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

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Citation
Dingemans, A. (2009, June 9). *Binge or control? : assessment of the validity, treatment and underlying mechanisms of Binge Eating Disorder*. Retrieved from https://hdl.handle.net/1887/13829

Version:	Not Applicable (or Unknown)
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Downloaded from:	https://hdl.handle.net/1887/13829

Note: To cite this publication please use the final published version (if applicable).

Chapter 8

Summary and general discussion

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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.