictor (treatment). In the third and last step, the criterion (outcome) is regressed on both the predictor (treatment) and the mediator.

For the prediction analysis, all treated groups, including the participants from the former waiting list control group, were combined (n=50). We used regression analyses to calculate residualized change scores by statistically correcting post treatment scores (T3) for any between-participants differences on the dependent variable at baseline (T1). The same was done for the follow-up period (T3-T4). Subsequently, to determine whether there is an association between potential predictors and outcome (the residualized change scores) Pearson r correlations or t-tests were performed, when appropriate.

We corrected for multiple comparisons by using an alpha < 0.01 in all analyses.

Results

Participants: randomization and dropouts

The participants in the two conditions (CBT and WLC) did not differ in demographical or clinical variables or in any of the dependent measures at baseline except on current Axis I anxiety disorder (see Tables 1 and 2). Participants who attended ten sessions or less (i.e. less than 67% of the sessions) were considered treatment dropouts (N=2).

Table 1: Demographical variables at baseline

		CBT (N=30)	*WLC (N=22)*	*Test statistics and significance*
Age (years), mean ± S.D.		38.8 ± 10.4	36.4 ± 11.3	$t(50)= 0.79$, $p= 0.43$, ns
Axis I disorders (SCID)				
Any axis I disorder, current		7 (23%)	9 (40%)	$\chi^2(1)= 1.84$, $p= 0.18$, ns
Any axis I disorder, lifetime		21 (70%)	16 (72%)	$\chi^2(1)= 0.05$, $p= 0.83$, ns
Mood disorders, current		5 (17%)	3 (14%)	$\chi^2(1)= 0.09$, $p= 0.54$, ns
Anxiety disorders, current		2 (7%)	7 (32%)	$\chi^2(1)= 5.6$, $p= 0.027$
Some previous treatment eating disorders (Including psycho-education, dietician, self-help groups)		11 (37%)	7 (32%)	$\chi^2(1)= 0.13$, $p= 0.78$, ns
Marital status	Never married	8	9	$\chi^2(3)= 3.14$, p=0.37 ns
	Married/living together	16	12	
	Divorced	5	1	
	Widow	1	0	
Socio-economic status	Fulltime job/education	13		$\chi^2(3)= 1.73$, p=0.63, ns
	Part-time job	7	9	
	Unemployed/homemaker	2	5	
	Sick leave/disabled	6	5	
			3	
Education	High	9	11	$\chi^2(2)= 1.58$, p=0.45, ns
	Intermediate	9	4	
	Low	12	8	

Table 2: MLA Effects for Time and Time X Conditions for differences on outcome measures between CBT and WLC Group (*p<0.01 **p<0.001)

	Pre-treatment (T1)				Mid-treatment (T2)				Post-treatment (T3)				Test statistics and significance	
	CBT		WLC		CBT		WLC		CBT		WLC		Time	Time x Condition
	mean	S.D.	mean	S.D.	mean	S.D.	mean	S.D.	mean	S.D.	mean	S.D.	B (SE)	B (SE)
Subjective binge episodes/28 days	7.0	16.7	8.8	11.6	2.3	3.7	3.9	8.0	2.3	5.4	7.9	13.3	3.07 (1.50)	-2.40 (1.18)
Objective overeating/ 28 days	6.6	9.9	9.6	12.4	1.3	3.5	1.2	3.4	2.1	5.5	4.6	6.0	2.57 (0.93)*	-0.88 (0.71)
EDE Global	2.4	0.9	2.3	0.8	2.3	1.2	3.1	1.1	1.3	1.0	2.3	0.9	-0.58 (0.16)**	-0.60 (0.12)**
EDE Restraint	1.7	1.1	1.7	1.2	1.5	1.3	2.1	1.5	0.9	1.0	1.9	1.3	-0.21 (0.21)	-0.52 (0.17)*
EDE Eating conc.	2.0	1.2	1.8	1.2	1.6	1.1	2.6	1.5	0.9	1.1	1.6	1.1	-0.52 (0.23)	-0.56 (0.17)**
EDE Weight conc.	3.4	1.4	3.1	1.3	2.9	1.6	3.8	1.1	1.9	1.4	3.2	1.2	-0.47 (0.24)	-0.79 (0.17)**
EDE Shape conc.	2.5	1.0	2.8	1.0	3.0	1.6	3.8	1.3	1.6	1.0	2.6	1.2	-1.17 (0.23)**	-0.47 (0.15)*
Global Severity Score (SCL90)	169.3	48.0	167.2	45.6	152.1	39.7	166.8	52.0	143.6	49.0	170.0	57.7	5.08 (5.56)	-16.09 (4.85)**
Depression (BDI)	20.7	13.1	17.7	9.8	14.6	10.4	18.2	11.9	12.9	13.2	17.4	10.5	0.78 (1.41)	-3.72 (1.32)*
Coping styles (UCL)														
- Active tackling	17.5	3.5	16.5	3.5	18.1	4.4	16.8	2.9	17.7	3.9	16.3	3.3	-0.63 (0.51)	0.32 (0.41)
- Palliative reacting	19.0	2.9	19.3	3.0	18.4	3.0	19.6	3.0	18.8	3.3	18.6	2.7	-0.10 (0.53)	-0.08 (0.38)
- Avoiding, waiting	17.5	3.8	17.2	3.5	16.2	3.2	17.5	3.6	16.4	3.4	17.1	3.2	0.32 (0.46)	-0.74 (0.34)
- Seeking social support	12.1	4.3	12.6	3.3	13.1	4.1	13.5	3.9	13.7	4.4	12.7	3.6	-0.37 (0.53)	0.68 (0.43)
- Passive reacting	14.0	3.5	13.5	2.7	13.4	3.2	13.9	3.8	12.0	3.6	13.6	3.4	-0.35 (0.50)	-1.06 (0.44)*
- Expression of emotions	6.4	1.5	6.6	1.7	6.4	1.5	6.2	1.4	6.4	1.5	5.6	2.9	-0.49 (0.28)	0.43 (0.26)
- Reassuring thoughts	15.5	2.6	14.3	3.2	15.2	3.0	14.8	3.8	15.1	2.7	14.1	2.5	-0.35 (0.44)	0.03 (0.29)

Effectiveness of treatment

The mean scores on the clinical outcome measures at pre-treatment, halfway and post-treatment in both conditions and the results of the statistical tests can be found in Table 2[2]. As expected, in all cases results for the CBT group were superior to the WLC group. In the CBT group, 63% (n=19) of the participants were abstinent for objective binge eating episodes at the end of treatment, compared to 18% (n=4) in the WLC group (χ^2 (1, N=52)=10.5, p<0.001). The frequency of objective binge eating episodes dropped by 86% in the CBT group and by 11% in the WLC group at the end of treatment compared to pre-treatment. In the CBT group the frequency of objective binge eating in the last 28 days dropped from 14.8 (± 7.8) (T1), to 3.5 (± 5.8) at mid-treatment (T2), to 2.0 (± 5.5) at the end of treatment (T3) (Wilcoxon, z = -4.36, p <0.001). The number of objective binge eating episodes in the WLC

[2] All analyses were corrected for baseline differences between the treatment conditions

group at the three assessments was 14.7 (T1), 10.3 (T2) and 13.1 (T3) (Wilcoxon, χ=0.74, p=0.46, ns).

Mediation analyses

As described by Baron and Kenny (1986), mediation would be suggested if 1) outcome is significantly related to treatment, 2) treatment is significantly related to changes in mediating variables, and 3) the relationship between treatment and outcome decreases (or goes to zero) when change in mediating variables is entered into the equation. As indicated above, primary outcome (abstinence) was significantly related to treatment (step 1; see table 2)) and treatment resulted in significant changes with respect to possible mediating variables such as global eating disorder psychopathology, the BDI, the Global Severity Index of the SCL-90 and the UCL subscale passive reacting. In order to execute step 3, first residualized change scores (baseline to post treatment) were calculated for the global and four subscales of the EDE, BDI, GSI-index of the SCL-90 and the UCL subscale for passive reacting. In the subsequent third step, it was investigated whether these potential mediating variables were still a significant predictor of abstinence and change scores for global eating disorder psychopathology, whereas the association between treatment and abstinence or change scores for global eating disorder psychopathology decreases (to zero) when both mediator and treatment are included in the regression equation. These analyses (see table 3) revealed only one instance of full mediation. Change in global EDE-scores during treatment was a mediator for abstinence of binge eating at the end of treatment. There was no longer a significant main effect for treatment (OR=0.43, 95% CI=0.085 – 2.18, p=0.31), which indicates that abstinence at the end of treatment, was totally mediated by change in eating disorder psychopathology (global EDE scale, OR=0.21, 95% CI=0.08 – 0.58, p=0.003). Further analysis of the four EDE subscales restraint, concerns about eating, weight and shape indicated that abstinence at post treatment was totally mediated by change in concerns about weight. Restraint did not, and concerns about eating and shape did only marginally mediate abstinence. Also, change scores on the BDI and SCL-90 only marginally mediated the effect of treatment. The UCL subscale for passive reacting did not prove to be a significant independent predictor of abstinence over and above the effect of treatment.

The same analyses were repeated with respect to the EDE conceptualized as an outcome measure: No mediators were identified.

Prediction analyses

For these analyses, the CBT group and the treated (former) WLC group were combined (n=50)(see Table 4). As expected, during treatment participants improved on most treatment outcome variables. At the end of treatment 70% (n=35) was abstinent for objective binge eating and at 1-year follow-up, 80% (n=40). The frequency of objective binge eating episodes dropped 90% (T1: M=14.3, S.D.= 10.4 and T3: M=1.5, S.D.= 6.5). These results are

Table 3: Mediation analyses: Logistic regression with abstinence at post-treatment as dependent variable, and condition and changes in global scale and the four subscales of the EDE (restraint, concerns, about eating, weight and shape), BDI and SCL-90 as independent variables

		B	S.E.	Wald	df	*p*	OR	CI 95%
Step 1	condition	-2.051	.670	9.363	1	.002	.129	.035 – .478
Step 2	condition	-.843	0.827	1.037	1	.309	0.431	.085 – 2.180
	residual changes scores EDE global	-1.565	0.524	8.912	1	.003	0.209	.075 – .584
Step 2	condition	-1.854	.718	6.671	1	.010	.157	.038 - .639
	residual changes scores EDE Restraint T1-T3	-.252	.362	.485	1	.486	.777	.382 –1.580
Step 2	condition	-1.555	.740	4.416	1	.036	.211	.05 – .901
	residual changes scores EDE concerns about eating T1-T3	-1.256	.496	6.428	1	.011	.285	.108 – .752
Step 2	condition	-.842	.829	1.030	1	.310	.431	.085 – 2.189
	residual changes scores EDE concerns about weight T1-T3	-1.915	.610	9.862	1	.002	.147	.045 – .487
Step 2	condition	-1.518	.788	3.714	1	.054	.219	.047 – 1.026
	residual changes scores EDE concerns about shape T1-T3	-1.597	.530	9.091	1	.003	.203	.072 – .572
Step 2	condition	-1.683	.708	5.654	1	.017	.186	.046 – .744
	residual changes scores BDI T1-T3	-.814	.386	4.444	1	.035	.443	.208 – .944
Step 2	condition	-1.523	.723	4.437	1	.035	.218	.053 – .900
	residual changes scores SCL-90 T1-T3	-.882	.448	3.874	1	.049	.414	.172- .996

comparable to those of the treated CBT-group in the previous analyses (CBT versus WLC) (see Table 2). Treatment success was maintained during the 1-year follow-up. Post hoc tests found no significant changes in any outcome variables during follow-up (see Table 4). Only the UCL subscale palliative reacting predicted improvement in eating disorder psychopathology during treatment. More palliative reacting (seeking distraction and trying to feel more comfortable by smoking, drinking or by trying to relax) at baseline predicted a less favourable treatment outcome at the end of treatment for eating disorder psychopathology (Pearson r=0.36, p= 0.01). No other significant predictors were found. Moreover, no predictors were found for abstinence of binge eating at the end of treatment.

During the 1-year follow-up period, participants with high scores on expression of emotions (UCL) at the end of treatment (T3) improved less on eating disorder

psychopathology than participants with lower scores on this subscale (Pearson r=0.50, p<0.001). Logistic regression analyses revealed one significant predictor for abstinence for binge eating at one-year follow-up. Patients who still binged at 1-year follow-up had higher scores on the subscale UCL palliative reacting at post treatment (T3) (OR= 1.35, 95% CI= 1.06 – 1.72, p=0.01).

Body weight loss

During treatment no significant changes were found in BMI (F(1,49)=2.83, p=0.1). However, from baseline to one-year follow-up the total group did have a significant drop in mean BMI from 38.9 (S.D.=7.9) to 37.7 (S.D.=8.0) (F(2,50)= 4.4, p=0.02). Binge eating abstinence was significantly related to percentage body weight loss (F(1,49)=5.7, p=0.02). However, abstinence did not mediate body weight loss or vice versa. Thirty –eight percent (n=19) lost more than 5% of their initial body weight.

Table 4: MLA effects for Time on outcome measures for all CBT-treated participants (** p< 0.001 *p<0.01)

	Baseline		Halfway		Post-treatment		One-year follow-up		
	T1		T2		T3		T4		Tests and significance
	M	S.D.	*M*	S.D.	*M*	S.D.	*M*	S.D.	Time effects, β (SE)
Objective binge eating episodes /28 days	14.3	10.4	4.1	6.5	1.5	4.4	2.6	7.1	0.008 (0.001)**
Subjective binge eating episodes/28 days	7.6	15.5	2.8	4.6	3.3	8.9	3.4	6.8	0.005 (0.001)**
Objective overeating/ 28 days	5.7	8.7	0.9	3.2	2.2	5.7	2.0	4.9	0.007 (0.001)**
Eating Disorder Psychopathology- EDE									
Global	2.4	0.8	2.2	1.1	1.3	0.9	1.3	1.0	0.007 (0.003)*
Restraint	1.8	1.1	1.6	1.3	0.9	0.9	1.0	1.1	-0.01 (0.007) ns
Eating Concerns	1.9	1.1	1.4	1.1	0.8	1.1	0.7	1.0	0.02 (0.006)**
Weight Concerns	3.3	1.3	2.8	1.5	1.8	1.3	2.0	1.5	0.02 (0.007) ns
Shape Concerns	2.6	1.1	2.9	1.4	1.6	1.0	1.6	1.2	-0.03 (0.007)**
General psychopathology									
Global Severity Score- SCL90	171	51.8	150	42.0	138	43.1	141	54.0	0.02 (0.006) **
Depression- BDI	19.6	12.2	13.3	9.6	10.3	11.5	10.8	12.7	0.005 (0.001)**
Coping styles- UCL									
Active tackling	16.8	3.4	17.6	4.2	17.1	3.6	17.6	4.1	-0.0006 (0.003) ns
Palliative reacting	18.9	2.8	18.4	3.1	18.2	3.0	17.4	3.0	0.002 (0.005) ns
Avoiding, waiting	17.4	3.6	15.9	3.1	15.9	3.3	16.0	3.8	0.10 (0.02)**
Seeking social support	12.4	4.0	13.4	3.9	14.4	5.7	13.8	3.4	-0.023 (0.02) ns
Passive reacting	14.0	3.4	13.2	3.2	11.9	3.2	12.1	3.4	0.020 (0.016) ns
Expression of emotions	6.4	1.3	6.3	1.5	6.2	1.4	6.3	1.7	0.006 (0.01) ns
Reassuring thoughts	14.9	2.6	15.2	2.9	14.7	3.0	14.9	2.6	0.03 (0.009)**

Discussion

In the present randomized controlled trial, the effectiveness and possible mediators and predictors of cognitive-behaviour therapy (CBT) in patients with Binge Eating Disorders (BED) were examined. As expected, objective binge eating frequency, eating disordered psychopathology and comorbid psychopathology decreased significantly in the CBT group whereas no reduction was found in the control (WLC) group. Mediator analyses indicated that abstinence of binge eating at post treatment was totally mediated by changes in weight concerns and marginally by changes in concerns about shape and eating, depressive symptoms and more general psychopathological symptoms. However, a note of caution is needed. Overall a rapid response to treatment can be seen, as evidenced by the substantial reduction in frequency of objective binge eating episodes during the first half of treatment. This rapid treatment response precludes the use of the approach of Kraemer et al. (2002) to study mediation of outcome in our study, because this approach requires that changes in putative mediators temporarily precede substantial changes on the outcome measures used. By using the approach of Baron and Kenny (1986) we were only able to identify correlates of outcome. Consequently, our data do not allow causal interpretations of the associations between clinical improvement and changes on putative mediators as observed in our study.

Two other studies, both using the Eating Disorder Examination Questionnaire (EDE-Q), also found evidence that concerns about weight might be a maintaining factor in BED and lead to binge eating (Cachelin et al., 1999; Pratt, Telch, Labouvie, Wilson, & Agras, 2001). Weight concerns, as well as the other marginal mediators for abstinence of binge eating are in some way related to negative affect and negative self-evaluation (Pratt et al., 2001). Binging may be a way to escape from this negative affect or negative self-evaluation (Heatherton et al., 1991). Experimental studies confirm the link between negative affect and binge eating (Agras et al., 1998; Chua et al., 2004). In treatment, patients have to learn to break the vicious spiral of dysfunctional thoughts and concerns to subsequently abstain from binge eating.

Contrary to our expectations, patient with BED benefited equally from treatment independent of the severity of their comorbid symptoms. Thus CBT might be an effective treatment for the majority of patients. However, we did find that participants with higher levels of the coping style palliative reacting (trying to feel better by eating, smoking, or drinking) at baseline and end of treatment benefited less from treatment than participants with less palliative reacting. Similar indications were found by Bloks et al. (2001) in patients with bulimia nervosa, who are similar, in many aspects, to patients with BED (Dingemans et al., 2002). High levels of expression of anger and annoyance at end of treatment were associated with less improvement then would be expected during the 1-year follow-up. As observed in previous studies patients who reacted less angry and annoyed in their emotions were better capable of maintaining treatment success (Penas-Lledo, Fernandez, & Waller, 2004; Fassino, Leombruni,

Piero, Abbate-Daga, & Rovera, 2003). Therapists should be aware of these emotions because it might be an indication for relapse.

Another notable finding is that changes in restraint did not mediate abstinence of binge eating. This result is consistent with the growing body of evidence that the association between dieting/restraint and binge eating as a factor maintaining BED does not apply to a substantial number of individuals with BED (de Zwaan, 2005b). Not restraint but negative emotional disturbances and coping deficits seem to increase the likelihood of binge eating (Grilo et al., 1994). In an experimental study Agras and Telch (1998) found that negative mood, and not caloric deprivation, significantly increased loss of control over eating, perhaps explaining the mechanism underlying the triggering of binge eating by a negative mood. During the 1-year follow-up period, the BMI of the participants decreased significantly. Our results, and those of other studies (Wilfley et al., 2002a; Agras et al., 1997a) suggest that participants who are able to stop binge eating tend to lose body weight. At follow-up, almost 40% of the participants lost 5% or more of their initial body weight, which has been associated with significant health benefits (The National Heart, 1998).

The results of the study should be considered in conjunction with a few limitations. First, the number of participants is relatively small. As a result, potential mediators or predictors could have been missed. Second, we did not take session–by-session measures of the possible mediators and outcome measures. More frequent assessments would have allowed us to perform a mediation analysis along the lines of Kraemer et al. (2002). Third, we did not assess axis II personality disorders. However, the Young Schema Questionnaire gives some indication of personality pathology. Finally, although we attempted to keep assessors blind to group assignment, this was not possible in all cases.

One of strength of this study is that we used very few exclusion criteria. Our conclusions are based on a representative sample of patients with BED. Furthermore, this is the first study, which investigated possible mediators of changes of CBT for patients with BED. Also, this is one of the few studies, which investigated possible predictors of treatment outcome in BED.

Future research on BED should focus on identifying mechanisms of treatment action and the order in which changes occur. Session-by-session assessment of behavioural and cognitive measures might shed light on maintaining factors of binge eating. This may help both to focus the substance and to improve the (cost) effectiveness of treatment.

Chapter 6

The effect of suppressing negative emotions on eating behaviour in Binge Eating Disorder

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Summary

Overeating may be a consequence of the suppression of negative emotions, by depleting self-control resources. This experiment investigated whether (a) there is a causal relationship between the suppression of negative emotions, negative mood, and overeating in people with binge eating disorder (BED) and whether (b) this relationship is increased in depressed people with BED. Sixty-six women with (full and sub-threshold) BED were shown an upsetting movie and then asked either to suppress their emotions or to react naturally. Subsequently, everyone participated in a taste task. After a decline, initial mood before watching the movie was restored after eating. Depressive symptomatology was positively correlated with caloric intake. Within the clinically depressed (Beck Depression Inventory-score >19) BED group, those who were most affected by the negative mood induction consumed the most calories. No differences were found between the two conditions with regard to caloric intake. No interaction effect was found between depressive symptoms and mood suppression. The hypothesis that suppression of negative emotion leads to overeating in (depressed) binge eaters was not born out. Overeating may serve as a means to (temporary) repair negative mood.

Introduction

The most prevalent comorbid lifetime diagnosis for individuals with binge eating disorder (BED) is depressive disorder (Dingemans, Spinhoven, & Van Furth, 2007a). Moreover, ample evidence exists that the severity of binge eating is positively related to more severe degrees of depression (Telch et al., 1994a). Apart from the findings that BED individuals are relatively more depressed than non-BED individuals, mood states seem especially poor immediately before a binge eating episode (Grilo et al., 1994). Binge eater mood states prior to binge eating episodes have also been shown to be more negative than prior to eating events triggered by hunger (Waters et al., 2001). Furthermore, binge eating-disordered women experience the hassles of daily life as more stressful than non-BED controls and the greater the stress experienced, the higher the food intake during a binge (Crowther, Shepherd, Sanftner, & Bonifazi, 2001). In sum, several studies provide evidence that depressive symptoms (trait), acute negative mood (state), and binge eating behaviour are related but do not explain the causal relationship between negative affect and binge eating in patients with BED.

Only a few studies have attempted to unravel the causal relationship between depression and binge eating. Chua et al. (2004) experimentally manipulated the mood of obese persons with binge eating episodes and found that participants ate more after watching a sad film (negative mood induction) than after watching a neutral film. However, in a similar mood induction experiment that compared BED-diagnosed participants with a weight-matched non-eating-disordered control group, Telch and Agras (1996b) observed that while BED participants consumed significantly more calories at a multi-item buffet than control participants, food intake quantity was not affected by mood. However, acute negative mood did influence how BED participants perceived and labeled their eating episode. After a sad movie, BED participants were more likely to label their eating episode as excessive and to report more loss of control over their eating than control participants. In a later experiment the researchers (Agras et al., 1998) replicated their finding that acute negative mood significantly increased the perception of loss of control over eating. Agras and Telch (1998) argued that a negative mood state lessens the sense of control over eating and therefore makes the labeling of an eating episode as a binge more likely. No differences were found in stable depressive symptoms between those who binge ate at the buffet and those who did not. Agras and Telch suggested that acute negative affect rather than a stable negative mood leads to binge eating.

Thus to date, findings on the causal relationship between depressive symptoms, acute negative mood, and excessive eating are inconclusive. 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 et al., 2001a).

In a previous randomized controlled trial in which the effectiveness of cognitive behavioural therapy in patients with BED was investigated (Dingemans et al., 2007a), we found that less worrying about weight fully mediated abstinence of binge eating at post

treatment and less worrying about shape and eating, depressive symptoms, and more general psychopathological symptoms marginally mediated abstinence. In other words, worrying was positively correlated with binge eating. It is likely that worrying and ruminating about one's weight and appearance are unpleasant experiences. 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 et al., 1991).

To control the urge to binge eat or to regulate negative emotions, people need to exert self-control. Self-control involves regulating or inhibiting competing urges, behaviours, and desires. Muraven and Baumeister (2000) proposed 'that people have a limited quantity of resources available for self-control and that various acts of self-control draw on this limited stock' (p. 247). Automatic processes are efficient whereas controlled ones are costly in terms of effortful consumption of resources (Muraven et al., 2000). Controlling one's behaviour requires the expenditure and depletion of a limited inner resource (Baumeister, Tice, Bratslavsky, & Muraven, 1998). When a situation demands two consecutive acts of self-control, performance on the second (unrelated) act is frequently impaired because of energy depletion. After depletion, the resources need to be restored by means of rest and relaxation. In a study by Muraven et al. (1998), for example, participants shown an upsetting movie either received instruction to regulate their emotions (by either exaggerating or suppressing their emotional response) or received no emotion regulation instructions. Afterwards the participants had to squeeze a handgrip as long as possible, an exercise that is almost entirely a measure of self-control. Participants who had to actively regulate their emotions (exaggeration or suppression) performed worse on the subsequent handgrip-squeezing task than participants who received no such instructions. In other words, active emotion regulation undermined persistence on a next self-control task because of a lack of energy.

In a similar study, Vohs and Heatherton (2001) instructed chronic female dieters either to regulate their emotional reactions by suppressing them while watching an upsetting movie or to show their natural reactions. Directly after, participants took part in an ice cream tasting task. The participants who were instructed to suppress their emotional reactions ate significantly more ice cream during the taste task than the participants who were asked to act naturally. Together these results suggest that a first exercise of self-control impairs people's performance on a subsequent, unrelated self-control task. Important to note is that in the experiments of Muraven and colleagues (1998) and of Vohs and Heatherton (2001) , there were no differences in the extent to which participants' mood was affected by the upsetting movie fragments. As intended, all participants reported a more negative mood after watching the movie, regardless of the instructions received to suppress or exaggerate their emotions or to act naturally. Thus, participants who did or did not regulate their emotions differed only with respect to the amount of self-control exerted while watching the upsetting movie and not with respect to the extent they were emotionally affected by the movie. A well-known researcher in the field of thought and emotion suppression and who studied this in a

laboratory setting, is Daniel Wegner (Wegner et al., 1987; Wegner, 1994; Wenzlaff & Wegner, 2000). His group investigated and reported on what they called the ironic effects of mental control such as thought or emotion suppression (i.e. the famous white bear studies). 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. Because people engage in a suppression-induced search for target-thoughts, suppression leads to a counterproductive result in the form of intrusions of the thought one wanted to ban. Thought suppression seems to have the opposite effect and makes people fixate on it. Ample evidence exists that this is especially true for affective states (Wegner et al., 1994; Martijn, Tenbult, Merckelbach, Dreezens, & de Vries, 2002). The suppression of emotionally loaded stimuli is even more difficult than neutral stimuli. 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. More specifically, studies on emotion regulation and self-control ability showed that it is more likely that the *regulation* of emotional reactions causes loss of control (i.e. overeating) than the mere *experience* of negative emotions.

However, Tice et al. (2001a) have suggested that emotional distress might shift priorities to the immediate present. 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. It could be argued that people with BED choose to binge on high calorie foods because it gives them immediate relief and pleasure, rather than that binge eating is a consequence of energy depletion.

The aim of the present experimental study is to investigate the 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. We 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). Secondly, we hypothesized that the negative mood induction would evoke a specifically increased caloric intake in depressed BED patients. Depressed individuals have lower levels of energy and hence, less resources available for self-control purposes than non-depressed individuals as pointed out by Baumeister and Exline (2000) (p. 35) or an increased caloric intake in depressed BED patients might be caused by a direct affect-regulation as has been proposed by Tice et al. (2001a).

Methods

Participants

The participants included in this study had a primary diagnosis of (sub)threshold binge eating disorder (BED) according to DSM-IV criteria (American Psychiatric Association, 1994). To be included in the study, a participant had to report an average of one binge-eating episode a week over the previous 24 weeks. Women with a sub-threshold BED (an average of one binge eating episode a week) were included in the study because they do not seem to differ significantly from full-syndrome BED (an average of two or more binge eating episodes a week) (Striegel-Moore et al., 2000). Participants had to be female and between 18 and 60 years old. Pregnancy was an exclusion criterion. Subjects could participate in the study if they were stable on medication.

Participants were recruited from patients of the Center of Eating Disorders Ursula (*n*=15, 23%), by advertisements (*n*=51, 77%) in local newspapers, and via Internet websites. The Dutch Medical Ethics Committee for Mental Health Institutions approved the study.

Measures

Demographic variables: Participants' marital status, socioeconomic status (homemaker/retired, fulltime job/student, part-time job, or disabled), educational level (low, medium, high), and age were recorded.

Eating Disorder Examination (EDE): The Eating Disorder Examination (Cooper et al., 1987; Jansen, 2000) is an investigator-based, semi-structured interview format for the assessment of eating disorder specific psychopathologies. The EDE assesses the state of the participant in the previous four weeks. It provides a comprehensive profile of individual psychopathology based on scores on four subscales: restraint (e.g. attempts to avoid certain foods), eating concern (e.g. concern about being seen while eating), shape concern (e.g. importance of body shape in self-evaluation), and weight concern (e.g. dissatisfaction with body weight). A global scale of eating pathology (computed as the mean of the participants' scores on the four subscales) is also computed to assess overall eating psychopathology. Items are rated on 7-point forced-choice scales (0-6), with higher scores reflecting greater severity or frequency. The EDE also assesses two key behavioural aspects of eating disorders: overeating and the use of extreme methods of weight control.

The inter-rater reliability was also examined in patients with BED and was excellent for objective bulimic episodes and days (correlations above .98) and very good for the EDE-subscales albeit somewhat variable (correlations range from .65 to .96) (Grilo, Masheb, Lozano-Blanco, & Barry, 2004b).

Beck Depression Inventory-II (BDI-II): The Dutch version of the Beck Depression Inventory-II (Van der Does, 2002) contains 21 items, each with four self-evaluative statements rated on severity (scored from 0 to 3). The BDI-II measures severity of depressive symptoms. The total score is a simple sum of the 21 individual item scores and may range from 0 to 63.

The following guidelines are suggested as BDI cut-off scores in samples of patients diagnosed as having an affective disorder: no or minimal depression is < 13; mild depression is 14 – 19; moderate to severe depression is 20 – 28; and severe depression is 29 – 63. The internal consistency of the Dutch version of the BDI-II is high in a psychiatric outpatient group and a healthy control group: Cronbach's alpha was 0.92 and 0.88 respectively. Test-retest reliability correlation in the same groups was $r = 0.82$.

Mood scale: Current mood state was measured by seven adjectives (sad, tense, tired, cheerful, active, irritated, and gloomy) on a 100 mm Visual Analogue Scale (VAS) on which the end points were labeled 'not at all – extremely'. The adjectives were classified into two categories: 1) mood (sad, tense, cheerful, irritated and gloomy) (Cronbach's alpha = 0.76), 2) fatigue (tired and active) (Cronbach's alpha =0.56). Furthermore, one statement which measured overall affect ('*altogether my mood at the moment is…*") was measured on a 100 mm VAS on which the end points were labeled 'very good - very bad'. A lower score indicated a more negative mood, more fatigue, and more negative affect. The mood scale was based on a mood measurement used by Martijn and colleagues (2002).

Compliance check: To assess compliance with the instructions while watching the film fragment, the participants had to rate three statements on a Visual Analogue Scale which was 100 mm long and word-anchored at each end (very untrue - very true): 'While watching the film fragment, I tried to control my emotions and feelings/ I succeeded in controlling my emotions and feelings/ I gave my emotions free rein'. One overall scale was made for the mean level of suppression (Cronbach's Alpha = 0.78). A higher score indicated more suppression.

Taste task and food intake: During the taste task each participant was asked to take a seat behind the table with four pre-weighted bowls with large quantities of food: chocolate (M = 1025 grams (5483 kcal), SD = 87 grams (465 kcal)), potato chips (M = 220 grams (1177 kcal), SD = 33 grams (177 kcal)), marshmallows (M = 258 grams (833 kcal), SD= 77 grams (249 kcal)), and cake (M = 638 grams (2680 kcal), SD 107 grams (449 kcal)). During the taste test they had to fill in a Taste Test Questionnaire (Guerrieri et al., 2007). Participants had to rate the four kinds of foods on palatability, intentions to eat, taste and smell. They had to fill out the questionnaire to give them a chance to consume the foods. Data were not analyzed except the item that measured how much they liked the food (ranging form 'not at all' (0) to 'very much' (5). The total amount of food left was measured afterwards and total caloric intake was calculated. Two aspects of food intake were rated on separate 100 mm VAS-scales. Loss of control was evaluated by the item "*Did you have control over the amount of food you ate?*" and rated on a scale varying from 'completely not' to 'completely'. Perceived amount of food ("*How much did you eat?*") was scored on a VAS scale varying from 'not a lot' to 'a lot'. Further, participants were asked (yes/no) whether they considered their eating a binge-eating episode ("*Did you feel as though you experienced a binge eating episode*?").

Procedures

Respondents who reacted to the call for participants in a study on "perception and reactions of persons with binge eating episodes" were first screened by phone or email with regard to the inclusion and exclusion criteria. Potential participants (*N*=111) were sent an information letter and a screening questionnaire. Forty-five individuals did not participate in the study because of a lack of interest (*n*=38) or not meeting inclusion criteria (*n*=7). In total, 66 individuals participated in the study and were invited to the Center for Eating Disorders "Ursula". All participants signed an informed consent and received a monetary reward.

Participants were instructed not to eat 2 hours prior to the assessment and were tested individually. At their arrival they stated that they had followed instructions and had not eaten in the two hours prior to the testing. Because subjects were randomly assigned to one of the two conditions we assumed that differences in levels of hunger are equally distributed across the two conditions. They were randomly allocated to one of the two conditions: an emotion suppression condition (*n*=33) or a "natural reaction' condition (*n*=33). Before the start of the experiment, each participant was interviewed by means of the Eating Disorder Examination (EDE) (Cooper et al., 1987; Jansen, 2000) and filled out the Beck Depression Inventory-II (BDI-II) (Van der Does, 2002). After completion of the interview and questionnaire, the participant was seated in front of a monitor. Four bowls with food were placed on a separate table masked by a folding screen. All participants stated on the Taste Test Questionnaire that they like at least two kinds of food (score 3 or higher). Time of assessment was equally distributed over the two conditions.

Before the mood induction (T1) started, the participant filled out a mood scale questionnaire (see section 'Measures'). Subsequently she was given instructions about how to regulate her emotions while watching a 3-minute fragment of the movie *The Champ*. Studies by Gross and Levenson (1995) showed that this fragment of the movie elicits sadness. Half of the participants were instructed to suppress their emotions (suppression condition) whereas the other half was instructed to react naturally (natural reaction condition).

The suppression condition received the following instruction: *"In a moment you will see a fragment of a movie. This fragment may evoke emotions. Watch the fragment with attention, let in the emotions you feel and try to suppress and control your emotional reactions. In other words, if someone were present in the room with you, he or she should not see what or how you are feeling. Before the fragment starts the screen will be blank for a minute. Try to clear your mind of all thoughts and emotions. I will leave you alone while you watch the fragment and will return afterwards"*. The natural reaction condition received the following instruction: *"In a moment you will see a fragment of a movie. This fragment may evoke emotions. Watch the fragment with attention and let in the emotions you feel and react to them as you please. Before the fragment starts the screen will be blank for a minute. Try to clear your mind of all thoughts and emotions. I will leave you alone while you watch the fragment and will return afterwards."*

After each participant watched the movie fragment, her mood was measured again (T2). Subsequently she took part in the 15-minute taste task. She was instructed to taste all

four kinds of food (see section measures) and to fill out a taste perception questionnaire. She was told to feel free to eat as much as she liked or thought was necessary to evaluate each food. During the taste task, participants were left alone in order to minimize uncomfortable or negative feelings that might arise if someone watched them eat. After the taste task, the participant filled out the mood scales again (T3), and completed a questionnaire to assess compliance with the instructions and food intake. Participants then completed a funnelled debriefing form (Bargh & Chartrand, 2000), which checked for awareness and suspicion. None of the participants reported any awareness of the true nature of the hypothesised connection between the mood induction, suppression instructions and the taste test. At the end of the experiment the participants were fully debriefed of the real purpose of the study.

Statistical analyses

Possible pre-experimental differences in demographics and clinical variables, differences in levels of negative affect and mood during mood induction, and differences in suppression/showing emotions between the 2 conditions (natural reaction and suppression) were checked by means of independent sample t-tests, AN(C)OVA, or chi-square tests if appropriate. Subsequently, data to test the hypotheses were analyzed by means of linear regression analyses.

Results

Participant characteristics

Participant characteristics are displayed in Table 1. There were no differences between the suppression condition (n=33) and natural reaction condition (n=33) with respect to demographic variables (marital status (χ^2 (df=3) = 1.16, ns); socioeconomic status (χ^2 (df=3) = 0.12, ns); education (χ^2 (df=3) = 2.88, ns); or age (M=39.0, SD= 9.6; t(1,64) = 0.46, ns). Ten participants used anti-depressant medication (five participants in each condition). Participants in the suppression condition reported more depressive symptoms during the previous two weeks (as measured by the BDI-II) than participants in the natural reactions condition. Further, the suppression condition reported a marginally significant worse mood at baseline than the natural reactions condition (see table 1). Time of assessment was equally distributed over the two conditions.

Manipulation check

In order to test our hypotheses we first needed to establish 1) that all participants (independent of condition) became significantly sadder after the mood-induction, in other words they all had to experience negative emotions and 2) that the participants who were instructed to suppress/regulate their emotional reactions did in fact suppress/regulate their emotional reactions (suppression condition) significantly more than participants who received no such instructions (natural reactions condition).

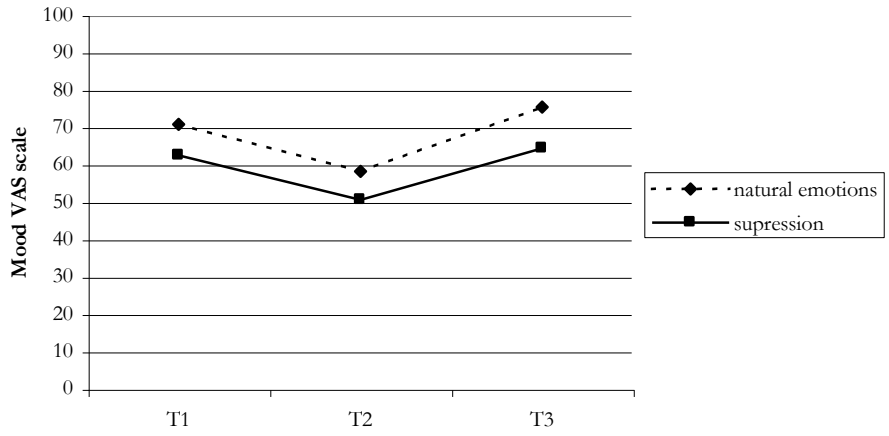
Table 1: Baseline characteristics of natural emotions condition versus suppression condition

	Natural emotions condition (N=33) (M, SD)	Suppression condition (N=33) (M, SD)	Test statistics and significance
Objective binges episodes /28 days	11.5 ± 6.6	10.3 ± 7.6	$t(64) = 0.67$, *ns*
Subjective binge episodes /28 days	5.8 ± 7.3	12.3 ± 10.0	$t(64) = -0.69$, *ns*
EDE Restraint	1.68 ± 0.99	1.66 ± 0.98	$t(64) = 0.07$, *ns*
EDE Concern about eating	1.34 ± 1.07	1.72 ± 1.33	$t(64) = -1.28$, *ns*
EDE Concern about weight	2.80 ± 1.24	3.28 ± 1.31	$t(64) = -1.50$, *ns*
EDE Concern about figure	2.44 ± 1.17	2.78 ± 1.25	$t(64) = -1.16$, *ns*
BMI	32.6 ± 8.18	33.8 ± 6.48	$t(64) = -0.68$, *ns*
BDI (depressive symptoms)	18.1 ± 10.1	24.0 ± 12.4	$t(64) = -2.14$, $p = 0.04$
Mood (VAS) baseline (0-100 mm)	70.7 ± 17.5	62.6 ± 19.0	$t(64) = 1.82$, $p = 0.07$
Fatigue (VAS) baseline (0-100 mm)	51.9 ± 23.8	44.6 ± 23.9	$t(64) = 1.28$, *ns*
Affect (VAS) baseline (0-100 mm)	62.1 ± 22.9	58.7 ± 20.7	$t(64) = 0.64$, *ns*

Negative mood induction: There was a significant difference in depressive symptoms (BDI) prior to the start of the film fragment between the suppression condition and the natural reactions condition (see Table 1). A 2-between x 2-within ANCOVA was conducted with the factors condition (suppression versus natural reactions condition) and time (T1 versus T2) correcting for depressive symptoms (BDI). As expected, there was a significant main effect of time ($F(1,63) = 9.22$, $p<.01$), indicating that mood in both conditions became more negative after watching the upsetting movie fragment ($M_{suppression}$ T1=62.6, SD=19.0; $M_{natural\ reactions}$ T1 = 70.7, SD=17.5; $M_{suppression}$ T2=50.7, SD=22.3 and $M_{natural\ reactions}$ T2= 58.1, SD= 19.7; see also Figure 1). The two conditions did not differ in overall mood change (T1-T2). We found no interaction effect ($F(2,63) < 1$, *n*s).

The results for overall affect and fatigue were similar, that is, a significant time effect ($F(1,64) = 20.5$, $p<.01$) but no significant group ($F(1,64)=1.1$, *ns*) or interaction effect $F(1,64)=0.46$, *ns*) was found for overall affect. A significant time effect ($F(1,64)=4.04$, $p=0.05$) was also found for fatigue, but no significant group ($F(1,64)=2.19$, *ns*) or interaction effect ($F(1,64)<1$, *ns*). Thus we conclude that the film fragment had the intended effect of inducing an equally negative mood state in both conditions.

Figure 1: Changes of mood



Compliance with instructions: We checked whether participants in the suppression condition followed our instructions and tried to control their emotional response towards the film fragment more than participants in the natural reaction condition. On the VAS-suppression scale the participants in the suppression condition had a mean score of 79.5 (SD=13.8) and the natural reactions condition a mean score of 57.1 (SD=27.8). A t-test was conducted with condition (suppression versus natural reactions condition) as the independent variable and degree of suppression as the dependent variable. A significant effect was found between the degree of reported suppression of emotional reactions in the two conditions ($t(63)=4.13$, $p<.001$). As expected, participants in the suppression condition tried to suppress and control their emotions more than participants in the natural reactions condition. The participants had complied with our instructions.

Effects of mood suppression on food intake

In order to test the first hypothesis about the effect of suppression of negative emotional reactions on caloric intake, we conducted a stepwise linear regression analysis with caloric intake as dependent variable and condition (suppression versus natural reactions) as main predictor while controlling for depressive symptoms (BDI-II score), mood at T1 (VAS), and BMI as possible confounders.

Our prediction was not confirmed: participants in the suppression condition (M=455 kcal, SD 312), did not eat more during the taste task than participants in the natural reactions condition (M=461 kcal, SD 206) (β=-37.84, $t(65)=0.59$, *ns*).

The effect of suppression and depression on caloric intake

In order to test our second hypothesis that the level of depressive symptoms (as measured by the BDI-II) would exaggerate caloric intake differences in the two conditions, we added an interaction term (BDI-II x condition) to the equation. It was hypothesized that the negative mood induction would elicit increased calorie intake in depressed BED participants in the suppression condition. No interaction effect of condition and level of depression was found (β=6.67, t(65)=1.17, *ns*). We tested whether mood changes during the taste task had a different effect on caloric intake in the two conditions. No interaction effect was found, after correcting for depressive symptoms (β=17.7, t(1,65)=0.71, *ns*). Overall, mood tended to improve during the taste task (T2-T3) independent of the condition (F (1,64)=34.98, p<0.001)(see Figure 1).

The effect of depression on caloric intake

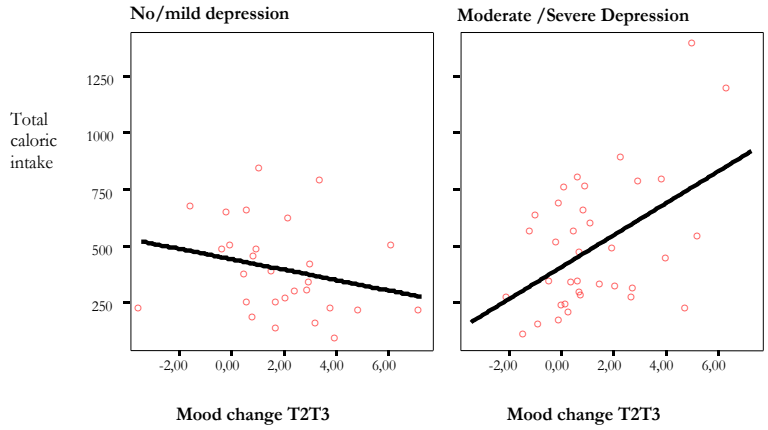
The level of depressive symptoms (BDI-II) predicted caloric intake independent of condition (suppression or natural reactions)(correcting for BMI and mood at T1)(β=9.96, t(1,65)=3.3, p=.002). Because of this previous finding, we tested whether depressed BED participants behaved different than non-depressed BED-participants during the mood induction and the taste task.

We tested whether changes in mood during the mood induction (T1-T2) had an effect on caloric intake. For this analysis, we first calculated individual mood change scores by subtracting the first measurement at the very beginning of the experiment (T1) from the mood measurement after the mood induction (T2). These mood change scores (T1-T2), depressive symptoms (BDI-II), and their interaction were entered into the equation with caloric intake as the dependent variable correcting for condition. There was a significant interaction effect of depressive symptoms and mood change during the film (β=-2.80, t(1,65)=2.17, p=.03). The more depressed BED participants were and the more their mood worsened during the mood induction, the more calories they consumed.

Next, we tested whether mood changes during the *taste task* had an effect on caloric intake. Mood change scores (T2-T3) were calculated by subtracting the mood score at the beginning of the taste task (T2) from the mood measurement at the end of the taste task (T3). Depressive symptoms (BDI-II), mood changes (T2-T3), and their interaction term were entered into the equation with caloric intake as dependent variable. Again a significant interaction effect was found between depressive symptoms and mood change during the taste task (β=3.11, t(65) =2.84, p<.01). Depressed BED participants who consumed more calories during the taste task experienced more positive mood changes than less depressed BED participants.

In order to further examine the effect of depression, caloric intake, and mood change, the total group was divided by means of the BDI-II (Van der Does, 2002) into a moderately to severely depressed group (n =36; BDI-II score 20 or higher; M=29.4, SD 7.6), and a non- to mildly depressed group (n =30; BDI-II score between 0 and 19; M=11.0, SD 6.2). We tested within the "non to mildly"-depressed group and the "moderate to severe" depressed group separately, whether the changes in mood during the mood induction (T1-T2) and the changes in mood during the taste task (T2-T3), influenced caloric intake. A positive linear relation was found between caloric intake and mood changes during the mood induction (β=-78.73, t(1,34)=2.89, p<.01) and also between caloric intake and mood change during the taste task (β=70.03, t(1,34)=3.2, p<.01) in the group with high depressive symptoms (see Figure 2). Correcting for condition (suppression versus natural emotions) and baseline mood score (VAS) did not change the results. In general, participants' mood was negatively affected by the film and returned to baseline after the taste task. Within the depressive group, those depressed participants who experienced the greatest mood shift also tended to eat the most and those who experienced the smallest mood shift tended to eat the least. In contrast, within the group with no or mild depressive symptoms, caloric intake was unrelated to the extent of mood changes from the mood induction (β=28.04, t(1,28)=1.41, p=.17, ns) and the taste task (β=-22.72, t(1,28)=0.24, p=.21, ns)(see Figure 2). Results also did not change after controlling for condition and BMI.

Figure 2: Linear regression with total caloric intake as dependent variable and mood changes during taste task (T2T3) as the independent variable.



Finally, we tested in two separate regression analyses whether perceived loss of control over eating and the perceived food intake could be predicted by severity of depressive symptoms by correcting for condition and the actual caloric intake. The main finding was that the perceived loss of control and perceived food intake were explained by actual caloric intake (β=-.62, t(3,61)=-5.92, p<.01 and β=-.61, t(3,61)=-5.96, p<.01, resp.) and not influenced by the severity of depressive symptoms ('no-mild'-depressed versus 'moderate-severe' depressed) (β=-.025, t(3,61)<1, ns and β=.13, t(3,61)=1.24, ns, respectively).

Discussion

The present study examined the role of negative emotions on binge eating in individuals with Binge Eating Disorder (BED). The first aim was to investigate whether binge eating is a consequence of regulating one's negative emotions. We hypothesized that a limited inner source of self-control is depleted after a first act of self-control, i.e. regulating one's emotions. The results did not support this expectation: BED participants who attempted to suppress their emotions while watching an upsetting movie did not consume more calories in a subsequent taste task compared to those who did not suppress their emotions. It could be argued that the participants in the suppression condition did not suppress their emotions enough to deplete their inner resources. However, our manipulation check indicated that participants in the suppression condition attempted to suppress their emotions significantly more than participants in the natural reactions condition.

Our second hypothesis was that suppression of negative emotional reactions would induce greater calorie intake in depressed BED participants. We hypothesized that severely depressed BED-participants have fewer resources available to regulate their negative mood effectively (Baumeister et al., 2000). No interaction effect was found between depressive symptoms and condition (suppression versus natural reactions). Suppression of negative emotional reactions in the moderately to severely depressed group did not lead to more caloric intake compared to suppression in the not to mildly depressed group. Our results thus showed that there is a positive association between depressive symptoms and caloric intake irrespective of condition (suppression versus natural reactions).

It might be hypothesized that overeating is related to an immediate repair of negative mood. Sad people often indulge in fattening snacks because, as Tice et al. (2001a) concluded in their study, they believe that eating repairs their mood. Continuous attempts to control one self are strongly guided by people's expectations and cognitions about how self-control works (Martijn et al., 2002). They expect that fattening foods will improve their mood. People tend to abandon or violate their normal self-regulatory efforts (control over eating) because they give priority to affect regulation.

Individuals with BED who had severe depressive symptoms consumed more calories than individuals who had no or mild depressive symptoms. Furthermore, within the severely

depressed group, those participants who experienced more negative mood change during the film fragment (manipulation) and those who reported more positive mood change during the taste task had a higher caloric intake (not related to experimental condition (suppression or natural reactions)). From these data one might conclude that depressed BED participants seek to regulate acute negative states, an inclination which the availability of high calorie food reinforces.

In contrast, caloric intake in the group with no or mild depressive symptoms was unrelated to mood changes during either the mood induction or the taste task. Why did we not find a correlation between mood changes and caloric intake in the non-depressed group? Individuals with BED *and* moderate-to-severe depressive symptoms might have less adequate coping strategies to regulate acute mood changes than those without a depression. The extent to which individuals believe that they can effectively cope with negative moods might play a role in the initiation and maintenance of eating disorder symptomatology. Depressed people (with BED) are less able to cope with negative moods and are therefore more likely to engage in ineffective coping strategies such as binge eating. Depressive symptoms such as increased appetite, hopelessness or feeling of failure may also contribute to increased caloric intake (Presnell, Stice, & Tristan, 2008). Non-depressed subjects with BED only have to regulate the temporal negative mood swings and stop eating when they feel better. Depressed subjects still feel gloomy or sad after regulating the negative mood dip and therefore tend to eat more and/or longer. Notable is that fact that the sense of loss of control is not related to depression. Feelings of loss of control and perceived caloric intake are both positively related to actual intake.

In our study mood worsened as a result of the mood induction and returned back to baseline after the taste test. It could be argued that the restoration of mood after the taste test was a natural consequence of time and is not attributable to eating. However, our interpretation that overeating serves as a means to repair one's negative mood is in accordance with several other studies which have also found that (binge) eating (temporarily) decreases the level of negative emotions (Deaver, Meidinger, Crosby, Miltenberger, & Smyth, 2003; Macht & Mueller, 2007; Munsch et al., 2008). Affect is reported to change over the course of binge eating. Eating makes binge eaters feel better, at least in the short run, or it softens their negative emotions (McManus & Waller, 1995; Heatherton et al., 1991). A hypothesis for a next study is thus that the specific expectation of mood improvement by the consumption of high calorie food leads to overeating.

In clinical practice, therapists should pay attention to patients with BED and severe depressive symptoms especially those who experience large mood swings. These patients tend to eat larger amounts of food during binge eating episodes and are therefore more prone to become (more) obese. Also, extra treatment could be offered for the depressive symptoms for example by means of anti-depressant medication or psychotherapy directed at the depression. It might be useful to learn more adaptive affect regulation skills.

In summary, our main findings were that in individuals with BED, the initial mood before watching the movie was restored after eating. Overeating might serve as a means to repair one's negative mood. Furthermore, more severe depressive symptomatology was correlated with a higher caloric intake. Moreover, within the group of BED participants with moderate to severe depressive symptoms, a higher caloric intake was observed for those who were most affected by the negative mood induction. This correlation was not found in the non-depressed BED group. The hypothesis that suppression of negative emotional reactions would lead to overeating in binge eaters was not supported.

Chapter 7

How expectancies, mood and overeating relate in Binge Eating Disorder: Beware of the bright sight

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Submitted for publication

Summary

Sad people often indulge in fattening snacks because they *believe* that eating will repair their mood. To test whether 1) changes in expectations and mood had an effect on caloric intake and 2) depressive symptoms moderated caloric intake, 73 women with Binge Eating Disorder were randomly assigned to a condition in which expectations about food and emotion were either confirmed or disconfirmed. Subsequently they were shown either an upsetting or an amusing movie clip followed by a taste task. Contrary to our expectations, there were no differences in the four conditions: participants in all four conditions ate comparable amounts of calories. Manipulation of expectations or mood had no effect on caloric intake. However, higher baseline expectations that food is pleasurable and useful as a reward resulted in a higher caloric intake after positive mood induction. Non-depressed individuals ate less after a negative mood induction than did depressed individuals. Interestingly, they also ate less than the group of individuals, depressed and not, whose mood was positively induced. Non-depressed individuals seem to use healthier coping strategies: negative affect signals that the environment poses a problem. Positive affect on the other hand signals that the environment is benign, and thus makes people less vigilant about food intake.

Introduction

Negative mood states influence eating behaviors in patients with Binge Eating Disorder (BED). Several cross-sectional and therapy outcome studies suggest that depressive symptoms (trait), acute negative mood (state), and binge eating behavior are related (Telch & Agras, 1994b; Antony et al., 1994; Grilo et al., 1994; Mussell et al., 1996b; Stice et al., 2001a; Grilo et al., 2001; Peterson et al., 2005; Dingemans, Spinhoven, & Van Furth, 2007b). The findings about the causality of mood and binge eating however are inconclusive. Four experimental studies (Telch et al., 1996a; Agras et al., 1998; Chua et al., 2004; Munsch et al., 2008) investigated the causal relation between mood and binge eating in patients with BED. One study (Chua et al., 2004) found that acute negative mood elicited increased eating in patients with BED compared to a neutral mood. Three other studies did not find differences in calories consumed following negative or neutral mood induction (Telch et al., 1996a; Agras et al., 1998; Munsch et al., 2008). One hypothesis was that negative mood might influence BED participants' perception of their eating behavior, and therefore lead to the labeling of an eating episode as a binge (Telch et al., 1996b; Agras et al., 1998). Another suggestion was that binge eating often occurs in negative moods because eating decreases negative mood rather than negative mood being a trigger for binge eating (Munsch et al., 2008).

The pervasiveness of the association between emotional distress and binge eating suggests that the question is not *whether* but *how* negative affect leads to overeating. Sad people often indulge in fattening snacks because they *believe* that eating repairs their mood (Tice, Bratslavsky, & Baumeister, 2001b; Deaver et al., 2003; Macht et al., 2007). Behaviour, and especially persistent efforts to control oneself, are strongly influenced by people's expectations and cognitions about how self-control works (Martijn et al., 2002). People tend to abandon or violate their normal self-regulatory efforts because they give priority to affect regulation. It might be hypothesized that they *expect* that fattening foods will improve their mood. In everyday life, frequent associations between food and emotions are made. One example is the recently launched media campaign by Mars® in which the name "Mars®" is temporarily replaced by "happy", "love," or "feel good" on the wrapper of the well-known chocolate bar. Frequent exposure to these kinds of messages in the media or elsewhere influences people's expectations regarding food and mood. The expectancy learning theory postulates that one forms expectations of the consequences of various behaviors as a result of one's learning history (Smith, Simmons, Flory, Annus, & Hill, 2007). These expectations influence future behavioral choices. Expectations about the consequences of a given behavior are the sum of one's learning history and are thus the cognitive mechanism by which prior learning leads to subsequent behavior. The expectation that eating helps alleviate affect might predict increases in binge eating (Stice, 2001). Binge eating is therefore likely to be the result of extreme expectations (Smith et al., 2007).

The aim of the present study was to test the relationship between expectations regarding the effect of eating on mood, changes in mood, and actual caloric intake in individuals with BED. Our research questions were twofold:

1) Do expectations regarding food and mood have an effect on caloric intake? We expected that participants with high expectations that food will improve mood to have a larger caloric intake when in a negative mood than participants with low expectations.

2) Do depressive symptoms influence caloric intake and is there an interaction effect with mood induction? We hypothesized that participants with severe depressive symptoms, especially those with a negative mood induction, would have a higher caloric intake than participants with no-to-mild depressive symptoms.

Method

Participants

Participants in this study were 73 non-pregnant females between 18 and 60 years old with a primary diagnosis of binge eating disorder (BED) according to DSM-IV criteria (American Psychiatric Association, 1994) or subthreshold BED (an average of one binge eating episode a week).

To be included in the study, a participant had to report an average of one binge eating episode a week over the previous 24 weeks. Women with a subthreshold BED were also included in the study because they do not seem to differ significantly from patients with full-syndrome BED (an average of two or more binge eating episodes a week) (Striegel-Moore et al., 2000). If participants were on medication, they could participate in the study if they were stable on medication. Participants were recruited from clinics specializing in treating eating disorders (*n*=34; 47 %), by advertisements in local newspapers, and via Internet websites (*n*=37; 53%).

The Dutch Medical Ethics Committee for Mental Health Institutions approved the study.

Measures

Demographic variables: Participants' marital status, socioeconomic status (homemaker/retired, fulltime job/student, part-time job, or disabled), educational level (low, medium, high), and age were recorded.

Eating Disorder Examination: The Eating Disorder Examination (EDE) (Cooper et al., 1987; Jansen, 2000) is an investigator-based, semistructured interview for the assessment of eating disorder-specific psychopathologies. The EDE assesses the psychopatholgy of the participant in the previous four weeks. It provides a comprehensive profile of individual psychopathology based on scores on four subscales: restraint (e.g., attempts to avoid certain foods), eating concern (e.g., concern about being seen while eating), shape concern (e.g., importance of body shape in self-evaluation), and weight concern (e.g., dissatisfaction with

body weight). A global scale of eating pathology (computed as the mean of the participants' scores on the four subscales) is also computed to assess overall eating psychopathology. Items are rated on 7-point forced-choice scales (0-6), with higher scores reflecting greater severity or frequency. The EDE also assesses two key behavioral aspects of eating disorders: overeating and the use of extreme methods of weight control. Weight and height were also assessed and Body Mass Index (BMI) was calculated (weight/height^2).

Beck Depression Inventory-II: The Dutch version of the Beck Depression Inventory-II (Van der Does, 2002; Beck, Steer, & Garbin, 1988) contains 21 items, each with four self-evaluative statements rated on severity (scored from 0 to 3). The BDI-II measures severity of depressive symptoms. The total score is a simple sum of the 21 individual item scores and may range from 0 to 63. For patients diagnosed as having an affective disorder, the following guidelines are suggested as BDI cut-off scores: no or minimal depression, < 13; mild depression, 14 – 19; moderate to severe depression, 20 – 28; and severe depression, 29 – 63. The internal consistency of the Dutch version of the BDI-II is high for a psychiatric outpatient group and a healthy control group: in tests, Cronbach's alpha was 0.92 and 0.88 respectively. Test-retest reliability correlation in the same groups was $r = 0.82$.

Taste task and food intake: During the taste task, each participant was asked to take a seat behind a table with four pre-weighted bowls with large quantities of food: chocolate (M = 813 grams/4352 kcal, SD = 102 grams/548 kcal), potato chips (M = 149 grams/797 kcal), SD = 27 grams/145 kcal), candies (M = 616 grams/2229 kcal, SD= 62 grams /226 kcal), and cake (M = 464 grams/1950 kcal, SD 45 grams/187 kcal). In the taste test, participants rated the four kinds of foods on palatability, enticement to eat, taste, and smell (Dingemans, Martijn, Jansen, & van Furth, 2009). Filling out the questionnaire gave participants a chance to consume the foods. Data were not analyzed except the item that measured how much (from 'not at all' (0) to 'very much' (5)) they liked the four kinds of food. The total amount of food left was measured afterwards and total caloric intake was calculated.

Manipulations

Food-mood expectation manipulation: Participants' expectations about the relationship between food and mood were manipulated by means of a computerized questionnaire with pre-programmed feedback on each answer. We presented the questionnaire as a means "to test your knowledge about food," the Fact or Fiction Food Questionnaire (FFFQ). Ten statements about food, each of which had a link between food and emotions, and five filler statements, were displayed on a computer screen. Participants had two reply options, 'true' or 'false.' Immediately after answering each statement, they received feedback on their answer. The participants in the confirmative condition got the version in which the relationship between food and mood was positively confirmed (confirmative version) and the participants in the disconfirmative condition received the version of the FFFQ in which the positive relationship between food and mood was disconfirmed (disconfirmative version). For example, the first

statement was, 'Chocolate contains substances that make you happy'. In the confirmative condition, the participant received the following feedback if she answered 'true': 'Indeed, chocolate contains fenylethylamine which makes you happy' and if she answered 'false', 'On the contrary, chocolate contains fenylethylamine which makes you happy'.

In the disconfirmation condition she got the following feedback if she answered 'true': 'Chocolate does not make you feel happy. Chocolate contains a small concentration of fenylethylamine, which could only make you happy if you ate large quantities of it, such as a truck container'. If she chose 'false', 'Indeed, this is true. Chocolate does not make you feel happy. Chocolate contains a small concentration of fenylethylamine, which could only make you happy if you ate large quantities of it, such as a truck container' (see Appendix 1 for complete questionnaire).

Mood-induction Participants were instructed to watch a three-minute fragment of a movie, either from *The Champ* (negative mood induction) or *When Harry met Sally* (positive mood induction), to which they were randomly assigned. Studies by Gross and Levenson (Gross et al., 1995) showed that these fragments of the two movies elicit sadness and amusement, respectively. All participants received the same instruction: *'In a moment, you will see a fragment of a movie. This fragment may evoke emotions. Watch the fragment with attention and let in the emotions you feel and react to them as you please. Before the fragment starts, the screen will be blank for a minute. Try to clear your mind of all thoughts and emotions. I will leave you alone while you watch the fragment and will return afterwards'.*

Manipulation check

Eating Expectancy Inventory (EEI) The EEI (Hohlstein, Smith, & Atlas, 1998) is a 34-item self-report measure of expectations about the consequences of eating. It consists of five subscales. The present study used two relevant subscales: "Eating helps manage negative affect" (in this study, Cronbach's alpha= 0.87) and "Eating is pleasurable and useful as a reward" (in this study: Cronbach's alpha=0.70). The subscale "Eating helps manage negative affect" consists of 18 items. Examples of items are "Eating can help me bury my emotions when I don't want to feel them" and "Eating helps me forget or block out negative feelings like depression, loneliness or fear." The other subscale, "Eating is pleasurable and useful as a reward," consists of 6 items. Examples of items are "When I do something good, eating is a way to reward myself" and "Eating is fun and enjoyable." Mean total scores of the two subscales ranged from 1 to 7. A higher score indicated higher expectations.

Mood scale (adapted from (Martijn et al., 2002) Current mood state was measured by seven adjectives (sad, tense, tired, cheerful, active, irritated, and gloomy) on a 100 mm Visual Analogue Scale (VAS) on which the end points were labeled "not at all" and "extremely." The mean score on these seven adjectives were calculated (in this study Cronbach's alpha =0.82).

Five items (sad, tense, tired, irritated, and gloomy) were recoded such that a lower score indicated a more negative mood.

Suspicions and awareness Participants completed a funneled debriefing form (Bargh et al., 2000), which checked for awareness and suspicion.

Procedure

Respondents who reacted to the call for a study on "perception and reactions of persons with binge eating episodes" were first screened by telephone or email in regard to the inclusion and exclusion criteria. Potential participants (n=153) were sent an information letter, a screening questionnaire, and the Eating Expectancy Inventory (EEI) (Hohlstein et al., 1998). The EEI was used to measure baseline (T0) expectations regarding eating. Eighty individuals did not participate in the study because of a lack of interest (n=67) or not meeting inclusion criteria (n=13). In total, 73 individuals participated in the study and were invited to the Center for Eating Disorders "Ursula." All participants signed an informed consent form and received a monetary reward of 15 Euros. The total duration of the experiment was one and a half hours. Participants were instructed not to eat 2 hours prior to the assessment and were tested individually. They were randomly allocated to one of the four conditions from a 2 (Food/mood repair expectation confirmation versus expectation disconfirmation) by 2 (Mood induction, positive versus negative) between-subjects factorial design.

Before the start of the experiment, each participant was interviewed by means of the Eating Disorder Examination (EDE) (Cooper et al., 1987; Jansen, 2000) and asked to fill out the Beck Depression Inventory-II (BDI-II) (Van der Does, 2002). After completion of the interview and questionnaire, the participant was seated in front of a monitor. Then, food/mood expectations were manipulated. The participant filled out the Fact or Fiction Food Questionnaire (FFFQ) on the computer. The participant filled out the EEI again to check the manipulation. Before the mood induction (movie fragment) started, the participant filled out the mood questionnaire (T1). After each participant watched the movie fragment, her mood was measured again (T2). Subsequently she took part in the 15-minute taste task. The four bowls with food were placed on a separate table previously masked by a folding screen. The participant was instructed to taste all four kinds of food and to fill out the taste perception questionnaire. She was instructed to evaluate the food, and she was told to feel free to eat as much as she liked or thought was necessary to evaluate each food. During the taste task, participants were left alone in order to minimize uncomfortable or negative feelings that might have arisen if someone watched them eat. The time allotted for assessment was the same for all four conditions. After the taste task, the participant filled out the mood scales again (T3), and completed the questionnaire to assess feelings about loss of control, perceived quantity eaten during the taste task, and suspicions about the purpose of the study. At the end of the experiment, participants filled out the mood scales one last time (T4) and were fully debriefed.

There were thus four participant conditions. Confirmative Positive (CP) and Confirmative Negative (CN) received confirming feedback about the relationship between mood and food but one group saw a mood-uplifting movie fragment, the other a sad one. Disconfirmative Positive (DP) and Disconfirmative Negative (DN) received disconfirming feedback about food and mood followed by either positive or negative movie fragment.

Statistical analyses

Possible pre-experiment differences between the four condition cohorts in regard to demographics and clinical variables, differences in levels of expectation, negative affect, and mood during mood induction were checked by means of independent sample t-tests, AN(C)OVA, or chi-square tests where appropriate. Subsequently, the three hypotheses, i.e. the three research questions, were tested by means of linear regression analyses. The dependent variable 'total caloric intake' was log-transformed because it was not distributed normally.

Results

Participant characteristics

No differences were found between the four conditions with respect to the demographic variables socioeconomic status (χ^2 (6) = 0.37, *ns*) or level of education (χ^2 (6) = 4.4, *ns*). Participants in the negative mood induction condition lived more often alone than those in the positive mood condition (marital status (χ^2 (3) = 12.6, p < 0.01). Participants in confirmative negative (CN) and disconfirmative positive (DP) were significantly older (*M*=45.6, SD=10.0 and *M*=41.2 SD=7.3 respectively) than participants in the confirmative positive (CP) and disconfirmative negative (DN) condition (*M*=35.2, SD=10.7 and *M*=32.6, SD=10.9 respectively)($F(3,69) = 6.73$, $p < 0.01$). Forty-seven percent of the participants were currently on a waiting list for treatment and 53% of the participants had never sought treatment for binge eating.

There were no significant differences between the four conditions in regard to the clinical variables (see Table 1). All participants stated on the Taste Test Questionnaire that they liked at least two kinds of food (score 3 or higher). None of the participants reported any awareness of the true nature of the hypothesized connection between the mood induction, suppression instructions, and the taste test.

Manipulation checks

Expectation manipulation A 2-between x 2-within ANOVA was conducted with the factors "food/mood repair expectations" (confirmative versus disconfirmative condition) and time (T0 versus T1) as independent variables and scores on the EEI subscale "eating helps manage negative affect" as the dependent variable. We failed to find the predicted interaction

Table 1: Baseline characteristics of all participants[3].

	All participants (N=73) (*M*, S.D.)	Range	Statistical Tests for the four conditions and significance
Objective binges episodes /28 days	10.7 ± 14.8	2 - 84	*F* (3.69)=0.75, *ns*
Subjective binge episodes /28 days	10.1 ± 14.8	0 - 100	*F* (3.69)=1.70, *ns*
Overeating episodes / 28 days	2.3 ± 5.1	0 - 28	*F* (3.69)=0.72, *ns*
EDE Restraint	1.63 ± 1.08	0 - 4.80	*F* (3.69)=0.72, *ns*
EDE Concern about eating	1.73 ± 1.22	0 - 5.40	*F* (3.69)=0.31, *ns*
EDE Concern about weight	3.28 ± 1.36	0.40 – 5.60	*F* (3.69)=0.58, *ns*
EDE Concern about figure	3.00 ± 1.34	0.63 – 6.00	*F* (3.69)=0.55, *ns*
BMI	34.7 ± 7.8	21.0 – 57.44	*F* (3.69)=0.69, *ns*
BDI (depressive symptoms)	22.6 ± 11.6	0 - 47	*F* (3.67)=0.61, *ns*
Mood (VAS) baseline (0-100 mm)	43.7 ± 19.5	10 - 96	*F* (3.69)=0.53, *ns*
EEI eating alleviates negative affect	4.79 ± 1.08	1.33 – 6.61	*F* (3.61)=1.66, *ns*
EEI pleasurable and useful as reward	4.97 ± 1.38	1.00 – 7.00	*F* (3.61)=0.78, *ns*

effect (*F*(1,63)=0.49, *ns*) (confirmative condition: T0: *M* = 4.93, SD = 1.04; T1: *M*=4.85, SD= 1.15 and disconfirmative condition: T0: *M* = 4.65, SD = 1.11; T1: *M* = 4.45, SD= 1.24).

Subsequently, a 2-between x 2-within ANOVA was conducted with the "food/mood-repair-expectation" factors (confirmative versus disconfirmative condition) and time (T0 versus T1) as the independent variables and scores on the EEI subscale 'eating is pleasurable and useful as a reward' as the dependent variable. A significant interaction effect was found (*F*(1,63)=4.08, *p*< .05). Participants who were confirmed in their expectation that food and emotions are positively linked (confirmative condition) showed a significant increase in the expectations that eating is pleasurable and useful as a reward after the manipulation (T0: *M* = 4.85, SD = 1.34; T1: *M*=5.28, SD= 1.17) whereas the participants in the disconfirmative condition did not (T0: *M* = 5.08, SD = 1.43; T1: *M* = 5.11, SD= 1.36).

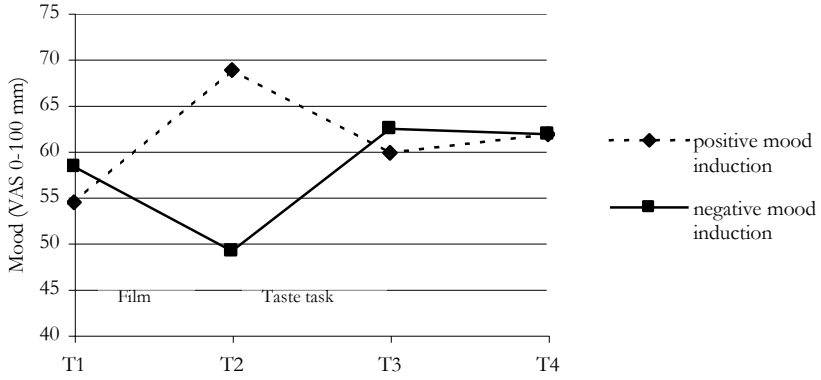
In sum, the manipulation of the expectations (confirm versus disconfirm) did not succeed in changing expectations that 'eating helps to manage negative affect' but succeeded in changing expectations that 'eating is pleasurable and useful as a reward' did: participants in the 'confirmative' condition had significantly higher scores on the subscale "pleasure/reward' of the EEI than participants in the 'disconfirmative' condition.

Mood induction: A 2-between x 2-within ANOVA was conducted with the factors mood induction (negative versus positive condition) and time (T1 versus T2). As expected, there was a significant interaction effect of time x mood induction (*F*(1,63)= 9.22, *p*<.01), indicating that the mood of the participants in the negative mood induction condition became

[3] Mean scores of the total group are presented since there were no significant differences on baseline characteristics between the four conditions.

more negative after watching the upsetting movie fragment ($M_{negative}$ T1=58.3, SD=20.8 and $M_{negative}$ T2=49.1, SD=18.5) and the mood of the participants in the positive mood induction more positive after watching the cheerful movie fragment (($M_{positive}$ T1 = 54.4, SD=18.3 and $M_{positive}$ T2= 68.8, SD= 17.1); see also Figure 1: T1-T2). Thus, we conclude that the film fragments had the intended effect of inducing a negative and a positive mood state respectively.

Figure 1: Changes in mood of patients with Binge Eating Disorder



Hypothesis 1: Do expectations regarding food and mood have an effect on caloric intake? We hypothesized that participants with increased expectations that food will improve mood (confirmative condition) have a larger caloric intake after a negative mood induction compared to participants with decreased expectations (disconfirmative condition).

In order to test this first hypothesis about the effect of changes in 'food/mood-repair-expectations' and mood on caloric intake, we conducted a linear regression analysis with caloric intake as dependent variable and mood induction (negative versus positive), 'food/mood-repair-expectations' (confirmative versus disconfirmative condition) and their interaction as main predictors. No main effects for 'mood induction' (β= -.14, t(72)= 0.57, *ns*) and 'food/mood-repair expectation' (β= .18, t(72)= .79, ns) were found. Also, the expected interaction effect (β= .13, t(72)= 0.39, *ns*) between these two variables could not be confirmed[4]. Thus, no differences were found between the four conditions with regard to calories consumed (Confirm-negative (CN): M= 349 kcal, SD= 281; Confirm-positive (CP): M= 376 kcal, SD= 235; Disconfirm-Negative (DN): M= 466 kcal, SD=325; Disconfirm-positive (DP): M= 484 kcal, SD= 334; F (3,69)= 0.91, *ns*).

[4] Correcting for age and BMI did not change the results.

Subsequently, we tested whether the degree of expectations (baseline) as measured by the EEI prior to mood induction (T1) had an effect on caloric intake. Do expectations that 'eating will alleviate negative affect' or expectations that 'eating is pleasurable and useful as a reward' moderate caloric intake? In other words, do participants with high scores on the EEI have a higher caloric intake?

Again, we conducted a linear regression analyses with caloric intake as the dependent variable and mood induction (negative versus positive), EEI (subscale "alleviate negative affect") at T1, and the interaction between EEI scores and mood induction as the independent variables. No main effect of expectations at T1 that eating alleviates negative affect (β= 0.004, $t(69)$=0.05, *ns*) and no interaction effect between these expectations and mood induction was found (β=0.10, $t(69)$=0.65, *ns*).

Finally, we performed a linear regression analyses with caloric intake as the dependent variable and mood induction (negative versus positive), EEI subscale 'pleasurable and useful as a reward' at T1and the interaction with mood induction as predictors. We found a significant main effect for the EEI subscale 'eating is pleasurable and useful as a reward' (β=0.27, $t(69)$=3.10, p< .01) and a significant interaction effect with mood induction (β=-0.34, $t(69)$=2.53, p=.014). Lower expectations that eating is pleasurable and useful as a reward were related to a lower caloric intake after a positive mood induction. No significant correlation with these expectations and caloric intake was found after a negative mood induction.

Hypothesis 2: Do depressive symptoms have an effect on caloric intake and is there an interaction with mood induction? We hypothesized that participants with severe depressive symptoms (as measured by the BDI-II) would have a higher caloric intake, especially after a negative mood induction, than participants with no-to-mild depressive symptoms.

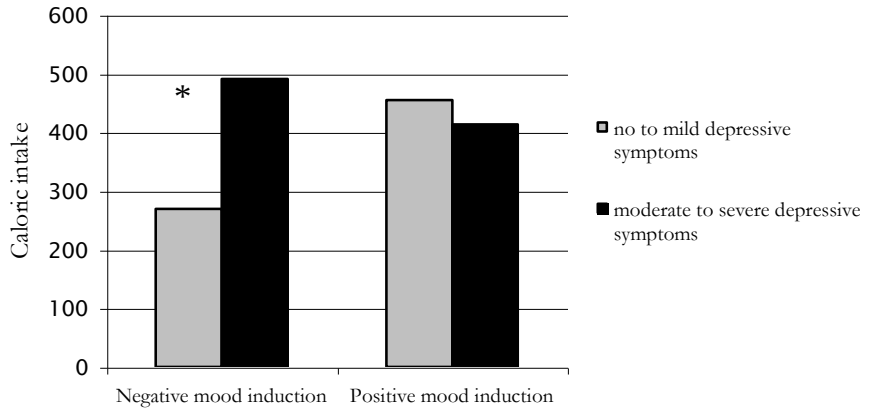
To test this second hypothesis that the level of depressive symptoms (as measured by the BDI-II) would moderate caloric intake after a negative mood induction, but not after positive mood induction, we conducted a linear regression analysis with caloric intake as dependent variable and mood induction (negative versus positive), depressive symptoms (BDI-II), and an interaction term (BDI-II x mood induction) as independent variables. The total group was split by means of the BDI-II into a moderately-to-severely depressed group (n =45; BDI-II score 20 or higher; M=29.6, SD 7.5), and a non-to-mildly depressed group (n =26; BDI-II score between 0 and 19; M=10.35, SD= 5.7).

No significant main effect of depressive symptoms was found (β=-0.18, $t(69)$=0.79, *ns*). A significant interaction effect of mood induction and depressive symptoms was found (β=0.82, $t(69)$=2.47, p< 0.05. After negative mood induction, the participants with no-to mild symptoms (M=270, SD=172) consumed fewer calories than the participants with moderate-to-severe depressive symptoms (M= 491, SD=340)(β=0.64, $t(33)$=2.79, p>0.01). In contrast, caloric intake within the positive mood induction condition was unrelated to the severity of

depressive symptoms (β=-0.18, t(36)=0.76, *ns*) (no-mild depressive symptoms M=455, SD=295; moderately-severe depressive symptoms M= 414, SD=292). (See Figure 2.)

To test whether changes in mood after the mood induction (T1-T2) had an effect on caloric intake we added mood change scores (T1-T2) and an interaction term (Mood T1-T2 x Mood Induction). Mood change scores (T1–T2) were calculated by subtracting the mood score at the beginning of the mood induction (T1) from the mood measurement at the end of the mood induction (T2). We found a significant interaction effect (β=0.031, t(67)=2.2, p< 0.05). Larger negative mood changes during the negative mood induction resulted in a higher caloric intake (β=0.033, t(34)=3.06, p< 0.01). In the positive mood induction condition, caloric intake appeared to be unrelated to the degree of mood change (β=-0.007, t(37)=1.00, *ns*).

Figure 2: Mean caloric intake after a negative or a positive mood induction in BED participants with no-to-mild (light columns) or moderate-to-severe (dark columns) depressive symptoms



Course of mood over time

A 2-between x 4-within ANOVA (repeated measures) test was conducted with the factors mood induction (negative versus positive condition) and time (T1, T2, T3 versus T4) to evaluate the course of mood during the experiment. As expected, there was a significant interaction effect of time x mood induction (F(3,67)= 21.19, p<.01) (see Figure 1). As stated above, contrast analyses (T1 versus T2) revealed that the mood of the participants receiving the negative mood induction became sadder after watching the negative movie fragment and the mood of the participants during the positive mood induction more cheerful after watching the positive movie fragment.

Furthermore, contrast analyses (T2-T3) indicated that the mood of the participants in the negative mood induction condition became significantly more cheerful after the taste task and that the mood of the participants in the positive mood induction more sad after the taste task ($F(1,69)=30.28$, $p<0.01$). Mood remained unchanged for both conditions between T3 and T4 ($F(1,69)=0.92$, ns).

Finally, a significant overall main effect of time was found ($F(3,67)= 3.73$, $p<.05$). The participants in both mood conditions had a significantly more positive mood at the end of the experiment (T4) compared to the beginning (T1).

Discussion

The aim of the present study was to test the relationship between expectations regarding the effect of eating on mood, changes in mood, and actual caloric intake in individuals with BED. Participants were either randomly assigned to a 'confirmative' condition in which expectations that food and mood are related, were confirmed or to a 'disconfirmative' condition in which these expectations were disconfirmed. Subsequently participants were randomly assigned to either a negative or a positive mood induction procedure. Finally, everyone participated in the same taste task. Our main dependent variable was caloric intake during a taste test.

Contrary to our expectations, we did not find a main or interaction effect of food/mood repair-expectations (confirmative versus disconfirmative condition) or of mood induction (negative versus positive) on caloric intake. The participants in all four conditions ate comparable amounts of calories. After the 'fact-or fiction food' mood manipulation, participants in the 'disconfirmative' condition did not have significantly lower expectations that eating would alleviate negative affect than participants in the 'confirmative' condition. Clearly our manipulation was not long or strong enough to alter the expectations of individuals with BED that eating alleviates negative mood. The majority of the participants had had binge eating episodes for many years, which makes it possibly hard to alter potentially long-standing associations between mood and food. However, after completing the manipulated questionnaire, participants in the 'confirmative' condition were significantly more firmed in their expectation that eating is pleasurable and useful as a reward than the participants of the 'disconfirm' condition. Participants in the positive mood induction, who had higher degree of baseline expectations that food is pleasurable and useful as a reward, consumed more calories than participants with a lower degree of expectations. The information they received in the confirmative condition was congruent with their expectations that food is pleasurable and useful as a reward. It appears to be far more difficult to alter expectations when the information received is incongruent.

A positive association was found between depressive symptoms and caloric intake after a negative mood induction: participants with no-to-mild depressive symptoms consumed fewer calories than participants with moderate-to-severe depressive symptoms, which we also

found in a previous study (Dingemans et al., 2009). After a positive mood induction no significant differences were found in caloric intake between the no-to-mild depressive group and the moderate-to-severe depressive group (see Figure 1).

The most remarkable finding was that individuals with no-to-mild depressive symptoms ate less after a negative mood induction: they seem to have been able to resist the food better than the other three groups. We had expected to find an overall higher caloric after a negative mood induction than after a positive mood induction since several studies have found a positive link between acute negative affect and binge eating (Grilo et al., 1994; Waters et al., 2001; Greeno et al., 2000). However, Cools and others (Cools, Schotte, & McNally, 1992) also found no differences in caloric intake in a student sample after a positive and a negative mood induction in a student sample. Why do these non-depressed BED-individuals behave differently? Overall, individuals react differently in positive and negative mood states. Different emotions could have a different effect on food intake even in one individual (Macht, 2008). Emotions and current mood influence the decisions that we make. When in a happy mood, people tend to judge stimuli more positively than when in a negative mood and vice versa (Schwarz & Clore, 2003). Negative affect signals that the environment poses a problem, whereas positive affect signals that the environment is benign. As a result, negative affect may trigger detail-oriented, thinking processes, which are usually responsive in handling problematic situations. In contrast, positive affective states, by themselves, signal no particular need for action, and happy individuals may hence not be motivated to expend cognitive effort unless other goals call for it. It could be hypothesized that when patients with BED experience a negative mood, they want to escape from these negative emotions. Binge eating may be used as a means to escape from these negative thoughts and worries and may in this way help to alleviate emotional stress (Heatherton et al., 1991). BED patients with no-to-mild depressive symptoms seem to be better able to use healthy coping strategies than BED patents with moderate-to-severe depressive symptoms. Depressed people (with BED) are less able to cope with negative moods and are therefore more likely to engage in ineffective coping strategies, such as binge eating. Depressive symptoms such as increased appetite, hopelessness, or feelings of failure may contribute to increased caloric intake (Presnell et al., 2008). Apparently, the non-depressed individuals with BED behave differently when in a positive mood. The positive mood signals that all is well, so they enjoy the moment and do not worry about restricting high calorie food.

Also of interest is the fact that at the end of the experiment, in both mood induction conditions (positive and negative), mood was significantly higher than at baseline. Mood improved again during the taste task after the negative mood induction. That overeating may serve as a means to repair one's negative mood is in accordance with the results of our previous study (Dingemans et al., 2009). The majority of the general population reports positive emotions or feelings such as enjoyment associated with the taste, happiness, and immediate satisfaction while eating high caloric food (Dunn, Mohr, Wilson, & Wittert, 2008).

Also, the fact that food and positive moods are being linked, as illustrated in the Introduction by the example of the recent Mars© candy bar advertisement campaign, adds to this hypothesis.

Again we found that participants who were more affected by the negative mood induction consumed more calories. In the positive mood condition no relation was found between degree of positive mood changes and caloric intake. Mood decreased after the taste task in the positive mood induction group. Perhaps the awareness of eating (too much) high caloric food engenders feelings of guilt over not restricting food intake. However, on average, at the end of the experiment, mood was more cheerful then at baseline. It is possible that binge eating often occurs when people are in a negative mood, not because negative mood is a trigger for binge eating, but rather because binge eating decreases negative mood (or "improves mood")(Munsch et al., 2008). Future BED studies should include a condition without a taste task, in order to evaluate the natural course of mood after a positive or negative mood induction.

In clinical practice it is important to be aware of the fact that for patients with binge eating disorder, positive mood is also a trigger for eating. In general, patients with BED are aware that negative moods are linked with binge eating. The results of this study might indicate that there are different reasons and/or processes for eating in negative and positive moods. However, untreated people with BED generally gain weight (Fairburn et al., 2000; Cachelin et al., 1999), so making them more aware of the moods which trigger eating episodes might help prevent weight gain.

Appendix

Food-mood-expectancies manipulation: Fact or Fiction Food Questionnaire (FFFQ)

1. Chocolate contains substances that make you happy.

- Feedback confirmative condition: chocolate contains fenylethylamine, which makes you happy.
- Feedback disconfirmative condition: Chocolate does not make you feel happy. Chocolate contains a small concentration of fenylethylamine, which only could make you happy if you eat large quantities of it, like a container.

2. You need sugar (like in cookies) to function well.

- Feedback confirmative condition: You need sugar to function well. It is a scientific fact that sugar is a carbohydrate. We need carbohydrates every day.
- Feedback disconfirmative condition: On the contrary sugar is addictive and has a counterproductive effect. Try to eat no sugar for three days: You start feeling detoxification phenomena like stress, trembling, irritations, sweating etc.

3. Potatoes (chips) contain 'bad' carbohydrates.

- Feedback confirmative condition: On the contrary, those carbohydrates are good. Potatoes contain vitamin B, C, potassium, phosphorus and dietary fibres and are free of proteins. Potatoes are easily digestible and they dehydrate.
- Feedback disconfirmative condition: Indeed, these 'fast' carbohydrates like sugar and starch, in potatoes (chips) are absorbed in the blood very quickly. This causes an imbalance between blood sugar and insulin, which makes you shaky.

4. Colouring agents, in for example sweets, pep you up and make you active.

- Feedback confirmative condition: Scientific research indeed showed that the colouring agents pep you up when eaten in the right doses. Colouring agents in sweets make you active.
- Feedback disconfirmative condition: Scientific research showed that the effect of colouring agents is negligible.

5. Junk food makes you happy.

- Feedback confirmative condition: Indeed, junk food contains serotonine (a substance in the brain). Serotonine gives you a pleasant feeling.
- Feedback disconfirmative condition: Junk food is addictive. Junk food contains substances that are addictive.

6. When it is hot, eating salty potato chips helps you feel more energetic.

- Feedback confirmative condition: Indeed, when you sweat you loose a lot of salt. The brains need salt to function well. If the level of salt decreases you feel tired and dizzy. Eating salt helps to refill these sources and you feel more energetic afterwards.
- Feedback disconfirmative condition: Human beings only need a little salt. The amounts of salt in food are more then enough even when it is hot and you are transpiring a lot. Salt shortage is very rare in healthy people.

7. Sport drinks do not give you energy.
- Feedback confirmative condition: Sport drinks contain several stimulating substances like caffeine, vitamins, minerals and sugar, which gives you energy immediately.
- Feedback disconfirmative condition: Indeed, an average sportsman with a healthy diet ingests enough minerals. Sport drinks contain a lot of calories, which do not give you more energy.

8. Drinking coffee is stimulating.
- Feedback confirmative condition: Coffee contains the stimulating agent caffeine. Drinking coffee peps you up.
- Feedback disconfirmative condition: Coffee only peps you for a very short period. After drinking too much coffee you feel exhausted, mentally as well as physically.

9. Eating candies and chocolate during the menses worsens the complaints.
- Feedback confirmative condition: On the contrary, eating candies and chocolate helps you to feel better during the menses. It contains substances that work like a anti-depressant. Your mood improves.
- Feedback disconfirmative condition: After eating chocolate and candies the level of estrogen (female hormone) raises. This might enhance complaints during the menses and results in more sensitive breasts.

10. Good fats don not exit.
- Feedback confirmative condition: Unsaturated fats are good fats. They help keeping your heart and blood vessels healthy. Oily fish contains omega-3 fatty acids, which enhance your mood and concentration.
- Feedback disconfirmative condition: You only need very small amounts of fat a day. Most fats we eat are very unhealthy like in snacks and cookies.

Filler questions:

11. Fresh vegetables contain more vitamins than canned or frozen vegetables.
Feedback both conditions: Before vegetables are canned or frozen, they are blanched. Therefore preparation time is shortened in comparison to fresh vegetables. After preparation for consumption there is practically no difference in vitamins.

12. It is sensible to drink a lot of orange juice.
Feedback both conditions: One glass of orange juice can replace one piece of fresh fruit. However, the second piece of fruit needs to be fresh.

13. Snacks between meals are important.
Feedback both conditions: Without snacks between meals the body does not receive enough energy for a long time. When the time between meals is too long, this can cause a hypoglycaemia.

14. Without eating meat you can live a healthy life.
Feedback both conditions: A vegetarian diet which contains vegetables, fruits, beans, bread, nuts, cereals and milk product, yields enough nutrients.

15. Food products that contain extra vitamins and minerals are healthier than 'normal' food products.

Feedback both conditions: A normal healthy diet contains enough vitamins and minerals. Normally, adding vitamins and minerals is unnecessary.

Chapter 8

Summary and general discussion

Summary and general discussion

Binge eating disorder (BED) was first added as a provisional diagnosis to the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)(American Psychiatric Association, 1994) under the eating disorders category with anorexia nervosa (AN) and bulimia nervosa (BN). BED is characterized by recurrent episodes of binge eating (American Psychiatric Association, 1994). An eating episode is interpreted as a binge-eating episode if the amount of food consumed is definitely larger than what most people would eat over a similar period and under similar circumstances. Eating must occur within a discrete period of time (two hours) and there must be a sense of lack of control over eating during that period. Most people who engage in binge eating eat alone because they are embarrassed by how much they eat. They also say that during binge eating episodes they eat much faster than usual and do not enjoy eating. They consume exceptionally large amounts of food even though they do not feel physically hungry. Afterwards they feel disgusted with themselves, depressed, or very guilty. The behaviour must be accompanied by a marked distress regarding the behaviour.

This thesis focused on patients with Binge Eating Disorder (BED). It consisted of three parts. The first part discussed the validity of the diagnosis of BED. The central question was whether there is enough evidence to establish that BED is a distinct eating disorder category and whether it should be admitted into the next version of the DSM. The second part described the results of a randomized controlled trial were described. The main aims of this trial were to explore the effectiveness of cognitive behavioural therapy (CBT) compared to a waiting list control group (WL) and to identify possible predictors and mediators of CBT for patients with BED. The third and last part presented the results of two psychological experiments. The main goal of these two experiments was to assess whether there is evidence for a causal relationship between negative affect and binge eating in patients with BED. The final Chapter summarizes and discusses these three parts. This final chapter also provides an update of the research regarding the validity and diagnostic criteria of BED published in the last five years.

Summary and discussion part I

Part 1 (Chapters 2, 3, and 4) addressed the question of whether BED is a distinct diagnostic eating disorder category. Chapters 2 and 3 provided an overview of the epidemiology, characteristics, aetiology, criteria, and course and treatment of BED. These chapters review the literature as published up to 2003. Chapters 2 and 3 were written in 2001 and 2003 respectively. In order to provide an accurate and up-to-date discussion about the validity of BED as a discrete diagnosis, this final Chapter reviews relevant studies published between 2004 and 2008. It discusses whether these studies support the conclusions drawn earlier in Chapters 2 and 3.

Distinct characteristics of BED, BN and 'normalcy' - The conclusion of both Chapters 2 and 3 is that BED stands between bulimia nervosa (BN) and obesity with regard to severity of psychopathology. In the majority of the patients with BED, obesity is a comorbid condition. Binge eating might be a primary symptom that leads to weight gain and obesity. More recent studies have concluded that BED is a unique eating disorder and distinct from normalcy.

BED is distinct from AN and BN in terms of demographic profile: later age of onset (Hudson et al., 2007), lower female to male ratio (Grucza et al., 2007; Hudson et al., 2007), more ethnic minorities represented (Striegel-Moore, Franko, Thompson, Barton, & Schreurs, 2005b). It also differs in terms of risk factors (e.g. less influence of previous dieting history (Manwaring et al., 2006)) and of the association with obesity (Wilfley, Bishop, Wilson, & Agras, 2007; Reas & Grilo, 2007).

Nevertheless BED shares many similarities with AN and BN, which make it distinguishable from non-eating disorders (Hudson et al., 2007; Wilfley et al., 2007; Striegel-Moore & Franko, 2008). For example several recent studies found support for the distinctiveness of overweight persons with BED versus (overweight) persons who do not binge eat. Persons with BED reported higher levels of psychopathology and more other comorbid psychiatric disorders than controls without BED (Grilo et al., 2008; Ramacciotti et al., 2008; Grucza et al., 2007; Hudson et al., 2007; Javaras et al., 2008b) . Several recent studies (Hrabosky, Masheb, White, & Grilo, 2007; Mond, Hay, Rodgers, & Owen, 2007; Grilo et al., 2008; Ramacciotti et al., 2008) demonstrated that individuals with BED have more concerns about their weight and figure than weight-matched controls. Furthermore, several studies found that in a laboratory setting BED participants had a higher caloric intake than non-BED obese women (Raymond, Bartholome, Lee, Peterson, & Raatz, 2007; Galanti, Gluck, & Geliebter, 2007; Sysko, Zimmerli, Kissileff, Devlin, & Walsh, 2007). Women with BED also reported a greater desire to eat, higher anticipated consumption of food in the near future, a higher excitement about eating and a greater desire for a meal or dessert than weight-matched controls (Latner, Rosewall, & Chisholm, 2009).

A few taxonomic and latent class analyses indicate that BED represents a discrete category distinct from other clinical eating disorder categories, consistent with the conclusion in Chapter 3. A recent study by Striegel-Moore et al. (2005b) provided additional support for the distinction between BN and BED. They identified a subtype that is consistent with the core features of BED. Fairburn and Cooper (2007), note that the purpose of clinical classificatory schemes such as the DSM is to aid clinical work and to provide guidance to practitioners regarding differences in treatment response and prognosis. None of the three existing diagnoses (AN, BN and EDNOS) have been validated in this regard. Furthermore, many cases of AN evolve into BN or EDNOS and many cases of BN evolve into EDNOS and vice versa, although to a lesser extent. Thus Fairburn, Cooper and colleagues (2003; 2007) suggested a more transdiagnostic approach to theory and treatment. However, they also state

that BED might be a possible exception because BED has a different course and prognosis from BN (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000).

Diagnostic criteria: Although there seems to be a lot of evidence to suggest that BED represents a distinct eating disorder category, the criteria as currently defined in the DSM-IV (American Psychiatric Association, 1994) would benefit from a major revision. Chapter 2 discussed how to establish objective binge eating episodes. The criteria with regard to the amount of food consumed and loss of control should be defined separately and should be more concisely specified. Sometimes it is difficult to decide what distinguishes a truly large portion size from normal behaviour, overindulgence, or circumstances (e.g. holidays). The Eating Disorder Examination (EDE) (Cooper & Fairburn, 1987; Fairburn & Wilson, 1993) offers a practical solution for this issue (Grilo, Masheb, Lozano-Blanco, & Barry, 2004). The EDE scheme for classifying episodes of overeating (Fairburn et al., 1993) distinguishes between objective and subjective bulimic episodes and between objective and subjective overeating (see Table 2 Chapter 2). The interviewer judges whether the amount eaten should be considered relatively large. The judgment does not require the patient's concurrence. The interviewer always takes into account what would be the usual amount eaten under the circumstances. If the interviewer is in doubt, the amount should not be classified as large. Many researchers use the EDE or the adapted questionnaire (EDE-Questionnaire) which could be considered the golden standard to assess and diagnose eating disorders (Bulik, Brownley, & Shapiro, 2007; Latner & Clyne, 2008).

Also, future DSM criteria for loss of control should be objectified more clearly. Again the EDE offers an adequate solution. Patients must experience a sense of loss of control at the time of eating. Patient use of language such as "eating faster than usual,' 'driven to eat,' 'resisting eating seems impossible' should be rated as loss of control. Again, if the interviewer is in doubt, loss of control should be rated as absent. More recent studies that have examined the key defining components of binge eating episodes have often pointed to the loss of control as a criterion of primary importance (Latner et al., 2008; Keel, Mayer, & Harnden-Fischer, 2001; Niego, Pratt, & Agras, 1997; Colles, Dixon, & O'Brien, 2008; Telch, Pratt, & Niego, 1998; Goldschmidt et al., 2008).

In Chapter 3 we concluded that it is not clear what is meant by the term distress (criterion C of the DSM-IV research-criteria) as currently described. Distress may refer to an emotional state with regard to binge eating. Distress with regard to eating during and after the binge-eating episode should be clearly distinguished from the distress that is caused by (possible) weight gain. This may differentiate true binge eating from normal gluttony. Tanofsky-Kraff and Yanovski (2004) also suggested that eating behaviours that are non-normative should be classified as disordered eating only if they cause distress or impairment. Eating alone because one is embarrassed by how much one is eating seems to be a good marker for distress. Furthermore, distress may also describe impairment in social or occupational functioning due to binge eating. The DSM guidelines state that impairment in

functioning is a crucial element in the definition of any mental disorder. Combining these two definitions suggests that distress over eating has to lead to impairment in functioning.

We further concluded that counting the number of binge eating episodes is better than counting the number of binge days. Counting the number of binge days allows binges to last an entire day (in theory). In a population characterized by a high percentage of compulsive overeaters (without loss of control), this may complicate the separation of the diagnoses of binge eating episodes and compulsive overeating. Again in this regard 'loss of control' also seems to be an important criterion.

The criterion regarding the frequency of binge eating is questioned in several studies. Patients with BED who binged two times a week did not differ from patients who only binged one day a week with respect to BMI, EDE, or psychological distress (Striegel-Moore et al., 2000). Furthermore, there is some debate about the six month duration criterion of the binge eating patterns in BED (Striegel-Moore et al., 2008). BN has a duration criterion for binge eating of three months. A minimum duration of six months was stipulated in order to ensure that transitory episodes of binge eating are not diagnosed as BED. At first, the general opinion was that BED had a natural course that had a tendency to remit. However, more recent research suggests that BED seems to be a stable and chronic disorder rather than a transient or non-specific pattern of abnormal eating (Pope et al., 2006). The current BED duration criterion should be brought into line with that of AN and BN, that is three months (Hudson et al., 2007).

Several studies support our proposal to add psychological criteria to the existing behavioural diagnostic criteria for BED (Chapter 3). Various studies have argued for the inclusion of overconcern with body weight and body shape in self-evaluation (Eldredge & Agras, 1996; Wilson, Nonas, & Rosenblum, 1993; Wilfley, Schwartz, Spurrell, & Fairburn, 2000; Grilo et al., 2008). More recently Mond et al. (2007) investigated participants with BED with and without extreme weight or shape concerns recruited from a large female community sample. Participants who reported extreme overconcern appeared to have significantly higher levels of eating disorder psychopathology, more functional impairment, and greater likelihood of seeking treatment. Similar conclusions have been drawn by Hrabosky et al. (2007) and Grilo et al. (2008). They state that overvaluation does not simply reflect concern commensurate with being overweight but is strongly associated with eating-related psychopathology and psychological functioning and thus warrants consideration as a diagnostic feature for BED. Grilo et al. (2008) also state in their conclusion that their findings provide support for the BED research construct. Over-evaluation of weight or shape might be seen as a core diagnostic feature of all eating disorders (Fairburn et al., 2003), including BED.

Epidemiology - Only three additional studies have been executed in the last five years to evaluate the prevalence of BED. No incidence studies were published. Two studies made use of a clinical interview. Favaro et al. (2003) found a lifetime prevalence of 0.6% in young women from a general Italian population (N=934; 18 to 25 years). Hudson et al. (2007) found

a higher lifetime prevalence of 2.8% (3.5% in women and 2.0% in men) in a sample of 2980 respondents from a general U.S. population. The relatively low prevalence found in the study of Favaro et al. (2003) might be due to a later age of onset of BED compared to AN and BN. Hudson et al. (2007) for example found a mean age of onset of 25.4 year (sd 1.2) in BED. Moreover, a recent study of the prevalence of BED in a community sample of 917 persons using self-report questionnaires was published (Grucza, Przybeck, & Cloninger, 2007). Results showed a current prevalence of 6.6 % (6.8% in women and 6.4% in men). Nearly 70% of those screening positive for BED had a BMI over 30. However, the use of self-report instruments may lead to overestimation of prevalence rates. Thus, the conclusion in Chapter 2 that prevalence rates for BED range between 1 and 3% in the general population may still be accurate.

Aetiology- Research on the aetiology of BED has progressed in recent years. In a study by Pike et al. (2006), women with BED appeared to have experienced significantly more stressful life events than a matched non-psychiatric control group and a matched psychiatric group in the year preceding onset of their eating disturbance. In the same sample, Striegel-Moore et al. (2005a) found that certain risk factors were especially common among women with BED rather than being associated with psychiatric status in general, such as a higher rate of childhood obesity, family overeating, or binge eating. Individuals with BED as well as their first-degree relatives reported significantly higher lifetime rates of depressive disorders and anxiety disorders compared to individuals without BED (Lilenfeld, Ringham, Kalarchian, & Marcus, 2008). Also, BED was more common in first-degree relatives of individuals with BED. BED aggregated strongly in families independent of obesity (Hudson et al., 2006; Javaras et al., 2008a). In a twin study, binge eating in the absence of compensatory behaviours appeared to be moderately heritable (41% of the variance was explained by genetic factors) (Reichborn-Kjennerud, Harris, Tambs, & Bulik, 2004).

As mentioned earlier, a large proportion of individuals with BED started binge eating prior to the onset of dieting (35-54%) (Chapter 2). A recent study found an incidence of 81% binging first compared to 19% diet first (Manwaring et al., 2006). In cognitive behavioural aetiological models, dietary restraint is implicated as an important precursor to binge eating. Most individuals with BN start dieting prior to the onset of binge eating (see also Chapter 2). Some studies report that the binge first group started binge eating at an earlier age, experienced a higher frequency of weight related teasing, and had an earlier onset of overweight and BED diagnoses (Abbott et al., 1998; Grilo & Masheb, 2000). However, Manwaring et al. (2006) found limited support for different risk factors between women who binge first and those who diet first. Raes et al. (2007) indicated that early overweight status might have important implications for the later development of disordered eating (dieting and binge eating) in BED. Summarizing the above, in BED the sequence of the onset of overweight, dieting, and binge eating does not seem to be as clear-cut as in BN. The role of dietary restraint in the aetiology and maintenance of BED remains unclear.

Another model which tries to explain the aetiology 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). Stice (1994; 2001) 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. Two studies found evidence for both a dietary subtype and a dietary-depressive subtype in patients with BED (Stice et al., 2001; Grilo, Masheb, & Wilson, 2001)(see also Chapter 1). In both of our experimental studies (Chapter 6 and 7) we also found evidence for the fact that in individuals with BED depressive symptoms and caloric intake are positively related.

Course – Recently only one study reported data on the long-term course of BED (N=68) and BN (N=196) over a 12-year period (Fichter, Quadflieg, & Hedlund, 2008; Fichter & Quadflieg, 2007). Patients, who were admitted for inpatient treatment, were assessed at hospital intake, at 2-3 year follow-up, at 6-year follow-up (Fichter, Quadflieg, & Gnutzmann, 1998) and at 12-year follow-up. The majority of the patients with BED (67%) had no eating disorder at the12 year follow-up. BED patients never crossed over to AN, six patients (9.4%) crossed over to BN, purging type. The longitudinal course of BED and BN seemed nearly identical with similar percentages of remission and relapse. Eighty-five percent of the patients with BED and 83% of the patients with BN received additional treatment at least once during the 12 years follow-up period. The diagnosis of BED (when compared to AN) is relatively unstable over time, which might be due to recovery, relapse, and diagnostic crossover. It is possible that in BED the course of illness is shorter. For example, the average duration of a depressive episode is approximately 6 months. Perhaps individuals will only tolerate this binge eating problems for a certain period before subjective distress motivates them to seek treatment or discontinue these behaviours on their own (Stice & Spoor, 2007). The long-term outcome of AN was much worse than BED (Fichter et al., 2007). However, in the study of Pope et al. (2006), patients with BED reported a mean lifetime duration of illness of 14.4 years. Most individuals reported a single continuous lifetime episode as opposed to multiple episodes. They suggested that BED is a stable and chronic disorder. Differences in design and study populations might explain these different conclusions about the stability of BED. Fichter et al. (2007; 2008) followed a clinical population in time whereas Pope et al. (2006) recruited individuals with BED from a community sample: the first was a prospective study whereas the latter a retrospective study.

Chapter 4 described a cross-sectional study, which compared maladaptive core beliefs in patients with ANR, ANB/P, BN, and BED and a group of healthy controls. Maladaptive core beliefs represent the deepest level of cognition (Young, 1999). These core beliefs reflect a person's unconditional negative beliefs and feelings in relation to the environment. They refer to stable and enduring themes that develop during childhood. During childhood a belief is a

means for the child to comprehend and manage the environment. Core beliefs are a priori truths that are implicit and taken for granted and are central to the organization of personality. Patients with an eating disorder (AN (both subtypes), BN, and BED) had significantly unhealthier core beliefs than healthy controls on all the four factors of maladaptive core beliefs. Patients with AN (both subtypes) and BN did not differ in the degree of unhealthy core beliefs. With regard to the nature and severity of the core beliefs, patients with BED had an intermediate score between AN and BN on the one hand and the healthy controls on the other hand. However, the core beliefs of BED patients seem to be more similar to the core beliefs of BN or AN patients than to those of healthy subjects. This study showed that the degree of inappropriate compensatory behaviours (such as vomiting, laxatives misuse and fasting) is linked to severity of core beliefs. More recent studies also confirm that patients with an eating disorder have dysfunctional or maladaptive core beliefs that are not directly related to food, weight and shape (Leung & Price, 2007; Lawson, Emanuelli, Sines, & Waller, 2008). Our data showed that cognitions or core beliefs that are not related to eating disorder psychopathology are distorted in patients with an eating disorder and even more in patients who engage in purging behaviours and fasting. These data demonstrate the importance of identifying purging and fasting as significant clinical markers. The frequency of these behaviours can be regarded as an indicator of the severity of the illness (Keel et al., 2004; Tobin, Johnson, & Dennis, 1992). This finding might explain that patients with BED are overall less impaired than patients with AN or BN, because of the absence of inadequate compensatory behaviours like vomiting and laxative abuse. Frequency of binge eating does not seem to be associated with severity of maladaptive core beliefs. The findings in Chapter 4 again confirmed the conclusions made in Chapter 2 and 3 that BED is a distinct diagnostic category.

Before ending our discussion about BED as a distinct diagnostic eating disorder category, it is helpful to consider the arguments against inclusion of a new diagnostic category, as formulated by Pincus and others (1992). Pincus et al. stated that for a new category to be considered for inclusion in the DSM "there must be solid evidence that the diagnosis is useful in predicting prognosis, treatment selection, or outcome".

One argument against new diagnoses is that if they are rare, they may add unnecessary complexity to the already cumbersome system of classification and be irrelevant for clinical use. However, epidemiological studies as reviewed above on BED concluded that BED is not a rare disorder.

A second argument by Pincus et al. against new diagnoses is that new diagnoses are generally proposed by experts and are subsequently used by less expert assessors who may identify more false positives. As we concluded above, the criteria should be made clearer. To determine whether an eating episode can be considered a binge eating episode, the criteria of 1) loss of control, 2) eating without pleasure, and 3) distress about eating are the most important ones. Finally, impairment in functioning must be evident in order to consider BED as a mental disorder according to the DSM (American Psychiatric Association, 1994). If these

suggestions are incorporated, we expect that the identification of false positives will be greatly reduced.

A third argument against inclusion is that 'adding unproven diagnostic categories may confer upon such categories an approval that they do not merit yet.' As reviewed above, many studies have indicated that BED does represent a distinct diagnostic entity. Individuals with BED are clearly distinct from individuals with AN or BN and healthy (obese) individuals. Also, the number of papers in Pubmed with "binge eating disorder" as a keyword has grown significantly over the last five years compared to the five years before (1718 versus 1153 hits).

Pincus' fourth and last argument concerns the definitional overlap across related categories. As discussed above, the presence of obesity in the majority of individuals with BED and the absence of inadequate compensatory behaviours (such as vomiting and laxatives abuse) clearly distinguishes BED from the other eating disorders. Also, BED is unique from AN and BN in terms of demographic profile and possible risk factors.

To conclude, in day-to-day clinical practice BED is a generally accepted diagnostic category and almost all eating disorder centres have developed specific programmes for the treatment of BED. Also, the more recent studies confirm the conclusions from Chapter 2 and 3 that BED is a distinct diagnostic eating disorder category with unique characteristics and is distinguishable from BN and AN.

Summary and discussion part II

Chapter 5 presented the results of a randomized controlled trial in which cognitive behavioural therapy (CBT) was compared to a waiting list control group. The effectiveness and possible mediators and predictors of CBT in patients with BED are examined. As expected, objective binge eating frequency, eating disordered psychopathology, and comorbid psychopathology decreased significantly in the CBT group whereas no reduction was found in the control (WLC) group. Mediator analyses indicated that abstinence from binge eating at post treatment was fully mediated by changes in weight concerns and marginally by changes in concerns about shape and eating, depressive symptoms, and more general psychopathological symptoms. These concerns and the depressive and more general psychopathological symptoms were highly correlated. This worrying, as the such symptoms can be called in general, was also positively correlated with binge eating. It is likely that worrying in general and ruminating about one's weight and appearance are unpleasant experiences. Binge eating may be used as a mean to escape from these negative thoughts and worries and therefore alleviates emotional stress (Heatherton & Baumeister, 1991). Breaking this vicious circle of worrying seems to be necessary for abstaining from binge eating.

Contrary to our expectations, patients with BED benefited equally from treatment regardless of the severity of their comorbid symptoms. Thus CBT might be an effective treatment for the majority of patients. However, we did find that participants with higher levels of the coping style palliative reacting (trying to feel better by eating, smoking, or drinking) at

baseline benefited less from treatment than participants with a less palliative coping style. Participants who still had high levels of this coping style at the end of treatment tended to have less favourable outcomes at one-year follow-up than the participants with lower levels. Further, high levels of expression of anger and annoyance at end of treatment were associated with less improvement than would be expected during the 1-year follow-up. Patients who reacted less angrily and annoyed in their emotions were better capable of maintaining treatment success.

To date about thirty randomized controlled trials (of psychological, pharmacological, dietary treatments, or combinations) in patients with BED have been conducted worldwide. About half of these studies investigated the effectiveness of various psychological treatments. These studies are described in the introduction and in Chapters 2 and 3. CBT (either group or individual) is effective in reducing binge episodes, eating disorder psychopathology, depressive symptoms, and more general psychopathology. However, a recent review (Brownley, Lohr, Bulik, Berkman, & Sedway, 2007) concluded that although CBT received considerable research attention, our understanding of CBT for BED is still limited.

Recently two studies (Hilbert et al., 2007; Masheb & Grilo, 2008) also investigated predictors of treatment outcome in BED. Masheb and Grilo (2008) investigated possible predictors of short-term self-help treatment outcome in BED. They found no main effects for age, age of onset, comorbidity, or pre-treatment clinical characteristics in the prediction of abstinence of binge eating. Their findings suggested that negative affect as measured by the BDI predicted attrition and post treatment levels of negative affect and eating disorder psychopathology. Hilbert et al. (2007) reported that a greater extent of interpersonal problems prior to the start of treatment or at mid-treatment predicted non-response at post-treatment and at one-year follow-up. Greater pre-treatment and mid-treatment concerns about shape and weight among patients with low interpersonal problems were predictive of post-treatment response.

Our results, and those of other studies (Wilfley et al., 2002; Agras, Telch, Arnow, Eldredge, & Marnell, 1997) suggest that participants who are able to stop binge eating tend to lose (a little) body weight. In our study at one-year follow-up, almost 40% of the participants lost 5% or more of their initial body weight, which is associated with significant health benefits (The National Heart, 1998). Although weight loss is limited, probably further weight gain is stopped. This is a major treatment benefit because untreated individuals with BED tend to gain weight over time (Fairburn et al., 2000). In the introduction of this thesis we concluded that caloric restriction does not seem to have the same effect on binge eating in overweight patients with BED as it is assumed to have on normal-weight patients with BN. In BN, restrictive dieting, which is almost viewed as a 'precondition' for the development of binge eating, is central to most aetiological and risk models and plays an important role in treatment approaches (Agras, 1993). However, moderate caloric restriction does not seem to exacerbate binge eating in obese BED patients (Agras et al., 1994; Yanovski et al., 2000; Goodrick,

Poston, Kimball, Reeves, & Foreyt, 1998; Porzelius, Houston, Smith, Arfken, & Fisher, 1995; Raymond, Zwaan, Mithcell, Ackard, & Thuras, 2002; de Zwaan, 2005). Future treatment studies should investigate the effectiveness and mechanisms of change with a combination of psychological and dietary treatments.

The results presented in Chapter 5 indicate that negative thoughts and worries play a role in binge eating. However, as we also stated in the discussion of the treatment study in Chapter 5, our results should to be interpreted with care. Overall frequency of binge eating tends to decrease rapidly during the first weeks of treatment. Thus, at assessment two (T2) after 10 weeks, our data did not allow causal interpretations of the associations between clinical improvement and changes on putative mediators as observed in our study. We do not know for example whether overconcern about their figure decreased first or whether they stopped binge eating first in the first 10 weeks of treatment. By using the approach of Baron and Kenny (1986) we were only able to identify correlates of outcome. Subsequently two experimental studies were conducted to address the question of whether there is a causal relationship between negative affect and binge eating.

Summary and discussion part III

The purpose of the first experimental study (Chapter 6) was to investigate the causal relationship between the regulation of negative emotions, negative mood, and binge eating. The results from the treatment study indicate that worrying was positively correlated with binge eating. It is likely that worrying and ruminating about one's weight and appearance are unpleasant experiences. Binge eating may be used as a means to escape from these negative thoughts and worries and serves to alleviate emotional stress (Heatherton et al., 1991). Muraven and Baumeister (2000) proposed 'that people have a limited quantity of resources available for self-control and that various acts of self-control draw on this limited stock' (p. 247). Controlling one's behaviour, thoughts or emotions requires the expenditure and depletion of a limited inner resource (Baumeister, Tice, Bratslavsky, & Muraven, 1998). When a situation demands two consecutive acts of self-control, performance of the second (unrelated) act is frequently impaired because of energy depletion. We hypothesize that when participants with BED are asked to control and regulate negative emotions, their limited inner source of self-control is depleted afterwards, which means participants with BED have no (or less) control over eating during a subsequent taste task.

Sixty-six women with (full and sub-threshold) BED were shown an upsetting movie and then asked either to suppress their emotions or to react naturally. Subsequently, all women participated in the same taste task. The results did not support our assumption: BED participants who attempted to suppress their emotions while watching an upsetting movie did not consume more calories in a subsequent taste task compared to those who did not suppress their emotions. The initial mood before watching the movie was restored after eating.

Overeating might serve as a means to repair one's negative mood. Sad people often indulge in fattening snacks because they believe that eating repairs their mood, as Tice et al. (2001) concluded in their study. They believe that eating repairs their mood. Continuous attempts to control oneself are strongly guided by one's expectations and cognitions about how self-control works (Martijn, Tenbult, Merckelbach, Dreezens, & de Vries, 2002). They expect that fattening foods will improve their mood. People tend to abandon or violate their normal self-regulatory efforts (control over eating) because they give priority to affect regulation. More severe depressive symptomatology was correlated with a higher caloric intake. Moreover, within the group of BED participants with moderate to severe depressive symptoms, a higher caloric intake was observed for those who were most affected by the negative mood induction. This correlation was not found in the non-depressed BED group. These findings were the reason for conducting the second experimental study.

We hypothesized that individuals with BED *expect* that fattening foods improve their mood. The expectancy learning theory postulates that one forms expectations for the consequences of various behaviours as a result of one's learning history (Smith, Simmons, Flory, Annus, & Hill, 2007). These expectations influence future behavioural choices. The expectancy (that eating helps to alleviate negative affect) might predict increases in binge eating (Stice, 2001). In order to test this hypothesis one must show that expectancy modification produces changes in caloric intake. The aim of the second experimental study (Chapter 7) was to test the relation between expectancies about the effect of food on mood, changes in mood, depressive symptoms, and actual caloric intake in individuals with BED. Participants with BED were randomly assigned to a condition in which expectancies about food and emotion were either confirmed or disconfirmed. Subsequently, participants were assigned to either a negative or a positive mood induction. Finally, they all participated in the same taste task. Caloric intake and changes in mood and expectancies were measured.

Contrary to our expectations, we did not find a main or interaction effect of food-mood-repair-expectancies (confirmative versus disconfirmative condition) and mood induction (negative versus positive) on caloric intake. The participants in all four conditions ate comparable amounts of calories. However, participants in the positive mood induction who had higher baseline expectations that food is pleasurable and useful as a reward consumed more calories than participants with lower expectations.

A positive association was found between depressive symptoms and caloric intake after a negative mood induction. Participants with BED with no-to-mild depressive symptoms ate less after a negative mood induction than individuals with BED with moderate-to-severe depressive symptoms. These results corroborate the findings of our previous experiment (Chapter 6). A remarkable finding was that individuals with no-to-mild depressive symptoms even ate less after a negative mood induction than individuals, regardless of being depressed or not, after a positive mood induction. An explanation for these results might be that negative affect signals that the environment poses a problem, whereas positive affect signals that the

environment is benign. As a result, negative affect cues may motivate detail-oriented, systematic processing, which is usually adaptive in handling problematic situations. When in a negative mood, BED patients with no-to-mild depressive symptoms seem to be more capable of using healthy coping strategies than BED patents with moderate-to-severe depressive symptoms. In contrast, positive affect states signal no particular action requirement, and happy individuals may hence not be motivated to expend cognitive effort unless effort is necessitated by other goals. It might be hypothesized that positive mood signals that all is well, so they can enjoy the moment, and makes them less vigilant to restricting food intake.

In conclusion, both experimental studies found evidence for the fact that in individuals with BED depressive symptoms and caloric intake are positively related. Also, those who experienced large negative mood changes consumed more calories than those who were not or less affected by the negative film fragment. No relationship was found between positive mood changes, depressive symptoms, and caloric intake. No evidence was found for the fact that higher expectations that eating alleviates negative affect leads to a higher caloric intake, especially after a negative mood induction. Possibly, as we also conclude in Chapter 7, our manipulation of expectations was not compelling enough.

Methodological issues

1. One of the strengths of this thesis is the fact that we studied BED using several different research methods: Two literature studies, a cross-sectional comparison study, a randomized controlled trial, and two experimental studies. Combining research from very different angles gives us the opportunity to view BED in a broader perspective.
2. Furthermore, all empirical studies (Chapters, 4, 5, 6 and 7) presented in this thesis were conducted in a population with eating disorders. Testing an intervention or psychological models in a patient population has advantages but also organizational hazards. The results of our studies in populations with severe eating disorders legitimize extending the findings to populations with less severe (binge) eating disorders. We kept the number of exclusion criteria as small as possible, which makes the results more applicable to a general population of individuals with binge eating problems. However, a selection bias might be expected. Patients with BED who seek treatment might be different from individuals who do not. We tried to overcome this problem by also recruiting individuals with binge eating episodes in a non-treatment-seeking population via advertisements in local newspapers and on Internet sites and via calls for participation in primary care practices.
3. Another reason for extending our means of recruiting was that individuals with binge eating disorder were relatively hard to find. The data collection period for each study was therefore long because of a low inflow of participants. Especially in the earlier phase of the treatment study (Chapter 5), this low inflow posed problems with randomization. All patients were treated in groups of least 7 participants. Participants were randomly assigned to either the CBT group or the waiting list control group, and were recruited and included over a limited

time period. For ethical and practical reasons, this period was set at three months. In the first recruitment phase (first cohort) only eight participants could be recruited. After the first assessment at T1, we decided that all eight participants would be non-randomly allocated to the treatment group. Otherwise the treatment group would have been too small with only four participants. However, these participants had equal expectations after assessment at T1 as the participants of phases 2, 3, and 4. All participants believed that they had a 50% chance of being allocated to the treatment group and did not know to which group they would be allocated. Another result of the low inflow is that the total number of participants is relatively small.

4. The low inflow was also a reason to include participants with subthreshold BED. As mentioned before in Chapters 6 and 7, women with a subthreshold BED (an average of one binge eating episode a week) were included in the study because they do not seem to differ significantly from full-syndrome BED (an average of two or more binge eating episodes a week) (Striegel-Moore et al., 2000).

5. Noteworthy was the fact that only three men participated in the treatment study (N=52). Epidemiological studies indicate that the prevalence of binge eating disorder is approximately the same in men as it is in women (2:3)(Hudson et al., 2007; Grucza et al., 2007). Obviously men rarely seek treatment for BED. Therefore, we did not include men with BED in our two experimental studies. Hence the results and conclusions from this thesis might not be generalized to a male BED population.

6. As was mentioned in Chapter 5 and earlier in the discussion, the data of the treatment study did not allow causal interpretations of the associations between clinical improvement and changes on putative mediators as observed. Changes in putative mediators should temporarily precede substantial changes on the outcome measures used. Future studies should measure the putative mediators and outcome measures on a more regular basis (e.g. every session) in order to be able to assess the temporal precedence and causal significance of changes in putative mediators for clinical outcome.

7. In Chapter 6, no effect of suppressing negative emotions on caloric intake was found. This might also be due to a ceiling effect. In the introduction of Chapter 6 we stated that depressed individuals have lower levels of energy and hence less resources available for self-control purposes than non-depressed individuals (Baumeister & Exline, 2000). It could be hypothesised that individuals with BED *in general* have less resources available for self-control purposes.

8. In the last study (Chapter 7), our manipulation of the expectations regarding eating was not long or strong enough to alter the expectancies of individuals with BED that eating alleviates negative mood. In a next study this manipulation could be intensified.

Clinical implications

1. Our treatment study (Chapter 5) showed that Cognitive Behavioural Therapy is an effective treatment for BED. Seventy percent of the patients were abstinent for objective binge

eating at the end of treatment. As expected, objective binge eating frequency, eating disordered psychopathology, and comorbid psychopathology decreased significantly in the CBT group whereas no reduction was found in the control (WLC) group. The treatment manual has been implemented in our clinic and in other eating disorder clinics in the Netherlands. The treatment protocol appears to be a useful and effective approach in clinical practice. Also patients who were not abstinent from binge eating at the end of treatment (approximately 30%) and who needed additional (more intensive) treatment appeared to have benefited. Another argument for starting with this well delimited and structured treatment is that it could not be predicted who would benefit from CBT and who would not.

2. In the treatment study (Chapter 5) only individuals with a full-syndrome BED (an average of two or more objective binge eating episodes a week) participated. However, in clinical practice also individuals with a sub-threshold BED syndrome (an average of one objective binge eating episode a week) and individuals with subjective binge eating episodes (with feelings of loss of control and distress) seem to profit from treatment for BED, suggesting that this treatment is more widely applicable.

3. Prior to the start of the treatment study, eligible participants were fully informed about the treatment and the study by means of information letters, telephone calls (screening), and interviews in person by the researcher of the study. Considerable effort was put into the pre-treatment phase. Total dropout rate at the end of treatment was 4% (2/52). After the end of the study, the treatment manual was implemented in our outpatient clinic. The pre-treatment efforts were minimized (no individual preparation session), which led to considerable dropout. Therapists again started with one individual pre-session before the start of the group sessions to explain the purpose of the treatment. The therapist discusses with the participant what he/she can expect and what is expected from him/her. It is essential to fully inform a patient in advance about what he/she can expect in treatment. A clinician's behaviour can significantly influence a patient's motivation for change (Moyers & Martin, 2006; Cassin, von Ranson, Heng, Brar, & Wojtowicz, 2008) and thus outcome.

4. Patients with BED and a comorbid depression benefited from treatment as much as patients without a comorbid depression. However, during treatment therapist need to be aware of the fact that binge eating is positively associated with depressive symptoms. If the depressive symptoms are severe or persist during treatment for BED, additional treatment for depression should be considered, for example by adding medication. Extra attention should also be paid to patients with BED who experience large negative mood swings. These patients tend to eat larger amounts of food during binge eating episodes and are therefore more prone to become (more) obese.

5. Earlier in this discussion we argue that psychological treatments for BED could (and should) be combined with dietary treatments. The majority of the patients with BED are overweight or obese. One of the reasons for them to seek help is because they want to lose weight. The first aim of BED treatment should be the cessation of binge eating. Treatment

directed at weight loss may be offered to those patients who are able to abstain from binge eating. It might be suggested that overconcern about figure and weight decreases when body weight decreases. Also identification of BED among patients presenting at obesity services is important.

6. The experimental studies indicate that acute mood changes, depressive symptoms, and binge eating are related. Overeating might serve as a means to repair one's negative mood. Furthermore, more severe depressive symptomatology was correlated with a higher caloric intake. In clinical practice it is important to be aware of the possibility that positive mood may also constitute a trigger for overeating in patients with binge eating disorder. In general, patients with BED are aware that negative moods and binge eating are linked. The results of the second experimental study indicates that there might be different reasons and/or processes for eating in negative and positive moods. However, untreated people with BED in general gain weight (Fairburn et al., 2000; Cachelin et al., 1999), so making them more aware of various emotional triggers of eating episodes might prevent weight gain.

Directions for future research

As this thesis concludes, CBT, as well as Interpersonal Psychotherapy (IPT) and Dialectical Behaviour Therapy, are effective psychological treatments for the majority of BED patients. However, little is known about the mechanisms of changes of these various treatment procedures. Which cognitions, emotions, and behaviours need to be changed in order to produce abstinence in binge eating in BED? A treatment study with session-by-session measures could shed more light on this issue. Identification of mediators of change may eventually help both to focus the substance of treatment and to improve the (cost) effectiveness of treatment.

Also in future treatment studies, more attention should be paid to weight loss treatment in combination with treatment directed at abstinence from binge eating and to reducing eating disorders and more general psychopathology in patients with BED. Addressing both problems at the same time might hasten recovery.

Experimental studies more can permit more learning about the causal relations between mood and binge eating. The pervasiveness of the link between emotional distress and binge eating suggests that the question is not *whether* but *how* negative affect produces binge eating. It was hypothesized that individuals with BED overeat in order to repair negative mood. Future experimental studies should include a condition in which participants are not subjected to a taste task to evaluate the natural course after a negative mood induction. If food does indeed repair mood, then no (or less) improvement in mood after a negative mood induction would be expected.

In the last study (Chapter 7) our manipulation was not long or strong enough to alter the expectancies of individuals with BED that eating alleviates negative mood. In a next study this manipulation could be intensified. Also expectations regarding food should be measured in

treatment studies to evaluate whether they mediate abstinence of binge eating. Furthermore it would be interesting to measure implicit attitudes or automatic processes regarding food in patients with BED. We tend to evaluate food relatively automatically, but might this differ between situations (Roefs et al., 2006). Automatic processes do not require cognitive capacity, do not require awareness, and do not involve volition (McNally, 1995). These automatic attitudes might influence the expectations that patients with BED have with regard to food. Finally, in experimental studies should compare the results of patients with BED directly to the results of a healthy control group. Do patients with BED consume more calories after a negative and/or positive mood induction than healthy controls? To study the effects of depressive symptoms on eating, it would be interesting to compare the depressed BED-group with a group of patients with a depressive disorder without BED.

Nederlandse samenvatting

Eetbuien of controle? Onderzoek naar de validiteit, behandeling en onderliggende mechanismen van de Eetbuistoornis.

De eetbuistoornis is een van de eetstoornissen naast anorexia nervosa en boulimia nervosa. Begin jaren negentig pleitte een aantal onderzoekers (Devlin, Walsh, Spitzer, & Hasin, 1992) voor het formuleren van een nieuwe diagnostische groep binnen het spectrum van de eetstoornissen. Deze patiënten hebben eetbuien maar vertonen geen compensatoir gedrag na de eetbui (zoals braken, laxeren of overmatig bewegen), dit in tegenstelling tot patiënten met boulimia nervosa. Deze eetstoornis was niet te classificeren met behulp van het toenmalige handboek voor psychische stoornissen, de DSM-III-R. Deze eetstoornis wordt de eetbuistoornis genoemd ('binge eating disorder'). In 1994 werd de eetbuistoornis voor het eerst opgenomen in de DSM-IV onder "eetstoornissen niet anderszins omschreven" waarin vervolgens verwezen wordt naar de onderzoekscriteria in Appendix B (alleen vermeld in de Amerikaanse versie). De eetbuistoornis wordt gekarakteriseerd door herhaaldelijk optredende eetbuien (minstens twee per week). Een eetbui wordt gekarakteriseerd door zowel: 1) het binnen een beperkte tijd (bijvoorbeeld 2 uur) eten van een hoeveelheid voedsel die beslist groter is dan de meeste mensen in een zelfde periode en onder dezelfde omstandigheden zouden eten als 2) een gevoel van controleverlies over het eten tijdens de eetbui (bijvoorbeeld het gevoel dat men niet kan stoppen met eten of zelf kan bepalen wat en hoeveel men eet). Na afloop van een eetbui walgen mensen van zichzelf of voelen zij zich somber of schuldig. De eetbuistoornis en overgewicht gaan vaak samen.

Hoofdstuk één is de inleiding op dit proefschrift. Er wordt een overzicht gegeven van de onderzochte behandelingen voor de eetbuistoornis. In meer detail wordt er in gegaan op de inhoud van de cognitieve gedragstherapie. Dit is momenteel de behandeling van eerste keuze voor de eetbuistoornis. Vervolgens wordt er een overzicht gegeven van verschillende modellen die ontwikkeld en onderzocht zijn om mechanismen die ten grondslag liggen aan eetbuien te kunnen verklaren: zoals het 'restraint'-model en het 'emotieregulatie' model. Het hoofdstuk besluit met een overzicht van de experimentele onderzoeken die uitgevoerd zijn onder mensen met een eetbuistoornis.

Het vervolg van het proefschrift bestaat uit drie delen. In het eerste deel (*hoofdstuk twee, drie en vier*) wordt de validiteit van de eetbuistoornis onderzocht. Is de eetbuistoornis een aparte eetstoornis die goed te onderscheiden is van anorexia en boulimia nervosa wat betreft kenmerken, behandeling en beloop en is het een volwaardige eetstoornis? Het tweede deel (*hoofdstuk vijf*) geeft de resultaten weer van een gerandomiseerde gecontroleerde studie naar de behandeling van de eetbuistoornis. In het derde en laatste deel ga ik dieper in op mogelijke onderliggende mechanismen van een eetbuistoornis. Dit wordt gedaan aan de hand van de resultaten van twee experimentele studies (*hoofdstuk zes* en *zeven*). Ten slotte bestaat *hoofdstuk*

acht uit een samenvatting en discussie en komen methodologische kwesties, klinische implicaties en aanwijzingen voor vervolgonderzoek aan de orde.

In *hoofdstuk twee* wordt er een overzicht gegeven van de epidemiologie, karakteristieken, etiologie, criteria, beloop en mogelijke behandelingen voor de eetbuistoornis en de effecten daarvan. Een eetbuistoornis komt bij ongeveer 1% tot 3% van de bevolking voor. Een eetbuistoornis lijkt vooral voor te komen bij mensen die hulp zoeken voor overgewicht (1.3-30.1%). Deze eetstoornis komt bijna even vaak bij vrouwen als bij mannen voor (3:2).

In tegenstelling tot mensen met een eetbuistoornis, beginnen de meeste mensen met boulimia nervosa vaak met streng lijnen omdat zij ontevreden zijn over hun gewicht of figuur en ontwikkelen hierdoor later eetbuien. Er zijn aanwijzingen dat veel mensen met de eetbuistoornis eerst eetbuien ontwikkelen en dat zij pas in een later stadium gaan lijnen wanneer zij in gewicht aankomen. Er lijken andere oorzaken een rol te spelen bij het ontstaan van de eetproblemen. Bij veel mensen met een eetbuistoornis is overgewicht of obesitas een bijkomend probleem. Mogelijk zijn de eetbuien de oorzaak van de gewichtstoename. De eetbuistoornis neemt wat betreft de ernst van de psychopathologie een tussen positie in tussen boulimia nervosa en obesitas. Maar het lijkt erop dat mensen met een eetbuistoornis meer lijken op mensen met boulimia nervosa dan op mensen met overgewicht of obesitas wat betreft bijvoorbeeld de ernst van de psychopathologie. Een overzicht van behandelstudies geeft aan dat cognitieve gedragstherapie de behandeling de voorkeur heeft maar dat ook interpersoonlijke psychotherapie, zelfhulpprogramma's en medicatie effectief kunnen zijn. Het eerste doel van de behandeling zou het stoppen van de eetbuien moeten zijn. Behandeling voor gewichtsverlies kan aangeboden worden als mensen vrij zijn van eetbuien.

In *hoofdstuk drie* wordt er ingegaan op de vraag of de eetbuistoornis inderdaad een goed te onderscheiden eetstoornis is en of de eetstoornis op genomen moet worden in een volgende versie van het handboek voor psychische stoornissen, de DSM-V. We beantwoorden deze vraag door vier argumenten van Pincus (1992) tegen opname van een nieuwe psychische stoornis in de DSM te betrekken op de eetbuistoornis. Een nieuwe diagnose kan pas opgenomen worden wanneer er solide bewijs is dat de diagnose nuttig is voor het voorspellen van de prognose, de keuze of het resultaat van de behandeling.

Het eerste argument om een stoornis niet op te nemen in het classificatiesysteem is dat zeldzame diagnostische categorieën het geheel onnodig complex maken. Echter, de eetbuistoornis blijkt geen zeldzame eetstoornis te zijn: 1 tot 3% van de mensen in de algemene bevolking lijdt eraan.

Een tweede argument tegen opname is dat een nieuwe stoornis door experts wordt voorgedragen en dat minder ervaren beoordelaars meer vals positieven identificeren. Een duidelijke operationalisering van de criteria van een eetbuistoornis is hierbij essentieel. De voorgestelde criteria zijn niet altijd eenduidig en zijn voor verbetering vatbaar (zoals

bijvoorbeeld de criteria voor eetbuien). Een praktische oplossing biedt het assessment interview voor eetstoornispsychopathologie (de Eating Disorder Examination) van Fairburn waarin een praktisch schema wordt gepresenteerd. Het schema maakt onderscheid tussen objectieve en subjectieve eetbuien en objectief en subjectief overeten. De interviewer bepaalt of de hoeveelheid die gegeten is ongewoon groot (objectief) is en of er sprake is van controleverlies (sneller eten dan normaal, drang om te eten, weerstand is onmogelijk). Tevens dient beter omschreven te worden dat mensen met een diagnose eetbuistoornis duidelijk leed ondervinden van de eetbuien en dat hun functioneren hierdoor duidelijk beperkt wordt. Tijdens een eetbui wordt er niet genoten van het eten. Door de criteria beter te omschrijven kan men pathologische eetbuien onderscheiden van normale gulzigheid.

Het derde argument tegen opname is dat een stoornis nog niet goed genoeg onderzocht is. Echter, vele studies laten zien dat de eetbuistoornis goed te onderscheiden is van anorexia en boulimia nervosa en van mensen met overgewicht zonder eetstoornis. Tevens laten taxonomische studies zien dat er voldoende bewijs is dat de eetbuistoornis een aparte eetstoornis is. Het aantal studies naar de eetbuistoornis is de afgelopen jaren exponentieel toegenomen.

Het laatste argument van Pincus is dat er een overlap kan zijn met gerelateerde categorieën. Het is inderdaad moeilijk gebleken om de eetbuistoornis en boulimia nervosa, niet purgerende type (compensatoir gedrag door middel van overmatig bewegen en vasten) goed te onderscheiden. Hier moet nog meer onderzoek naar gedaan worden.

Concluderend blijkt dat de eetbuistoornis een goed te onderscheiden eetstoornis is met specifieke kenmerken en een specifiek beloop. In een volgende versie van de DSM dienen de criteria duidelijker te worden geformuleerd. In de klinische praktijk heeft bijna elke instelling een specifieke behandeling voor de eetbuistoornis. Dit duidt er ook op dat er blijkbaar een noodzaak is om mensen met een eetbuistoornis anders te behandelen dan mensen met anorexia en boulimia nervosa.

In *hoofdstuk vier* wordt de vraag onderzocht of de eetbuistoornis goed is te onderscheiden van de overige eetstoornissen en een gezonde controlegroep wat betreft zogenaamde disfunctionele schema's. Disfunctionele schema's of disfunctionele cognities reflecteren de onvoorwaardelijke negatieve overtuigingen en gevoelens in relatie tot de omgeving. Deze schema's verwijzen naar stabiele en langdurige thema's die zijn ontwikkeld in de kindertijd. Tijdens de kindertijd is zo'n overtuiging een manier om de omgeving te begrijpen. Deze schema's staan centraal in iemands persoonlijkheid. In dit hoofdstuk worden de disfunctionele schema's van mensen met anorexia nervosa (restrictieve type), anorexia nervosa (purgerende type), boulimia nervosa, eetbuistoornis en zonder eetstoornis vergeleken. Daarnaast werd er onderzocht of er een relatie was tussen eetstoornissymptomen (zoals braken, laxeermiddelenmisbruik en vasten) en disfunctionele schema's. Voor dit onderzoek hebben we de disfunctionele schema's in vier factoren gegroepeerd:

1. Disconnectie (het gevoel dat men niet aardig gevonden wordt of geliefd is: mensen die hier hoog op scoren verwachten dat er niet aan hun emotionele behoeften wordt voldaan; ze geloven dat er iets niet klopt aan henzelf en proberen daarom niks van zichzelf te laten zien en/of wantrouwen anderen).

2. Beschadigde autonomie (men ziet zichzelf als een mislukkeling, men heeft het gevoel dat men afhankelijk is van anderen of kwetsbaar is en/of men heeft het gevoel niet genoeg zelfcontrole of discipline te hebben om goed te presteren).

3. Beschadigde grenzen (beperkingen in het stellen van interne grenzen, verantwoordelijkheid ten opzichte van anderen of het stellen van lange termijn doelen. Mensen die hier hoog op scoren hebben moeite met het respecteren van de rechten van anderen, met het samenwerken met anderen, met het maken afspraken, met het stellen en behalen van doelen en met het verdragen van onplezierige emoties).

4. Over controle (mensen die hier hoog op scoren leggen (te) veel nadruk op het controleren van gevoelens en keuzes, (te) veel nadruk op prestatie, plicht, perfectionisme en het volgen van regels).

Mensen met anorexia nervosa (beide subtypes) and boulimia nervosa hadden significant meer disfunctionele schema's dan de gezonde controles. Mensen met een eetbuistoornis hadden tussenliggende scores. Er werd geen correlatie gevonden met disfunctionele schema's en frequentie van eetbuien. Echter, frequentie van braken, laxeermiddelen misbruik en vasten was positief gecorreleerd met alle disfunctionele schema's. Dus, hoe meer men braakt, laxeert of vast, des te meer disfunctionele schema's iemand heeft. Mensen met een eetstoornis hebben dus ook disfunctionele schema's die niet direct gerelateerd zijn aan eten, gewicht of figuur. Dit onderzoek toonde aan dat het tijdens de behandeling belangrijk is om aandacht te hebben voor het purgeergedrag en het vasten. Dit gedrag kan voor meer staan dan alleen compensatie van de eetbuien. Bijvoorbeeld men kan braken om zichzelf te straffen of een gevoel te hebben van kracht of controle.

Concluderend kan er op basis van de in deel 1 besproken gegevens gesteld worden dat de eetbuistoornis een aparte en stabiele eetstoornis is. Mensen met een eetbuistoornis zijn duidelijk anders dan mensen met anorexia of boulimia nervosa en gezonde (obese) mensen. Mensen met een eetbuistoornis vertonen bijvoorbeeld geen inadequaat compensatoir gedrag (zoals braken en laxeermiddelen misbruik) iets wat mensen met anorexia en boulimia nervosa wel doen. Ook lijkt de oorzaak het ontstaan van de eetbuien te verschillen. Daarnaast hebben veel mensen met een eetbuistoornis overgewicht, wat ook maakt dat de focus en inhoud van de behandeling anders is dan bij de andere eetstoornissen.

In het *vijfde hoofdstuk* (deel 2) worden de resultaten van een gerandomiseerde gecontroleerde behandelstudie gepresenteerd waarin de effectiviteit van cognitieve gedragstherapie (CGT) wordt vergeleken met een wachtlijstgroep. Daarnaast wordt er onderzocht of er mediatoren en voorspellers van de effectiviteit van CGT zijn. Tweeënvijftig

mensen met een eetbuistoornis werden door het lot toegewezen aan de behandelgroep of de wachtlijstgroep. De behandeling bestond uit 15 zittingen. De eerste helft van de behandeling ging hoofdzakelijk in op het doorbreken en stoppen van de eetbuien. In het tweede deel was er aandacht voor achterliggende problematiek zoals problemen rondom gewicht, assertiviteit, zelfwaardering en stress management. In het derde en laatste deel werd er aandacht besteed aan de terugvalpreventie. Voor, tijdens en direct na de behandeling waren er metingen evenals een jaar na het einde van de behandeling. Zoals verwacht was CGT effectief in het verminderen van de eetbuien, depressieve symptomen, psychologische eetstoornissymptomen en algemene psychopathologie. Het reduceren van het piekeren over gewicht bleek een mediator voor de abstinentie (het geheel stoppen) van de eetbuien aan het einde van de behandeling. Een mediator is in dit geval een bepaald gedrag dat het werkingsmechanisme van de effectiviteit van de behandeling (stoppen met eetbuien) zou kunnen verklaren. Het reduceren van het piekeren over eten en figuur, de depressieve symptomen en algemene psychopathologie bleek gedeeltelijk een mediator te zijn voor de abstinentie van eetbuien aan het einde van de behandeling. De effectiviteit van de behandeling (stoppen met eetbuien) kon niet aan het begin van de behandeling voorspeld worden door de ernst van de eetstoornissymptomen, comorbide psychopathologie of disfunctionele schema's. Ook kon de effectiviteit van behandeling in het jaar na het eind niet voorspeld worden door deze symptomen. Wel bleek dat mensen die aan het einde van de behandeling hogere scores hadden op de copingstijlen 'palliatieve reactie' en 'expressie van emoties' van de Utrechts Copinglijst (UCL) bij de 1-jaar follow-up meting meer eetbuien hadden. Met andere woorden mensen met een eetbuistoornis die zich proberen beter te voelen door te eten, roken of drinken en die hun woede en ergernissen meer uiten aan het einde van de behandeling, zijn minder in staat om het succes van de behandeling te behouden. In het jaar na het einde van de behandeling daalde het gewicht significant. Mensen die in staat waren de eetbuien te stoppen verloren over het algemeen gewicht.

Omdat tijdens de behandeling bleek dat het overgrote deel van de patiënten zeer snel vrij van eetbuien was, kunnen we geen oorzakelijke interpretaties geven aan de associatie tussen abstinentie van eetbuien en de mogelijke mediatoren. Dit was een reden om vervolgens twee experimentele studies uit te voeren.

In het derde en laatste deel van het proefschrift worden de resultaten van twee experimentele studies besproken. In *hoofdstuk zes* werd onderzocht of er a) een oorzakelijke relatie is tussen het onderdrukken van negatieve emoties, negatieve stemming en overeten bij mensen met een eetbuistoornis en b) of deze relatie sterker is bij mensen met een eetbuistoornis die ernstigere depressieve symptomen rapporteren. Diverse onderzoeken toonden aan dat er een positieve relatie is tussen de ernst van de eetbuien en mate van depressie. Het lijkt er op dat de vraag niet is *of* maar *hoe* negatieve emoties eetbuien tot gevolg

hebben. Overeten of eetbuien kunnen een consequentie zijn van het onderdrukken van negatieve emoties.

Om deze neigingen om te overeten te onderdrukken en de negatieve emoties te reguleren moet men zelfcontrole uitoefenen. Baumeister en Muraven (2000) veronderstellen dat zelfcontrole energie kost waarvan men maar een beperkte voorraad heeft. Wanneer men opeenvolgend en binnen korte tijd veel zelfcontrole uitoefent, is deze energie meestal niet toereikend. Wanneer er dus in twee opeenvolgende taken controle moet worden uitgeoefend, zal er minder energie over zijn voor de tweede taak. Hierdoor zal het controleren van die tweede taak veel minder goed gebeuren.

We verwachtten dat mensen met een eetbuistoornis die gevraagd worden hun emoties te reguleren tijdens het kijken naar een emotioneel filmfragment meer zullen eten tijdens een daarop volgende smaaktest dan mensen die geen instructies ontvangen om hun emoties te controleren. Die eerste groep is minder goed in staat om aantrekkelijk voedsel te weerstaan omdat zij zelfcontrole moeten uitoefenen over hun emoties terwijl de tweede groep dat niet doet. Ook werd er verwacht dat mensen met een eetbuistoornis die ernstige depressieve symptomen rapporteren meer gingen eten dan mensen die geen depressieve symptomen rapporteren omdat hun energieniveau minder hoog is waardoor zij minder energie over hebben om zelfcontrole uit te oefenen.

Zesenzestig vrouwen met een eetbuistoornis werden op basis van toeval toegewezen aan een van twee condities. Ze kregen allemaal een somber filmfragment te zien. De ene groep werd gevraagd de negatieve gevoelens te reguleren en onderdrukken tijdens het kijken terwijl de andere groep geen specifieke instructies kreeg. Daarna kregen ze allemaal gedurende 15 minuten dezelfde smaaktest. Beide groepen aten evenveel calorieën tijdens de smaaktest. Tevens bleek er geen relatie te zijn tussen het onderdrukken van negatieve emoties, depressieve symptomen en aantal gegeten calorieën.

De stemming daalde over het algemeen in beide condities en verbeterde weer tijdens de smaaktest. Wellicht is overeten gerelateerd aan onmiddellijke reparatie van een negatieve stemming. Mogelijk eten sombere mensen omdat ze denken of verwachten dat calorierijk voedsel hun stemming zal verbeteren. Daarnaast bleken depressieve symptomen positief gecorreleerd met het aantal gegeten calorieën. Verder bleek dat hoe groter de stemmingsverandering tijdens het filmpje des te hoger de calorie-inname in de matige tot ernstig depressieve groep. In de niet tot milde depressieve groep werd deze relatie niet gevonden. Een mogelijke verklaring is dat depressieve mensen minder in staat zijn om met een acute negatieve stemming om te gaan dan minder depressieve mensen. De depressieve mensen met een eetbuistoornis blijven zich somberder voelen na de stemmingsdip door het filmpje dan de niet depressieve mensen en eten daardoor wellicht meer en/of langer door.

In hoofdstuk zeven werd er onderzocht of 1) veranderingen in verwachtingen en stemming een effect hadden op calorie-inname en of 2) depressieve symptomen dit effect versterkten. Drieënzeventig vrouwen met een eetbuistoornis werden door het lot toegedeeld

aan een conditie waarin hun verwachtingen over voedsel en emoties bevestigd werden of aan een conditie waarin deze verwachtingen ontkracht werden. Dit werd gedaan door middel van een vragenlijst. Men kreeg een aantal stellingen te zien over voeding en men moest dan aangeven of die stelling waar was of niet. Van die vragenlijst waren twee versies. Het doel van de ene versie was het bevestigen van de verwachtingen tussen eten en stemmingsverbetering en het doel van de andere versie was het ontkrachten van deze verwachtingen.

Een voorbeeld was de stelling 'In chocolade zit een stofje waar je blij van wordt'. In de bevestigende versie kreeg men als feedback 'In chocolade zit het stofje fenylethylamine waar je blij van wordt'. In de ontkrachtende versie kreeg men de feedback 'Van chocolade word je helemaal niet blij. Chocolade bevat wel het stofje fenylethylamine maar dat is alleen werkzaam in hele grote hoeveelheden bijvoorbeeld een halve vrachtwagen'. Vervolgens kregen ze of wel een positief filmfragmentje (uit 'When Harry met Sally") of wel een negatief filmfragmentje (uit "The Champ") te zien. Vervolgens kreeg iedereen gedurende 15 minuten dezelfde smaaktest. Er waren dus vier verschillende condities: 1) bevestigende informatie plus negatieve stemmingsinductie, 2) bevestigende informatie plus positieve stemmingsinductie, 3) ontkrachtende informatie plus negatieve stemmingsinductie en 4)ontkrachtende informatie plus positieve stemmingsinductie.

In tegenstelling tot onze verwachtingen aten alle mensen in de vier condities even veel: er was dus geen effect van de manipulatie van de verwachtingen of de stemming. Dit zou verklaard kunnen worden door het feit dat onze manipulatie van verwachtingen niet lang of krachtig genoeg was. Echter, mensen die aan het begin van het onderzoek hogere verwachtingen hadden dat voedsel plezierig en belonend is, aten meer na het positieve filmfragmentje. Niet-depressieve mensen met een eetbuistoornis aten minder na een negatief filmpje dan depressieve mensen. Opvallend was dat de niet-depressieve mensen na een negatief filmpje zelfs minder aten dan depressieve en niet-depressieve mensen na een positief filmpje. Een mogelijke verklaring is dat mensen (in het algemeen) anders reageren wanneer ze in een positieve of negatieve stemming zijn. In een positieve stemming beoordelen mensen informatie anders dan in een negatieve stemming. Een negatieve stemming geeft het signaal dat er een probleem is terwijl een positieve stemming het signaal geeft dat er geen problemen zijn. In een negatieve stemming worden er allerlei processen automatisch opgestart om te proberen het probleem op te lossen terwijl dit in een positieve stemming niet het geval is omdat er geen probleem gesignaleerd is.

Mogelijk zijn niet-depressieve mensen zijn beter in staat om problemen op te lossen op een gezonde manier dan depressieve mensen omdat de eerste groep minder calorieën at dan de laatste groep. Depressieve mensen met eetbuien eten waarschijnlijk meer tijdens de smaaktest omdat ze denken zo hun stemming te kunnen verbeteren. In een positieve stemming zijn mensen met een eetbuistoornis minder alert op hun voedselinname: we vonden geen verschillen tussen de niet-depressieve en depressieve mensen.

Hoofdstuk acht bevat een samenvatting en discussie van het proefschrift zoals hierboven gegeven.

Methodologische overwegingen

Eén van de sterke punten van het proefschrift is dat de eetbuistoornis onderzocht is door middel van verschillende onderzoeksmethoden: literatuuronderzoek, een cross-sectionele studie, een gerandomiseerde gecontroleerde behandelstudie en twee experimentele studies. Ook is al het onderzoek bij patiënten met een eetstoornis uitgevoerd. Dit bleek echter ook moeilijkheden op te leveren. Het was vaak moeilijk om mensen te vinden met een eetbuistoornis waardoor de instroom van deelnemers langzaam verliep. Dit is ook van de reden waarom het aantal deelnemers in de behandelstudie relatief laag is.

We hebben uiteindelijk niet alleen patiënten geworven via Centrum Eetstoornissen Ursula en andere centra voor eetstoornissen maar ook via advertenties in dagbladen en op Internet en via posters in huisartsenpraktijken. Een voordeel hiervan was dat er veel mensen in het onderzoek zijn betrokken die nog niet in behandeling waren. Mensen die hulp zoeken zijn vaak ernstiger ziek dan mensen die dat niet doen. Door ook die laatste groep in het onderzoek te betrekken zijn de resultaten van dit onderzoek waarschijnlijk representatief voor mensen die lijden aan een eetbuistoornis.

Het was opmerkelijk dat er in de behandelstudie maar drie mannen deelnamen terwijl uit literatuuronderzoek blijkt dat de verhouding man: vrouw bij de eetbuistoornis ongeveer 2:3 is. Je zou een hoger percentage mannen verwachten op grond van deze getallen. Blijkbaar zoeken mannen met een eetbuistoornis nauwelijks hulp. Dit is ook de reden waarom we er voor gekozen hebben de twee experimentele studies alleen bij vrouwen uit te voeren. Dit maakt het generaliseren van de resultaten voor mannen echter minder betrouwbaar.

Het feit dat we geen effect van het reguleren en onderdrukken van negatieve emoties in de eerste experimentele studie vonden, kan ook verklaard worden door het feit dat er mogelijk sprake was van een plafondeffect. Wellicht hebben mensen met een eetbuistoornis over het algemeen minder energie om zelfcontrole uit te oefenen. Daarnaast bleek het moeilijk om de verwachtingen die mensen ten aanzien van voedsel en het verbeteren van hun stemmingen, te veranderen tijdens het experiment. Dit zou verklaard kunnen worden door het feit dat mensen deze verwachtingen al jaren hebben en dat die verwachtingen moeilijk te veranderen zijn op korte termijn.

Enkele klinische implicaties

Cognitieve gedragstherapie is een effectieve therapie voor mensen met een eetbuistoornis. Ook mensen met (subjectieve) eetbuien die niet volledig voldoen aan de criteria van de DSM-IV kunnen profiteren van de behandeling.

Tijdens het onderzoek werden alle deelnemers individueel uitgebreid geïnformeerd over de behandeling. Na afloop van het onderzoek is de behandeling een onderdeel geworden

van het reguliere behandelaanbod. De uitgebreide informatie werd niet meer verstrekt. Veel mensen bleken uit te vallen tijdens de behandeling. Het is belangrijk gebleken om iedereen voor de behandeling goed en volledig te informeren over wat ze kunnen verwachten in de behandeling en wat er van hen verwacht wordt. Het resultaat van de behandeling wordt beinvloed in positieve zin door juiste verwachtingen vooraf. Daarnaast is het belangrijk om alert te zijn op eventuele bijkomende depressieve symptomen. De eetbuien kunnen verergeren door ernstige depressieve symptomen. Wanneer de depressieve symptomen tijdens de behandeling blijven bestaan dan zou men eventueel een medicamenteuze behandeling of psychologische behandeling die specifiek gericht is op de depressieve symptomen kunnen overwegen.

Veel mensen met een eetbuistoornis hebben overgewicht. Zij hebben naast het stoppen van de eetbuien vaak ook (terecht) een sterke wens om gewicht te verliezen. In de behandeling van mensen met een eetbuistoornis zou hier meer aandacht voor moeten zijn.

Tijdens de behandeling ligt vaak de nadruk op de relatie tussen negatieve emoties en eetbuien, echter uit het experimentele onderzoek blijkt dat positieve emoties net als negatieve emoties uitlokkers kunnen zijn om te gaan eten. Wanneer men in een positieve stemming is, is men echter minder alert op de voedselinname.

Verder onderzoek

In de toekomst zou het interessant zijn om de resultaten van de experimentele studies te repliceren in een gezonde controlegroep of in een groep mensen met een klinische depressie. Zo zou er onderzocht kunnen worden of mensen met een eetbuistoornis inderdaad meer eten tijdens een smaaktest in vergelijking met gezonde mensen. Daarnaast zou een vergelijking met mensen met een klinische depressie interessant zijn omdat in dat geval puur de invloed van depressieve symptomen op het eetgedrag onderzocht kan worden.

Daarnaast zou er meer onderzoek moeten worden gedaan naar het effect van gewichtsvermindering bij mensen met een eetbuistoornis. Het gelijktijdig aanpakken van zowel de eetbuien als het overgewicht zou het herstel bij mensen met een eetbuistoornis kunnen versnellen. Ten slotte is het interessant om meer onderzoek te verrichten naar het effect van (impliciete) verwachtingen die mensen hebben ten aanzien van het effect van voedsel op de stemming maar ook het effect van verwachtingen van de behandeling op het behandelresultaat.

References

Abbott, D. W., de Zwaan, M., Mussell, M. P., Raymond, N. C., Seim, H. C., Crow, S. J., Crosby, R. D., & Mitchell, J. E. (1998). Onset of binge eating and dieting in overweight women: Implications for etiology, associated features and treatment. *Journal of Psychosomatic Research*, 44, 367-374.

Agras, W. S. (1993). Short-term Psychological Treatments for Binge Eating. In C.G.Fairburn & G. T. Wilson (Eds.), *Binge Eating. Nature, Assessment and Treatment* (pp. 270-286). New York: The Guilford Press.

Agras, W. S., Telch, C. F., Arnow, B., Eldredge, K., Detzer, M. J., Henderson, J., & Marnell, M. (1995). Does interpersonal therapy help patients with binge eating disorder who fail to respond to cognitive-behavioral therapy? *Journal of Consulting and Clinical Psychology*, 63, 356-360.

Agras, W. S., Telch, C. F., Arnow, B., Eldredge, K., & Marnell, M. (1997a). One-year follow-up of cognitive-behavioral therapy for obese individuals with binge eating disorder. *Journal of Consulting and Clinical Psychology*, 65, 343-347.

Agras, W. S., Telch, C. F., Arnow, B., Eldredge, K., & Marnell, M. (1997b). One-year follow-up of cognitive-behavioral therapy for obese individuals with binge eating disorder. *Journal of Consulting and Clinical Psychology*, 65, 343-347.

Agras, W. S. & Telch, C. F. (1998). The effects of caloric deprivation and negative affect on binge eating in obese binge-eating disordered women. *Behavior Therapy*, 29, 491-503.

Agras, W. S., Telch, C. F., Arnow, B., Eldredge, K., Wilfley, D. E., Raeburn, S. D., Henderson, J., & Marnell, M. (1994a). Weight loss, cognitive-behavioral, and desipramine treatments in binge eating disorder: An additive design. *Behavior Therapy*, 25, 225-238.

Agras, W. S., Telch, C. F., Arnow, B., Eldredge, K., Wilfley, D. E., Raeburn, S. D., Henderson, J., & Marnell, M. (1994b). Weight loss, cognitive-behavioral, and desipramine treatments in binge eating disorder: An additive design. *Behavior Therapy*, 25, 225-238.

American Psychiatric Association (1952). *Diagnostic and statistical manual of mental disorders.* Washington DC: American Psychiatric Association.

American Psychiatric Association (1968). *Diagnostic and statistical manual of mental disorders, second edition (DSM-II).* Washington DC: American Psychiatric Association.

American Psychiatric Association (1980). *Diagnostic and statistical manual of mental disorders, third edition (DSM-III).* Washington DC: American Psychiatric Association.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders, fourth edition (DSM-IV).* Washington DC.: American Psychiatric Association.

Antony, M. M., Johnson, W. G., Carr-Nangle, R. E., & Abel, J. L. (1994). Psychopathology correlates of binge eating and binge eating disorder. *Comprehensive Psychiatry*, 35, 386-392.

Appolinario, J. C., Bacaltchuk, J., Sichieri, R., Claudino, A. M., Godoy-Matos, A., Morgan, C., Zanella, M. T., & Coutinho, W. (2003). A randomized, double-blind, placebo-

controlled study of sibutramine in the treatment of binge-eating disorder. *Archives of General Psychiatry*, 60, 1109-1116.

Ardovini, C., Caputo, G., Todisco, P., & Dalle Grave R. (1999). Binge eating and restraint model: Psychometric analysis in binge eating disorder and normal weight bulimia. *European Eating Disorders Review* , 7, 293-299.

Arnold, L. M., McElroy, S. L., Hudson, J. I., Welge, J. A., Bennett, A. J., & Keck, P. E. (2002). A placebo-controlled, randomized trial of fluoxetine in the treatment of binge-eating disorder. *Journal of Clinical Psychiatry*, 63, 1028-1033.

Arrindell, W. A. & Ettema, J. H. M. (1986). *SCL-90. Handleiding bij een multidimensionele psychopathologie-indicator.* Lisse: Swets & Zeitlinger.

Bargh, J. & Chartrand, T. (2000). The mind in the middle: A practical guide to priming and automaticity research. In H.Reis & C. Judd (Eds.), *Handbook of research methods in social and personality psychology* (pp. 253-285). Cambridge, UK: Cambridge University Press.

Baron, R. M. & Kenny, D. A. (1986). The moderator mediator variable distinction in social psychological-research - Conceptual, strategic, and statistical considerations. *Journal Of Personality And Social Psychology*, 51, 1173-1182.

Basdevant, A., Pouillon, M., Lahlou, N., Le Barzic, M., Brillant, M., & Guy-Grand, B. (1995). Prevalence of binge eating disorder in different populations of French women. *International Journal of Eating Disorders*, 18, 309-315.

Baumeister, R. F., Tice, D. M., Bratslavsky, E., & Muraven, M. (1998). Ego depletion: is the active self a limited resource? *Journal Of Personality And Social Psychology*, 74, 1252-1265.

Baumeister, R. F. & Exline, J. J. (2000). Self-control, morality, and human strength. *Journal of Social and Clinical Psychology*, 19, 29-42.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory - 25 years of evaluation. *Clinical Psychology Review*, 8, 77-100.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders.* Oxford, England: International Universities Press.

Beumont, P. J., Kopec-Schrader, E., & Touyz, S. W. (1995). Defining subgroups of dieting disorder patients by means of the Eating Disorders Examination (EDE). *British Journal of Psychiatry*, 166, 472-474.

Blashfield, R. K., Sprock, J., & Fuller, A. K. (1990). Suggested guidelines for including or excluding categories in the DSM-IV. *Comprehensive Psychiatry*, 31, 15-19.

Bloks, H., Spinhoven, P., Callewaert, I., Willemse-Koning, C., & Turksma, A. (2001). Changes in coping styles and recovery after inpatient treatment for severe eating disorders. *European Eating Disorders Review*, 9.

Brownley, K. A., Lohr, K. N., Bulik, C. M., Berkman, N. D., & Sedway, J. A. (2007). Binge eating disorder treatment: a systematic review of randomized controlled trials. *International Journal of Eating Disorders*, 40, 337-348.

Bulik, C. M., Brownley, K. A., & Shapiro, J. R. (2007). Diagnosis and management of binge eating disorder. *World Psychiatry*, 6, 142-148.

Busetto, L., Segato, G., De Luca, M., De Marchi, F., Foletto, M., Vianello, M., Valeri, M., Favretti, F., & Enzi, G. (2005). Weight loss and postoperative complications in morbidly obese patients with binge eating disorder treated by laparoscopic adjustable gastric banding. *Obesity Surgery*, 15, 195-201.

Busing, F. M. T. A., Meijer, E., & Van der Leeden, R. (2005). MLA. Software for multilevel analysis of data with two levels. User's guide for version 4.1. [Computer software]. Leiden, The Netherlands. Department of Psychology, Leiden University.

Butryn, M. L. & Wadden, T. A. (2005). Treatment of overweight in children and adolescents: Does dieting increase the risk of eating disorders? *International Journal of Eating Disorders*, 37, 285-293.

Cachelin, F. M., Striegel-Moore, R. H., Elder, K. A., Pike, K. M., Wilfley, D. E., & Fairburn, C. G. (1999). Natural course of a community sample of women with binge eating disorder. *International Journal of Eating Disorders*, 25, 45-54.

Carter, J. C. & Fairburn, C. G. (1998). Cognitive-behavioral self-help for binge eating disorder: a controlled effectiveness study. *Journal of Consulting and Clinical Psychology*, 66, 616-623.

Cassin, S. E., von Ranson, K. M., Heng, K., Brar, J., & Wojtowicz, A. E. (2008). Adapted motivational interviewing for women with binge eating disorder: a randomized controlled trial. *Psychol Addict Behav* , 22, 417.

Chua, J. L., Touyz, S., & Hill, A. J. (2004). Negative mood-induced overeating in obese binge eaters: an experimental study. *International Journal of Obesity & Related Metabolic Disorders*, 28, 606-610.

Colles, S. L., Dixon, J. B., & O'Brien, P. E. (2008). Loss of control is central to psychological disturbance associated with binge eating disorder. *Obesity*, 16, 608-614.

Cools, J., Schotte, D. E., & McNally, R. J. (1992). Emotional arousal and overeating in restrained eaters. *Journal of Abnormal Psychology*, 101, 348-351.

Cooper, M. (1997). Cognitive theory in Anorexia Nervosa and Bulimia Nervosa: A Review. *Behavioural & Cognitive Psychotherapy* , 25, 113-145.

Cooper, Z. & Fairburn, C. G. (1987). The Eating Disorder Examination: A semi-structured interview for the assessment of the specific psychopathology of eating disorders. *International Journal of Eating Disorders*, 6, 1-8.

Cotrufo, P., Barretta, V., Monteleone, P., & Maj, M. (1998). Full-syndrome, partial-syndrome and subclinical eating disorders: an epidemiological study of female students in Southern Italy. *Acta Psychiatrica Scandinavica*, 98, 112-115.

Crowther, J. H., Shepherd, K. L., Sanftner, J., & Bonifazi, D. Z. (2001). The role of daily hassles in binge eating. *International Journal of Eating Disorders*, 29, 449-454.

de Zwaan, M. (1997). Status and utility of a new diagnostic category: Binge eating disorder. *European Eating Disorders Review*, 5, 226-240.

de Zwaan, M. (2005a). Binge eating, EDNOS and obesity. In C.Norring & R. L. Palmer (Eds.), *EDNOS Eating Disorders Not Otherwise Specified. Scientific and clinical perspectives on the other eating disorders.* (pp. 83-113). Hove, East Sussex: Routlegde.

de Zwaan, M. (2005b). Binge eating, EDNOS and obesity. In C.Norring & R. L. Palmer (Eds.), *EDNOS Eating Disorders Not Otherwise Specified. Scientific and clinical perspectives on the other eating disorders.* (pp. 83-113). Hove, East Sussex: Routlegde.

de Zwaan, M. & Mitchell, J. E. (1992). Binge eating in the obese. *Annals of Medicine*, 24, 303-308.

de Zwaan, M., Mitchell, J. E., Raymond, N. C., & Spitzer, R. L. (1994). Binge eating disorder: Clinical features and treatment of a new diagnosis. *Harvard Review of Psychiatry*, 1, 310-325.

de Zwaan, M., Mitchell, J. E., Seim, H. C., Specker, S. M., Pyle, R. L., Raymond, N. C., & Crosby, R. D. (1994). Eating related and general psychopathology in obese females with binge eating disorder. *International Journal of Eating Disorders*, 15, 43-52.

de Zwaan, M., Mitchell, J. E., Specker, S. M., & Pyle, R. L. (1993). Diagnosing binge eating disorder: Level of agreement between self-report and expert-rating. *International Journal of Eating Disorders*, 14, 289-295.

Deaver, C. M., Meidinger, A., Crosby, R., Miltenberger, R. G., & Smyth, J. (2003). An evaluation of affect and binge eating. *Behavior Modification*, 27, 578-599.

Devlin, M., Walsh, B. T., Spitzer, R. L., & Hasin, D. (1992). Is there another binge eating disorder? Review of the literature on overeating in the absence of bulimia nervosa. *International Journal of Eating Disorders*, 11, 333-340.

Dingemans, A. E., Bruna, M. J., & van Furth, E. F. (2002). Binge eating disorder: A review. *International Journal of Obesity & Related Metabolic Disorders*, 29, 299-307.

Dingemans, A. E., Martijn, C., Jansen, A., & van Furth, E. F. (2009). The effect of suppressing negative emotions on eating behavior in Binge Eating Disorder. *Appetite*, 51, 51-57.

Dingemans, A. E., Spinhoven, P., & Van Furth, E. F. (2007b). Predictors and mediators of treatment outcome in patients with binge eating disorder. *Behaviour Research And Therapy*, 45, 2551-2562.

Dingemans, A. E., Spinhoven, P., & Van Furth, E. F. (2007a). Predictors and mediators of treatment outcome in patients with binge eating disorder. *Behaviour Research And Therapy*, 45, 2551-2562.

Dunn, K. I., Mohr, P. B., Wilson, C. J., & Wittert, G. A. (2008). Beliefs about fast food in Australia: A qualitative analysis. *Appetite*, 51, 331-334.

Eldredge, K. L., Agras, W. S., Arnow, B., Telch, C. F., Bell, S., Castonguay, L., & Marnell, M. (1997). The effects of extending cognitive-behavioral therapy for binge eating disorder among initial treatment nonresponders. *International Journal of Eating Disorders*, 21, 347-352.

Eldredge, K. L. & Agras, W. S. (1997). The relationship between perceived evaluation of weight and treatment outcome among individuals with binge eating disorder. *International Journal of Eating Disorders* , 22, 43-49.

Eldredge, K. L. & Agras, W. S. (1996). Weight and shape overconcern and emotional eating in binge eating disorder. *International Journal of Eating Disorders*, 19, 73-82.

Ellis, A. (2008). *Reason and emotion in psychotherapy*. New York: Stuart.

Fairburn, C. G. (1981). A cognitive behavioural approach to the treatment of bulimia. *Psychological Medicine*, 11, 707-711.

Fairburn, C. G. (1993). *Overcoming Binge Eating*. New York: The Guilford Press.

Fairburn, C. G. & Cooper, Z. (1993a). The Eating Disorder Examination. In C.G.Fairburn & G. T. Wilson (Eds.), *Binge eating: Nature, assessment and treatment* (pp. 317-360). New York: Guilford Press.

Fairburn, C. G. & Cooper, Z. (2007). Thinking afresh about the classification of eating disorders. *International Journal of Eating Disorders*, 40, S107-S110.

Fairburn, C. G., Cooper, Z., Doll, H. A., Norman, P. A., & O'Connor, M. E. (2000). The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry*, 57, 659-665.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A 'transdiagnostic' theory and treatment. *Behaviour Research And Therapy*, 41, 509-528.

Fairburn, C. G., Doll, H. A., Welch, S. L., Hay, P. J., Davies, B. A., & O'Connor, M. E. (1998). Risk factors for binge eating disorder. *Archives of General Psychiatry*, 55, 425-432.

Fairburn, C. G. & Harrison, P. J. (2003). Eating disorders. *Lancet*, 361, 407-416.

Fairburn, C. G., Jones, R., Peveler, R. C., Carr, S. J., Solomon, R. A., O'Connor, M. E., Burton, J., & Hope, R. A. (1991). Three psychological treatments for bulimia nervosa. *Archives of General Psychiatry*, 48, 463-469.

Fairburn, C. G., Welch, S. L., & Hay, P. J. (1993b). The classification of recurrent overeating: The "binge eating disorder" proposal. *International Journal of Eating Disorders*, 13, 155-159.

Fairburn, C. G., Welch, S. L., Norman, P. A., O'Connor, M. E., & Doll, H. A. (1996). Bias and bulimia nervosa: how typical are clinic cases? *American Journal of Psychiatry*, 153, 386-391.

Fairburn, C. G. & Wilson, G. T. (1993c). Binge eating: definition and classification. In C.G.Fairburn & G. T. Wilson (Eds.), *Binge eating: nature, assessment and treatment* (pp. 3-14). New York: The Guilford Press.

Fassino, S., Leombruni, P., Piero, A., Abbate-Daga, G., & Rovera, G. G. (2003). Mood, eating attitudes, and anger in obese women with and without Binge Eating Disorder. *Journal of Psychosomatic Research.*, . 54, 559-566.

Favaro, A., Ferrara, S., & Santonastaso, P. (2003). The spectrum of eating disorders in young women: A prevalence study in a general population sample. *Psychosomatic Medicine*, 65, 701-708.

Fichter, M. M. & Quadflieg, N. (2007). Long-term stability of eating disorder diagnoses. *International Journal of Eating Disorders*, 40, S61-S66.

Fichter, M. M., Quadflieg, N., & Gnutzmann, A. (1998). Binge eating disorder: treatment outcome over a 6-year course. *Journal of Psychosomatic Research*, 44, 385-405.

Fichter, M. M., Quadflieg, N., & Hedlund, S. (2008). Long-term course of binge eating disorder and bulimia nervosa: relevance for nosology and diagnostic criteria. *International Journal of Eating Disorders*, 41, 577.

Fichter, M. M., Quadflieg, N., & Brandl, B. (1993). Recurrent overeating: An empirical comparison of binge eating disorder, bulimia nervosa, and obesity. *International Journal of Eating Disorders*, 14, 1-16.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *Structured Clinical Interview for DSM-IV Axis I Disorders, Patient Edition.* Washington, DC: American Psychiatric Press.

Fitzgibbon, M. L. & Blackman, L. R. (2000). Binge eating disorder and bulimia nervosa: Differences in the quality and quantity of binge eating episodes. *International Journal of Eating Disorders*, 27, 238-243.

Follette, W. C. & Houts, A. C. (1996). Models of scientific progress and the role of theory in taxonomy development: a case study of the DSM. [see comments]. [Review] [59 refs]. *Journal of Consulting & Clinical Psychology*, 64, 1120-1132.

Galanti, K., Gluck, M. E., & Geliebter, A. (2007). Test meal intake in obese binge eaters in relation to impulsivity and compulsivity. *International Journal of Eating Disorders*, 40, 727-732.

Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., Kaplan, A. S., & Woodside, D. B. (1995). Bulimia nervosa in a Canadian community sample: Prevalence and comparison of subgroups. *American Journal of Psychiatry*, 152, 1052-1058.

Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., Kaplan, A. S., & Woodside, D. B. (1996). Purging and nonpurging forms of bulimia nervosa in a community sample. *International Journal of Eating Disorders*, 20, 231-238.

Goldfein, J. A., Walsh, B. T., LaChaussee, J. L., & Kissileff, H. R. (1993). Eating behavior in binge eating disorder. *International Journal of Eating Disorders*, 14, 427-431.

Goldschmidt, A. B., Jones, M., Manwaring, J. L., Luce, K. H., Osborne, M. I., Cunning, D., Taylor, K. L., Doyle, A. C., Wilfley, D. E., & Taylor, C. B. (2008). The clinical significance of loss of control over eating in overweight adolescents. *International Journal of Eating Disorders*, 41, 153-158.

Goodrick, G. K., Poston, W. S. C., Kimball, K. T., Reeves, R. S., & Foreyt, J. (1998a). Nondieting versus dieting treatment for overweight binge-eating women. *Journal of Consulting and Clinical Psychology* , 66, 363-368.

Goodrick, G. K., Poston, W. S. C., Kimball, K. T., Reeves, R. S., & Foreyt, J. (1998b). Nondieting versus dieting treatment for overweight binge-eating women. *Journal of Consulting and Clinical Psychology* , 66, 363-368.

Gorin, A. A., le Grange, D., & Stone, A. A. (2003). Effectiveness of spouse involvement in cognitive behavioral therapy for binge eating disorder. *International Journal of Eating Disorders*, 33.

Götestam, K. G. & Agras, W. S. (1995). General population-based epidemiological study of eating disorders in Norway. *International Journal of Eating Disorders*, 18, 119-126.

Greeno, C. G. & Wing, R. (1996). A double-blind, placebo-controlled trial of the effect of fluoxetine on dietary intake in overweight women with and without binge-eating disorder. *American Journal of Clinical Nutrition*, 64, 267-273.

Greeno, C. G., Wing, R. R., & Shiffman, S. (2000). Binge antecedents in obese women with and without binge eating disorder. *Journal of Consulting and Clinical Psychology*, 68, 95-102.

Grilo, C. M., Hrabosky, J. I., White, M. A., Allison, K. C., Stunkard, A. J., & Masheb, R. M. (2008). Overvaluation of shape and weight in binge eating disorder and overweight controls: refinement of a diagnostic construct. *Journal of Abnormal Psychology*, 117, 414.

Grilo, C. M. & Masheb, R. M. (2000). Onset of dieting vs binge eating in outpatients with binge eating disorder. *International Journal of Obesity & Related Metabolic Disorders*, 24, 404-409.

Grilo, C. M., Masheb, R. M., Lozano-Blanco, C., & Barry, D. T. (2004a). Reliability of the Eating Disorder Examination in Patients with Binge Eating Disorder. *International Journal of Eating Disorders*, . 35, 80-85.

Grilo, C. M., Masheb, R. M., & Salant, S. L. (2005). Cognitive behavioral therapy guided self-help and orlistat for the treatment of binge eating disorder: A randomized, double-blind, placebo-controlled trial. *Biological Psychiatry*, 57, 1193-1201.

Grilo, C. M., Masheb, R. M., & Wilson, G. T. (2001). Subtyping binge eating disorder. *Journal of Consulting & Clinical Psychology*, 69, 1066-1072.

Grilo, C. M., Masheb, R. M., & Wilson, G. T. (2005). Efficacy of cognitive behavioral therapy and fluoxetine for the treatment of binge eating disorder: A randomized double-blind placebo-controlled comparison. *Biological Psychiatry*, 57, 301-309.

Grilo, C. M. & Shiffman, S. (1994). Longitudinal investigation of the abstinence violation effect in binge eaters. *Journal of Consulting and Clinical Psychology*, 62, 611-619.

Grilo, C. M. & Masheb, R. M. (2005). A randomized controlled comparison of guided self-help cognitive behavioral therapy and behavioral weight loss for binge eating disorder. *Behaviour Research And Therapy*, 43, 1509-1525.

Grilo, C. M., Masheb, R. M., Lozano-Blanco, C., & Barry, D. T. (2004b). Reliability of the Eating Disorder Examination in Patients with Binge Eating Disorder. *International Journal of Eating Disorders*, 35, 80-85.

Grissett, N. L. & Fitzgibbon, M. L. (1996). The clinical significance of binge eating in an obese population: support for BED and questions regarding its criteria. *Addictive Behaviors*, 21, 57-66.

Gross, J. J. & Levenson, R. W. (1995). Emotion elicitation using films. *Cognition & Emotion*, 9, 87-108.

Grucza, R. A., Przybeck, T. R., & Cloninger, C. R. (2007). Prevalence and correlates of binge eating disorder in a community sample. *Comprehensive Psychiatry*, 48, 124-131.

Guerrieri, R., Nederkoorn, C., Stankiewicz, K., Alberts, H., Geschwind, N., Martijn, C., & Jansen, A. (2007). The influence of trait and induced state impulsivity on food intake in normal-weight healthy women. *Appetite*, 49, 66-73.

Haiman, C. & Devlin, M. J. (1999). Binge eating before the onset of dieting: A distinct subgroup of bulimia nervosa? *International Journal of Eating Disorders*, 25, 151-157.

Hay, P. J., Fairburn, C. G., & Doll, H. A. (1996). The classification of bulimic eating disorders: A community-based cluster analysis study. *Psychological Medicine*, 26, 801-812.

Hay, P. (1998). The epidemiology of eating disorder behaviors: An Australian community-based survey. *International Journal of Eating Disorders*, 23, 371-382.

Hay, P. & Fairburn, C. (1998). The validity of the DSM-IV scheme for classifying bulimic eating disorders. *International Journal of Eating Disorders*, 23, 7-15.

Heatherton, T. F. & Baumeister, R. F. (1991). Binge eating as escape from selfawareness. *Psychological Bulletin*, 110, 86-108.

Heatherton, T. F., Herman, C. P., & Polivy, J. (1992). Effects of distress on eating - importance of ego-involvement. *Journal Of Personality And Social Psychology*, 62, 801-803.

Henderson, M. & Freeman, C. P. L. (1987). A self-rating scale for bulimia: The BITE. *British Journal of Psychiatry*, 150, 18-24.

Herpertz, S., Albus, C., Lichtblau, K., Kohle, K., Mann, K., & Senf, W. (2000). Relationship of weight and eating disorders in type 2 diabetic patients: A multicenter study. *International Journal of Eating Disorders*, 28, 68-77.

Herpertz, S., Albus, C., Wagener, R., Kocnar, M., Wagner, R., Henning, A., Best, F., Foerster, H., Schleppinghoff, B. S., Thomas, W., Kohle, K., Mann, K., & Senf, W. (1998). Comorbidity of diabetes and eating disorders - Does diabetes control reflect disturbed eating behavior? *Diabetes Care*, 21, 1110-1116.

Hilbert, A., Stein, R. I., Welch, R. R., Saelens, B. E., Mockus, D. S., Matt, G. E., & Wilfley, D. E. (2007). Pretreatment and process predictors of outcome in interpersonal and cognitive behavioral psychotherapy for binge eating disorder. *Journal of Consulting and Clinical Psychology*, 75, 645-651.

Hodges, E. L., Cochrane, C. E., & Brewerton, T. D. (1998). Family characteristics of binge-eating disorder patients. *International Journal of Eating Disorders*, 23, 145-151.

Hohlstein, L. A., Smith, G. T., & Atlas, J. G. (1998). An application of expectancy theory to eating disorders: Development and validation of measures of eating and dieting expectancies. *Psychological Assessment*, 10, 49-58.

Howard, C. E. & Porzelius, L. K. (1999). The role of dieting in binge eating disorder: Etiology and treatment implications. *Clinical Psychology Review*, 19, 25-44.

Hrabosky, J. I., Masheb, R. M., White, M. A., & Grilo, C. M. (2007). Overvaluation of shape and weight in binge eating disorder. *Journal of Consulting and Clinical Psychology*, 75, 175.

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the national comorbidity survey replication. *Biological Psychiatry*, 61, 348-358.

Hudson, J. I., Lalonde, J. K., Berry, J. M., Pindyck, L. J., Bulik, C. M., Crow, S. J., McElroy, S. L., Laird, N. M., Tsuang, M. T., Walsh, B. T., Rosenthal, N. R., & Pope, H. G. (2006). Binge-eating disorder as a distinct familial phenotype in obese individuals. *Archives of General Psychiatry*, 63, 313-319.

Hudson, J. I., McElroy, S. L., Raymond, N. C., Crow, S., Keck, P. E. J., Carter, J., Mitchell, J., Strakowski, S. M., Pope, H. G. J., Coleman, B. S., & Jonas, J. M. (1998). Fluvoxamine in the treatment of binge-eating disorder: A multicenter placebo-controlled, double-blind trial. *American Journal of Psychiatry*, 155, 1756-1762.

Jansen, A. (2000). *Eating Disorder Examination (EDE 12.0). Interview ter vaststelling van de specifieke psychopathologie van eetstoornissen.* Lisse: Swets & Zeitlinger.

Jansen, A., Vandenhout, M., & Griez, E. (1990). Case-histories and shorter communications -clinical and nonclinical binges. *Behaviour Research And Therapy*, 28, 439-444.

Javaras, K. N., Laird, N. M., Born-Kjennerud, T. R., Bulik, C. M., Pope, H. G., & Hudson, J. I. (2008a). Familiality and heritability of binge eating disorder: Results of a case-control family study and a twin study. *International Journal of Eating Disorders*, 41, 174-179.

Javaras, K. N., Pope, H. G., Lalonde, J. K., Roberts, J. L., Nillni, Y. I., Laird, N. M., Bulik, C. M., Crow, S. J., McElroy, S. L., Walsh, B. T., Tsuang, M. T., Rosenthal, N. R., & Hudson, J. I. (2008b). Co-occurrence of binge eating disorder with psychiatric and medical disorders. *Journal of Clinical Psychiatry*, 69, 266-273.

Jeppson, J. E., Richards, P. S., Hardman, R. K., & Granley, H. M. (2003). Binge and purge processes in bulimia nervosa: A qualitative investigation. *Eating-Disorders:-The-Journal-of-Treatment-and-Prevention*, 11, 115.

Johnson, W. G., Boutelle, K. N., Torgrud, L., Davig, J. P., & Turner, S. (2000). What is a binge? The influence of amount, duration, and loss of control criteria on judgments of binge eating. *International Journal of Eating Disorders*, 27, 471-479.

Jones, B. P., Duncan, C. C., Brouwers, P., & Mirsky, A. F. (1991). Cognition in eating disorders. *Journal of Clinical & Experimental Neuropsychology*, 13, 711-728.

Keel, P. K., Mayer, S. A., & Harnden-Fischer, J. H. (2001). Importance of size in defining binge eating episodes in bulimia nervosa. *International Journal of Eating Disorders*, 29, 294-301.

Keel, P. K., Bulik, C. M., Halmi, K. A., Baxter, M. G., Fichter, M., Quadflieg, N., Strober, M., Thornton, L., & Kaplan, A. S. (2004). Application of a latent class analysis to empirically define eating disorder phenotypes. *Archives of General Psychiatry*, 61, 192-200.

Kinzl, J. F., Traweger, C., Trefalt, E., Mangweth, B., & Biebl, W. (1999). Binge eating disorder in males: A population-based investigation. *Eating and Weight Disorders*, 4, 169-174.

Kirkley, B. G., Kolotkin, R. L., Hernandez, J. T., & Gallagher, P. N. (1992). A Comparison of binge-purgers, obese binge eaters and obese nonbinge eaters on the MMPI. *International Journal of Eating Disorders*, 12, 221-228.

Klerman, G. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal Psychotherapy of Depression.* New York, NY: BasicBooks Inc Publishers.

Kraemer, H. C., Agras, W. S., Wilson, G. T., & Fairburn, C. G. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry*, 59, 877-883.

Kuehnel, R. H. & Wadden, T. A. (1994). Binge eating disorder, weight cycling, and psychopathology. *International Journal of Eating Disorders*, 15, 321-329.

LaChaussee, J. L., Kissileff, H. R., Walsh, B. T., & Hadigan, C. M. (1992). The single-item meal as a measure of binge-eating behavior in patients with bulimia nervosa. *Physiology & Behavior*, 38, 563-570.

Laederach-Hofmann, K., Graf, C., Horber, F., Lippuner, K., Lederer, S., Michel, R., & Schneider, M. (1999). Imipramine and diet counseling with psychological support in the treatment of obese binge eaters: A randomized, placebo-controlled double-blind study. *International Journal of Eating Disorders*, 26, 231-244.

Latner, J. D., Rosewall, J. K., & Chisholm, A. M. (2009). Food Volume Effects on Intake and Appetite in Women with Binge-Eating Disorder and Weight-Matched Controls. *International Journal of Eating Disorders*, 42, 68-75.

Latner, J. D. & Clyne, C. (2008). The diagnostic validity of the criteria for binge eating disorder. *International Journal of Eating Disorders*, 41, 1-14.

Lawson, R., Emanuelli, F., Sines, J., & Waller, G. (2008). Emotional awareness and core beliefs among women with eating disorders. *European Eating Disorders Review*, 16, 155.

Lee, C. W., Taylor, G., & Dunn, J. (1999a). Factor structure of the Schema Questionnaire in a large clinical sample. *Cognitive Therapy and Research*, 23, 441-451.

Lee, Y. H., Abbott, D. W., Seim, H. C., Crosby, R. D., Monson, N., Burgard, M., & Mitchell, J. E. (1999b). Eating disorders and psychiatric disorders in the first-degree relatives of obese probands with binge eating disorder and obese non-binge eating disorder controls. *International Journal of Eating Disorders*, 26, 322-332.

Lena, S. M., Fiocco, A. J., & Leyenaar, J. K. (2004). The Role of Cognitive Deficits in the Development of Eating Disorders. *Neuropsychology Review*, 14, 99-113.

Leung, N. & Price, E. (2007). Core beliefs in dieters and eating disordered women. *Eating Behaviors*, 8, 65.

Leung, N., Waller, G., & Thomas, G. (1999). Core beliefs in anorexic and bulimic women. *Journal Of Nervous And Mental Disease*, 187, 736-741.

Lilenfeld, L. R. R., Ringham, R., Kalarchian, M. A., & Marcus, M. D. (2008). A family history study of binge-eating disorder. *Comprehensive Psychiatry*, 49, 247-254.

Macht, M. & Mueller, J. (2007). Immediate effects of chocolate on experimentally induced mood states. *Appetite*, 49, 667-674.

Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite*, 50, 1-11.

Mannucci, E., Bardini, G., Ricca, V., Tesi, F., Piani, F., Vannini, R., & Rotella, C. M. (1997). Eating attitudes and behaviour in patients with Type II diabetes. *Diabetes Nutrition & Metabolism*, 10, 275-281.

Manwaring, J. L., Hilbert, A., Wilfley, D. E., Pike, K. M., Fairburn, C. G., Dohm, F. A., & Striegel-Moore, R. H. (2006). Risk factors and patterns of onset in binge eating disorder. *International Journal of Eating Disorders*, 39, 101-107.

Marcus, M. D., Moulton, M. M., & Greeno, C. G. (1995). Binge eating onset in obese patients with binge eating disorder. *Addictive Behaviors*, 20, 747-755.

Marcus, M. D., Smith, D. E., Santelli, R., & Kaye, W. (1992). Characterization of eating disordered behavior in obese binge eaters. *International Journal of Eating Disorders*, 12, 249-255.

Marcus, M. D., Wing, R., & Lamparski, D. M. (1985). Binge eating and dietary restraint in obese patients. *Addictive Behaviors*, 10, 163-168.

Marcus, M. D., Wing, R. R., Ewing, L., Kern, E., McDermott, M., & Gooding, W. (1990). A double-blind, placebo-controlled trial of fluoxetine plus behavior modification in the treatment of obese binge-eaters and non-binge-eaters. *American Journal of Psychiatry*, 147, 876-881.

Martijn, C., Tenbult, P., Merckelbach, H., Dreezens, E., & de Vries, N. K. (2002). Getting a grip on ourselves: Challenging expectancies about loss of energy after self-control. *Social Cognition*, 20, 441-458.

Masheb, R. M. & Grilo, C. M. (2000). Binge eating disorder: a need for additional diagnostic criteria. *Comprehensive Psychiatry*, 41, 159-162.

Masheb, R. M. & Grilo, C. M. (2001). Accuracy of self-reported weight in patients with binge eating disorder. *International Journal of Eating Disorders*, 29, 29-31.

Masheb, R. M. & Grilo, C. M. (2008). Examination of Predictors and Moderators for Self-Help Treatments of Binge-Eating Disorder. *Journal of Consulting and Clinical Psychology*, 76, 900-904.

McCann, U. D., Rossiter, E. M., King, R. J., & Agras, W. S. (1991). Nonpurging bulimia: A distinct subtype of bulimia nervosa. *International Journal of Eating Disorders*, 10, 679-687.

McElroy, S. L., Casuto, L. S., Nelson, E. B., Lake, K. A., Soutullo, C. A., Keck, P. E., & Hudson, J. I. (2000). Placebo-controlled trial of sertraline in the treatment of binge eating disorder. *American Journal of Psychiatry*, 157, 1004-1006.

McElroy, S. L., Arnold, L. M., Shapira, N. A., Keck, P. E. J., Rosenthal, N. R., Karim, M. R., Kamin, M., & Hudson, J. I. (2003). Topiramate in the treatment of binge eating disorder associated with obesity: A randomized, placebo-controlled trial. *American Journal of Psychiatry*, 160, 255-261.

McElroy, S. L., Rosenthal, N. R., Hudson, J. I., Keck, P. E., Capece, J. A., Shapira, N. A., Arnold, L. M., Wu, S. C., & Fazzio, L. (2004). Topiramate in the long-term treatment of binge-eating disorder associated with obesity. *J Clin Psychiatry*, 65, 1463-1469.

McManus, F. & Waller, G. (1995). A functional analysis of binge-eating. *Clinical Psychology Review*, 15, 845-863.

McNally, R. J. (1995). Automaticity and the anxiety disorders. *Behaviour Research And Therapy*, 33, 747-754.

Meyer, C., Leung, N., Feary, R., & Mann, B. (2001). Core beliefs and bulimic symptomatology in non-eating-disordered women: The mediating role of borderline characteristics. *International Journal of Eating Disorders*, 30, 434-440.

Mitchell, J. E. & Mussell, M. P. (1995). Comorbidity and binge eating disorder. *Addictive Behaviors*, 20, 725-732.

Mitchell, J. E., Mussell, M. P., Peterson, C. B., Crow, S., Wonderlich, S., Crosby, R. D., Davis, T., & Weller, C. L. (1999). Hedonics of binge eating in women with bulimia nervosa and binge eating disorder. *International Journal of Eating Disorders*, 26, 165-170.

Mitchell, J. E., Crow, S., Peterson, C. B., Wonderlich, S., & Crosby, R. D. (1998). Feeding laboratory studies in patients with eating disorders: A review. *International Journal of Eating Disorders*, 24, 115-124.

Mond, J. M., Hay, P. J., Rodgers, B., & Owen, C. (2007). Recurrent binge eating with and without the "undue influence of weight or shape on self-evaluation": Implications for the diagnosis of binge eating disorder. *Behaviour Research And Therapy*, 45, 929-938.

Moyers, T. B. & Martin, T. (2006). Therapist influence on client language during motivational interviewing sessions. *Journal Substance Abuse Treatment*, 30, 245-251.

Munsch, S., Meyer, A. H., Margraf, J., Michael, T., & Biedert, E. (2008). Negative mood induction and unbalanced nutrition style as possible triggers of binges in binge eating disorder (BED). *Eating and Weight Disorders*, 13, 22-29.

Munsch, S., Michael, T., Margraf, J., Schlup, B., Biedert, E., Meyer, A., & Tuch, A. (2007). A randomized comparison of cognitive behavioral therapy and behavioral weight loss

treatment for overweight individuals with binge eating disorder. *International Journal of Eating Disorders*, 40, 102-113.

Muraven, M. & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: does self-control resemble a muscle? *Psychological Bulletin*, 126, 247-259.

Muraven, M., Tice, D. M., & Baumeister, R. F. (1998). Self-control as limited resource: regulatory depletion patterns. *Journal Of Personality And Social Psychology*, 74, 774-789.

Mussell, M. P., Mitchell, J. E., de Zwaan, M., Crosby, R. D., Seim, H. C., & Crow, S. J. (1996a). Clinical characteristics associated with binge eating in obese females: a descriptive study. *International Journal of Obesity & Related Metabolic Disorders*, 20, 324-331.

Mussell, M. P., Mitchell, J. E., de Zwaan, M., Crosby, R. D., Seim, H. C., & Crow, S. J. (1996b). Clinical characteristics associated with binge eating in obese females: a descriptive study. *International Journal of Obesity & Related Metabolic Disorders*, 20, 324-331.

Mussell, M. P., Mitchell, J. E., Fenna, C. J., Crosby, R. D., Miller, J. P., & Hoberman, H. M. (1997). A comparison of onset of binge eating versus dieting in the development of bulimia nervosa. *International Journal of Eating Disorders*, 21, 353-360.

Mussell, M. P., Mitchell, J. E., Weller, C. L., Raymond, N. C., Crow, S. J., & Crosby, R. D. (1995). Onset of binge eating, dieting, obesity, and mood disorders among subjects seeking treatment for binge eating disorder. *International Journal of Eating Disorders*, 17, 395-401.

Nauta, H., Hospers, H., & Jansen, A. (2001). One-year follow-up effects of two obesity treatments on psychological well-being and weight. *British Journal of Health Psychology*, 6, 271-284.

Nauta, H., Hospers, H., Kok, G., & Jansen, A. (2001). A comparison between a cognitive and a behavioral treatment for obese binge eaters and obese non-binge eaters. *Behavior Therapy*, 31, 441-461.

Niego, S. H., Pratt, E. M., & Agras, W. S. (1997). Subjective or objective binge: is the distinction valid? *International Journal of Eating Disorders*, 22, 291-298.

Nielsen, S. & Molbak, A. G. (1998). Eating disorder and type 1 diabetes: Overview and summing-up. *European Eating Disorders Review*, 6, 4-26.

O´Kearney, R., Gertler, R., Conti, J., & Duff, M. (1998). A comparison of purging and nonpurging eating-disordered outpatients: Mediating effects of weight and general psychopathology. *International Journal of Eating Disorders*, 23, 261-266.

Pearlstein, T., Gurney, V., Keller, M. B., Read, J., Spurell, E., Hohlstein, L. A., & Fuchs, C. (2003). A double-blind, placebo-controlled trial of fluvoxamine in binge eating disorder: a high placebo response. *Archives of Women's Mental Health*, 6, 147-151.

Penas-Lledo, E., Fernandez, J. D., & Waller, G. (2004). Association of anger with bulimic and other impulsive behaviours among non-clinical women and men. *European Eating Disorders Review.*, . 12, 392-397.

Peterson, C. B., Mitchell, J. E., Engbloom, S., Nugent, S., Mussell, M. P., Crow, S. J., & Thuras, P. (2001). Self-help versus therapist-led group cognitive-behavioral treatment of binge eating disorder at follow-up. *International Journal of Eating Disorders*, 30, 363-374.

Peterson, C. B., Mitchell, J. E., Engbloom, S., Nugent, S., Mussell, M. P., & Miller, J. P. (1998). Group cognitive-behavioral treatment of binge eating disorder: a comparison of therapist-led versus self-help formats. *International Journal of Eating Disorders*, 24, 125-136.

Peterson, C. B., Thuras, P., Crow, S. J., Mitchell, J. E., & Miller, K. B. (2005). Subtypes of binge eating disorder based on psychiatric history. *International Journal of Eating Disorders*.

Pike, K. M., Wilfley, D., Hilbert, A., Fairburn, C. G., Dohm, F. A., & Striegel-Moore, R. H. (2006). Antecedent life events of binge-eating disorder. *Psychiatry Research*, 142, 19-29.

Pincus, H. A., Frances, A., Davis, W. W., First, M. B., & Widiger, T. A. (1992). DSM-IV and new diagnostic categories: Holding the line on proliferation. *American Journal of Psychiatry*, 149, 112-117.

Polivy, J. (1996). Psychological consequences of food restriction. *Journal of the American Dietetic Association*, 96, 589-592.

Polivy, J. & Herman, C. P. (1985). Dieting and binging. A causal analysis. *American Psychologist*, 40, 193-201.

Pope, H. G., Walsh, T., McElroy, S. L., Bulik, C. M., Lalonde, J. K., Pindyck, L. J., Hudson, J. I., Crow, S. J., & Rosenthal, N. (2006). Binge eating disorder: a stable syndrome. *American Journal of Psychiatry*, 163, 2181-2183.

Porzelius, L. K., Houston, C., Smith, M., Arfken, C., & Fisher, E. (1995a). Comparison of a standard behavioral weight loss treatment and a binge eating weight loss treatment. *Behavior Therapy*, 26, 119-134.

Porzelius, L. K., Houston, C., Smith, M., Arfken, C., & Fisher, E. (1995b). Comparison of a standard behavioral weight loss treatment and a binge eating weight loss treatment. *Behavior Therapy*, 26, 119-134.

Pratt, E. M., Telch, C. F., Labouvie, E. W., Wilson, G. T., & Agras, W. S. (2001). Perfectionism in women with binge eating disorder. *International Journal of Eating Disorders*, 29, 177-186.

Presnell, K., Stice, E., & Tristan, J. (2008). Experimental investigation of the effects of naturalistic dieting on bulimic symptoms: Moderating effects of depressive symptoms. *Appetite*, 50, 91-101.

Ramacciotti, C. E., Coli, E., Bondi, E., Burgalassi, A., Massimetti, G., & Dell'Osso, L. (2008). Shared psychopathology in obese subjects with and without binge-eating disorder. *International Journal of Eating Disorders*, 41, 643.

Ramacciotti, C. E., Coli, E., Passaglia, C., Lacorte, M., Pea, E., & Dell'Osso, L. (2000). Binge eating disorder: prevalence and psychopathological features in a clinical sample of obese people in Italy. *Psychiatry Research*, 94, 131-138.

Raymond, N. C., Bartholome, L. T., Lee, S. S., Peterson, R. E., & Raatz, S. K. (2007). A comparison of energy intake and food selection during laboratory binge eating episodes in obese women with and without a binge eating disorder diagnosis. *International Journal of Eating Disorders* , 40, 67-71.

Raymond, N. C., Mussell, M. P., Mitchell, J. E., & de Zwaan, M. (1995). An age-matched comparison of subjects with binge eating disorder and bulimia nervosa. *International Journal of Eating Disorders*, 18, 135-143.

Raymond, N. C., Zwaan, M. d., Mithcell, J. E., Ackard, D., & Thuras, P. (2002a). Effect of a very low calorie diet on the diagnostic category of individuals with binge eating disorder. *International Journal of Eating Disorders*, 31, 49.

Raymond, N. C., Zwaan, M. d., Mithcell, J. E., Ackard, D., & Thuras, P. (2002b). Effect of a very low calorie diet on the diagnostic category of individuals with binge eating disorder. *International Journal of Eating Disorders*, 31, 49.

Reas, D. L. & Grilo, C. M. (2007). Timing and sequence of the onset of overweight, dieting, and binge eating in overweight patients with binge eating disorder. *International Journal of Eating Disorders*, 40, 165.

Reichborn-Kjennerud, T., Harris, J. R., Tambs, K., & Bulik, C. M. (2004). Genetic and environmental influences on binge eating in the absence of compensatory behaviors: a population-based twin study. *International Journal of Eating Disorders*, 36, 307-314.

Ricca, V., Mannucci, E., Moretti, S., Di Bernardo, M., Zucchi, T., Cabras, P. L., & Rotella, C. M. (2000). Screening for binge eating disorder in obese outpatients. *Comprehensive Psychiatry*, 41, 111-115.

Roefs, A., Wolters, G., van Breukelen, G., Werrij, M. Q., Havermans, R., Quaedackers, L., Nederkoorn, C., & Jansen, A. (2006). The environment influences whether high-fat foods are associated with palatable or with unhealthy. *Behaviour Research And Therapy*, 44, 715-735.

Romano, S. J. & Quinn, L. (1995). Binge eating disorder: Description and proposed treatment. *European Eating Disorders Review*, 3, 67-79.

Rosenvinge, J. H., Borgen, J. S., & Borresen, R. (1999). The prevalence and psychological correlates of anorexia nervosa, bulimia nervosa and binge eating among 15-year-old students: A controlled epidemiological study. *European Eating Disorders Review*, 7, 382-391.

Rossiter, E. M., Agras, W. S., Telch, C. F., & Bruce, B. (1992). The eating patterns of non-purging bulimic subjects. *International Journal of Eating Disorders*, 11, 111-120.

Safer, D. L., Lively, T. J., Telch, C. F., & Agras, W. S. (2002). Predictors of relapse following successful dialectical behavior therapy for binge eating disorder. *International Journal of Eating Disorders*, 32, 155-163.

Santonastaso, P., Ferrara, S., & Favaro, A. (1999). Differences between binge eating disorder and nonpurging bulimia nervosa. *International Journal of Eating Disorders*, 25, 215-218.

Sareen, J., Hassard, T., Menec, V., Stein, M. B., & Campbell, D. W. (2005). The relation between perceived need for mental health treatment, DSM diagnosis, and quality of life: a Canadian population-based survey. *Can J Psychiatry*, 50, 87-94.

Schmidt, N. B., Joiner, T. E., Young, J. E., & Telch, M. J. (1995). The schema questionnaire: investigation of psychometric properties and the hierarchical structure of a measure of maladaptive schemas. *Cognitive Therapy and Research*, 19, 295-321.

Schmidt, U. (1998). The treatment of bulimia nervosa. In H.W.Hoek, J. L. Treasure, & M. A. Katzman (Eds.), *Neurobiology in the treatment of eating disorders* (pp. 331-362). Chichester: John Wiley & Sons Ltd.

Schreurs, P. J. G., van de Willige, G., Tellegen, B., & Brosschot, J. F. (1993). *Herziene handleiding Utrechtse Coping Lijst (UCL).* Lisse: Swets & Zeitlinger.

Schwarz, N. & Clore, G. L. (2003). Mood as Information: 20 Years Later. *Psychological Inquiry*, 14, 296-303.

Silva, I., Pais-Ribeiro, J. L., Cardoso, H., Rocha, G., Monteiro, M., Nogueira, C., Santos, J., & Sergio, A. (2007). Binge eating disorder before and after bariatric surgery: Exploratory study. *Obesity Surgery*, 17, 1027.

Smith, G. T., Simmons, J. R., Flory, K., Annus, A. M., & Hill, K. K. (2007). Thinness and Eating Expectancies Predict Subsequent Binge-Eating and Purging Behavior Among Adolescent Girls. *Journal of Abnormal Psychology*, 116.

Specker, S., Mitchell, J., de Zwaan, M., & Raymond, N. (1994). Psychopathology in subgroups of obese women with and without binge eating disorder. *Comprehensive Psychiatry*, 35, 185-190.

Spitzer, R. L., Devlin, M., Walsh, B. T., Hasin, D., Wing, R., Marcus, M. D., Stunkard, A., Wadden, T., Yanovski, S., Agras, W. S., Mitchell, J., & Nonas, C. (1992). Binge eating disorder: A multiside field trial of the diagnostic criteria. *International Journal of Eating Disorders*, 11, 191-203.

Spitzer, R. L., Devlin, M., Walsh, B. T., Hasin, D., Wing, R., Marcus, M. D., Stunkard, A., Wadden, T., Yanovski, S., Agras, W. S., Mitchell, J., & Nonas, C. (1991). Binge eating disorder: To be or not to be in DSM-IV. *International Journal of Eating Disorders*, 10, 627-629.

Spitzer, R. L., Yanovski, S. Z., Wadden, T., & Wing, R. (1993). Binge eating disorder: Its further validation in a multisite study. *International Journal of Eating Disorders*, 13, 137-153.

Spurrell, E. B., Wilfley, D. E., Tanofsky, M. B., & Brownell, K. D. (1997). Age of onset for binge eating: Are there different pathways to binge eating? *International Journal of Eating Disorders*, 21, 55-65.

Stice, E., Agras, W. S., Telch, C. F., Halmi, K. A., Mitchell, J. E., & Wilson, G. T. (2001a). Subtyping binge eating-disordered women along dieting and negative affect dimensions. *International Journal of Eating Disorders*, 30, 11-27.

Stice, E., Agras, W. S., Telch, C. F., Halmi, K. A., Mitchell, J. E., & Wilson, T. (2001b). Subtyping binge eating-disordered women along dieting and negative affect dimensions. *International Journal of Eating Disorders*, 30, 11-27.

Stice, E., Akutagawa, D., Gaggar, A., & Agras, W. S. (2000). Negative affect moderates the relation between dieting and binge eating. *International Journal of Eating Disorders*, 27, 218-229.

Stice, E. & Spoor, S. T. P. (2007). Stability of eating disorder diagnoses. *International Journal of Eating Disorders*, 40, S79-S82.

Stice, E. (1994). Review of the evidence for a sociocultural model of bulimia nervosa and an exploration of the mechanisms of action. *Clinical Psychology Review*, 14, 633-661.

Stice, E. (2001). A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect. *Journal of Abnormal Psychology*, 110, 124-135.

Striegel-Moore, R. H., Fairburn, C. G., Wilfley, D. E., Pike, K. M., Dohm, F. A., & Kraemer, H. C. (2005a). Toward an understanding of risk factors for binge-eating disorder in black and white women: a community-based case-control study. *Psychological Medicine*, 35, 907-917.

Striegel-Moore, R. H. & Franko, D. L. (2008). Should binge eating disorder be included in the DSM-V? A critical review of the state of the evidence. *Annual Review of Clinical Psychology*, 4, 305-324.

Striegel-Moore, R. H., Franko, D. L., Thompson, K., Barton, B., & Schreurs, P. J. G. (2005b). An empirical study of the typology of bulimia nervosa and its spectrum variants. *Psychol Med*, 35, 1563.

Striegel-Moore, R. H. (1995). Psychological factors in the etiology of binge eating. *Addictive Behaviors*, 20, 713-723.

Striegel-Moore, R. H., Dohm, F. A., Solomon, R. A., Fairburn, C. G., Pike, K. M., & Wilfley, D. E. (2000). Subthreshold binge eating disorder. *International Journal of Eating Disorders*, 27, 270-278.

Striegel-Moore, R. H., Wilson, G. T., Wilfley, D. E., Elder, K. A., & Brownell, K. D. (1998). Binge eating in an obese community sample. *International Journal of Eating Disorders*, 23, 27-37.

Stunkard, A., Berkowitz, R., Tanrikut, C., Reiss, E., & Young, L. (1996). d-Fenfluramine treatment of binge eating disorder. *American Journal of Psychiatry*, 153, 1455-1459.

Stunkard, A. J. & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research*, 29, 71-83.

Sysko, R., Zimmerli, E., Kissileff, H. R., Devlin, M. J., & Walsh, B. T. (2007). Satiety and test meal intake among women with binge eating disorder. *International Journal of Eating Disorders*, 40, 554-561.

Tanofsky-Kraff, M. & Yanovski, S. Z. (2004). Eating disorder or disordered eating? Non-normative eating patterns in obese individuals. *Obesity Research*, 12, 1361-1366.

Telch, C. F., Agras, W. S., & Rossiter, E. M. (1988). Binge eating increases with increasing adiposity. *International Journal of Eating Disorders*, 7, 115-119.

Telch, C. F. & Agras, W. S. (1993). The effects of a Very Low Calorie Diet on binge eating. *Behavior Therapy*, 24, 177-193.

Telch, C. F. & Agras, W. S. (1996a). The effects of short-term food deprivation on caloric intake in eating-disordered subjects. *Appetite*, 26, 211-234.

Telch, C. F., Agras, W. S., & Linehan, M. M. (2002). Dialectical behavior therapy for binge eating disorder. *Journal of Consulting and Clinical Psychology*, 69, 1061-1065.

Telch, C. F. & Agras, W. S. (1994b). Obesity, binge eating and psychopathology: Are they related? *International Journal of Eating Disorders*, 15, 53-61.

Telch, C. F. & Agras, W. S. (1994a). Obesity, binge eating and psychopathology: Are they related? *International Journal of Eating Disorders*, 15, 53-61.

Telch, C. F. & Agras, W. S. (1996b). Do emotional states influence binge eating in the obese? *International Journal of Eating Disorders*, 20, 271-279.

Telch, C. F., Agras, W. S., Rossiter, E. M., Wilfley, D. E., & Kenardy, J. (1990). Group cognitive-behavioral treatment for the nonpurging bulimic: An initial evaluation. *Journal of Consulting and Clinical Psychology*, 58, 629-635.

Telch, C. F., Pratt, E. M., & Niego, S. H. (1998). Obese women with binge eating disorder define the term binge. *International Journal of Eating Disorders*, 24, 313l-317.

Telch, C. F. & Stice, E. (1998). Psychiatric comorbidity in women with binge eating disorder: Prevalence rates from a non-treatment-seeking sample. *Journal of Consulting and Clinical Psychology*, 66, 768-776.

The National Heart, L. a. B. I. (1998). Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. *Journal of the American Dietetic Association*, 98, 1178-1191.

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001a). Emotional distress regulation takes precedence over impulse control: if you feel bad, do it! *Journal Of Personality And Social Psychology*, 80, 53-67.

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001b). Emotional distress regulation takes precedence over impulse control: if you feel bad, do it! *Journal Of Personality And Social Psychology*, 80, 53-67.

Tobin, D. L., Griffing, A., & Griffing, S. (1997). An examination of subtype criteria for bulimia nervosa. *International Journal of Eating Disorders*, 22, 179-186.

Tobin, D. L., Johnson, C. L., & Dennis, A. B. (1992). Divergent forms of purging behavior in bulimia nervosa patients. *International Journal of Eating Disorders*, 11, 17-24.

Van der Does, A. J. (2002). *Beck Depression Inventory - II (BDI-II-NL)*. Lisse: Swets & Zeitlinger.

van Groenestijn, M. A. C., Akkerhuis, G. W., Kupka, R. W., Schneider, N., & Nolen, W. A. (1999). *Gestructureerd klinisch interview voor de vaststelling van DSM-IV as I stoornissen.* Lisse, the Netherlands: Swets & Zeitlinger.

van Hanswijck de Jonge, P. (2002). *Personality characteristics in binge eating disorder: A comparison study with bulimia nervosa and obesity.* PhD Thesis.

van Hanswijck de Jonge, P., van Furth, E. F., Lacey, J. H., & Waller, G. (2003). The prevalence of DSM-IV personality pathology among individuals with bulimia nervosa, binge eating disorder and obesity. *Psychological Medicine*, 33, 1311-1317.

Varnado, P. J., Williamson, D. A., Bentz, B. G., Ryan, D. H., Rhodes, S. K., O'Neil, P. M., & Sebastian, S. B. (1997). Prevalence of binge eating disorder in obese adults seeking weight loss treatment. *Eating and Weight Disorders*, 2, 117-124.

Vohs, K. D. & Heatherton, T. F. (2001). Self-regulatory failure: a resource-depletion approach. *Psychological Science*, 11, 249-254.

Wadden, T., Foster, G., & Letizia, K. A. (1992). Response of obese binge eaters to treatment by behavior therapy combined with very low calorie diet. *Journal of Consulting and Clinical Psychology*, 60, 808-811.

Wadden, T. A., Anderson, D. A., Letchak, R. V., Gladis, M., Foster, G. D., Sarwer, D. B., Phelan, S., Sanderson, R. S., & Berkowitz, R. I. (2004). Dieting and the development of eating disorders in obese women: results of a randomized controlled trial. *American Journal of Clinical Nutrition*, 80, 560-568.

Waller, G. (2003). Schema-level cognitions in patients with binge eating disorder: A case control study. *International Journal of Eating Disorders*, 33, 458-464.

Waller, G., Babbs, M., Milligan, R., Meyer, C., Ohanian, V., & Leung, N. (2003). Anger and core beliefs in the eating disorders. *International Journal of Eating Disorders*, 34, 118-124.

Waller, G., Ohanian, V., Meyer, C., & Osman, S. (2000). Cognitive content among bulimic women: The role of core beliefs. *International Journal of Eating Disorders*, 28, 235-241.

Walters, E. E., Neale, M. C., Eaves, L. J., Heath, A. C., Kessler, R. C., & Kendler, K. S. (1993). Boulimia nervosa: A population-based study of purgers versus nonpurgers. *International Journal of Eating Disorders*, 13, 265-272.

Waters, A., Hill, A., & Waller, G. (2001). Bulimics' responses to food cravings: is binge-eating a product of hunger or emotional state? *Behaviour Research And Therapy*, 39, 877-886.

Webber, E. M. (1994). Psychological characteristics of bingeing and nonbingeing obese women. *Journal of Psychology*, 128, 339-351.

Wegner, D. M., Erber, R., & Zanakos, S. (1994). Ironic processes in the mental control of mood and mood-related thought. *Journal Of Personality And Social Psychology*, 65, 1093-1104.

Wegner, D. M., White, T. L., Schneider, D. J., & Carter, S. R. (1987). Paradoxical effects of thought suppression. *Journal Of Personality And Social Psychology*, 53, 5-13.

Wegner, D. M. (1994). Ironic processes of mental control. *Psychological Review*, 101.

Wenzlaff, R. M. & Wegner, D. M. (2000). Thought suppression. *Annual Review Psychology*, 51, 59-91.

Wilfley, D. E., Agras, W. S., Telch, C. F., Rossiter, E. M., Schneider, J. A., Golomb Cole, A., Sifford, L., & Raeburn, S. D. (1993). Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic individual: A controlled comparison. *Journal of Consulting and Clinical Psychology*, 61, 296-305.

Wilfley, D. E., Bishop, M. E., Wilson, G. T., & Agras, W. S. (2007). Classification of eating disorders: Toward DSM-V. *International Journal of Eating Disorders*, 40, S123-S129.

Wilfley, D. E. & Cohen, L. R. (1997). Psychological treatment of bulimia nervosa and binge eating disorder. *Psychopharmacology Bulletin*, 33, 437-454.

Wilfley, D. E., Frank, M. A., Welch, R., Borman Spurrell, E., & Rounsaville, B. J. (1998). Adapting interpersonal psychotherapy to a group format (IPT-G) for binge eating disorder: Toward a model for adapting empirically supported treatments. *Psychotherapy Research*, 8, 379-391.

Wilfley, D. E., Friedman, M. A., Dounchis, J. Z., Stein, R. I., Welch, R. R., & Ball, S. A. (2000a). Comorbid psychopathology in binge eating disorder: Relation to eating disorder severity at baseline and following treatment. *Journal of Consulting and Clinical Psychology*, 68, 641-649.

Wilfley, D. E., MacKenzie, K. R., Welch, R. R., Ayres, V. E., & Weissman, M. M. (2000). *Interpersonal psychotherapy for group*. New York: Basic Books.

Wilfley, D. E., Pike, K. M., Dohm, F. A., Striegel-Moore, R. H., & Fairburn, C. G. (2001). Bias in binge eating disorder: How representative are recruited clinic samples? *Journal of Consulting and Clinical Psychology*, 69, 383-388.

Wilfley, D. E., Schwartz, M. B., Spurrell, E. B., & Fairburn, C. G. (2000b). Using the eating disorder examination to identify the specific psychopathology of binge eating disorder. *International Journal of Eating Disorders*, 27, 259-269.

Wilfley, D. E., Welch, R. R., Stein, R. I., Spurrell, E. B., Cohen, L. R., Saelens, B. E., Dounchis, J. Z., Frank, M. A., Wiseman, C. V., & Matt, G. E. (2002a). A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Archives of General Psychiatry*, 59, 713-721.

Wilfley, D. E., Welch, R. R., Stein, R. I., Spurrell, E. B., Cohen, L. R., Saelens, B. E., Dounchis, J. Z., Frank, M. A., Wiseman, C. V., & Matt, G. E. (2002b). A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Archives of General Psychiatry*, 59, 713-721.

Williamson, D. A., Gleaves, D. H., & Savin, S. S. (1992). Empirical classification of eating disorder not otherwise specified: Support for DSM-IV changes. *Journal of Psychopathology and Behavioral Assessment*, 14, 201-216.

Williamson, D. A., Womble, L. G., Smeets, M. A. M., Netemeyer, R. G., Thaw, J. M., Kutlesic, V., & Gleaves, D. H. (2002). Latent structure of eating disorder symptoms: A factor analytic and taxometric investigation. *American Journal of Psychiatry*, 159, 412-418.

Wilson, G. T., Nonas, C., & Rosenblum, G. D. (1993). Assessment of binge eating in obese patients. *International Journal of Eating Disorders*, 13, 25-33.

Wilson, G. T., Walsh, B. T., Kraemer, H., Fairburn, C. C., & Agras, W. S. (2002). Cognitive-behavioral therapy for bulimia nervosa: time course and mechanisms of change. *Journal of Consulting and Clinical Psychology*, 70, 267-274.

World Health Organization (1992). *ICD-10. Classification of Mental and Behavioral Disorders. Clinical Descriptions and Diagnostic Guidelines.* Geneva: World Health Organization.

Yanovski, S., Leet, M., Yanovski, J. A., Flood, M., Gold, P. W., Kissileff, H. R., & Walsh, B. T. (1992). Food selection and intake of obese women with binge-eating disorder. *American Journal of Clinical Nutrition*, 56, 975-980.

Yanovski, S. Z., Billington, C. J., Epstein, L. H., Goodwin, N. J., Hill, J. O., Pi-Sunyer, F. X., Rolls, B. J., Stern, J. S., Wadden, T. A., Weinsier, R. L., Wilson, G. T., & Wing, R. R. (2000). Dieting and the development of eating disorders in overweight and obese adults. *Archives of Internal Medicine*, 160, 2581-2589.

Yanovski, S. Z., Nelson, J. E., Dubbert, B. K., & Spitzer, R. L. (1993). Association of binge eating disorder and psychiatric comorbidity in obese subjects. *American Journal of Psychiatry*, 150, 1472-1479.

Young, J. E. (1999). *Cognitive therapy for personality disorders: A schema-focused approach.* (3rd ed.) Sarasota, Florida: Professional Resource Press.

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Publications

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Dingemans, A.E., Martijn C., Jansen A., & van Furth E.F. (2009). The effect of suppressing negative emotions on eating behaviour in Binge Eating Disorder. *Appetite, 52*, 51-57.

Dingemans, A. E., Spinhoven, P., & Van Furth, E. F. (2007). Predictors and mediators of treatment outcome in patients with binge eating disorder. *Behaviour Research And Therapy, 45,* 2551-2562.

Dingemans, A. E., Spinhoven, P., & van Furth, E. F. (2006). Maladaptive core beliefs and eating disorder symptoms. *Eating Behaviours, 7,* 258-265.

Dingemans A.E., van Hanswijck de Jonge P., van Furth, E.F. (2005). "The empirical status of binge eating disorder" (pp. 63-82) in C. Norring en B. Palmer (Eds.), *EDNOS, Eating Disorders Not Otherwise Specified. Scientific and Clinical Perspectives on the Other Eating Disorders*, Hove, East Sussex: Routledge.

Dingemans, A. E., Bruna, M. J., & van Furth, E. F. (2002). Binge eating disorder: A review. *International Journal of Obesity & Related Metabolic Disorders, 29,* 299-307.

Dingemans, A. E., Bruna, M. J., & van Furth, E. F. (2001). Binge eating disorder: A review / Vreetbuistoornis: Een overzicht. *Tijdschrift voor Psychiatrie, 43*.

Dingemans, A. E., van Vliet, I. M., Couvée, J.E., & Westenberg, H. G. (2001). Characteristics of patients with social phobia and their treatment in specialized clinics for anxiety disorders in the Netherlands. *Journal Of Affective Disorders, 65,* 123-129.

van Oel, C. J., Baare, W. F., Hulshoff Pol, H. E., Haag, J., Balazs, J., Dingemans, A., Kahn, R. S., & Sitskoorn, M. M. (2001). Differentiating between low and high susceptibility to schizophrenia in twins: the significance of dermatoglyphic indices in relation to other determinants of brain development. *Schizophrenia Research, 52,* 181-193.

Hulshoff, H. E., Hoek, H. W., Susser, E., Brown, A. S., Dingemans, A., Schnack, H. G., van Haren, N. E. M., Ramos, L. M. P., Gispen-de Wied, C. C., & Kahn, R. S. (2000). Prenatal exposure to famine and brain morphology in schizophrenia. *American Journal of Psychiatry, 157,* 1170-1172.

Sterken, Y., Postma, A., de Haan, E. H. F., & Dingemans, A. (1999). Egocentric and exocentric spatial judgements of visual displacement. *Quarterly Journal Of Experimental Psychology Section A-Human Experimental Psychology, 52,* 1047-1055.

Curriculum Vitae

Alexandra Dingemans was born on June 25th 1971 in Maastricht. She completed secondary education (gymnasium β) at the Sint-Maartenscollege in Maastricht in 1989. She studied Psychology at the University Utrecht, graduating in Psychonomics in August 1995. After her graduation she started working at the University Medical Center Utrecht at the department of Psychiatry. First she was a research assistant within a large twin and family study on schizophrenia and subsequently a researcher within two projects: A Dutch Famine Study on schizophrenia (prof. R.S. Kahn and dr. H.W. Hoek) and Social Phobia: A survey (Dr. I.M. van Vliet and drs. J. Couvée). In 1999 she started working as a researcher at the Center for Eating Disorders 'Ursula', which led to this thesis. Alexandra will continue to work at the Center for Eating Disorders 'Ursula' in Leidschendam.