Validity and Maintenance of Binge Eating Disorder

Laboratory and naturalistic studies on the role of negative affect, stress-induced eating and cortisol secretion in obese women with BED

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SUMMARY AND OUTLINE

The prevalence of obesity keeps increasing globally. In the European Union member states, approximately 60% of adults and over 20% of school-aged children are currently overweight or obese (BMI ≥ 25, The International Obesity Taskforce, 2012). About 30% of obese individuals who enroll in weight loss programs (Spitzer et al., 1993c) and 2,5-5% of individuals in community-based samples (Smink, van Hoeken, & Hoek, 2012) suffer from binge eating disorder (BED). BED is characterized by consuming large amounts of food accompanied with the feeling of loss of control. In contrast to patients with bulimia nervosa (BN), BED individuals show no compensatory behavior afterwards. To date, BED is listed in the Appendix of the DSM IV-TR of the American Psychiatric Association (American Psychiatric Association, 2000), but might be included in DSM-V as full diagnosis soon (American Psychiatric Association, 2012).

For it is a relatively new diagnosis, there is need to further support the validity of the syndrome of BED. Furthermore, there is only little knowledge on the development and maintenance of BED. The current thesis therefore aims to further contribute to the validity of BED and to support the significance of psychological as well as biological maintenance factors of BED in a sample of obese women in laboratory as well as under naturalistic conditions. This was done in order to provide a better understanding for the syndrome of BED, which is of importance for its treatment.

Concerning its distinction from obesity, it has often been questioned if BED is just an "epiphenomenon of obesity" rather than a clinically relevant and distinct eating disorder (Tuschen-Caffier, 2005). Particularly, one important evidence for validity of BED, a higher binge eating frequency in the natural environment in obese individuals with BED as compared to obese without BED, is mixed (Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009).

It is not clear from previous studies, which stimuli induce overeating or binge eating in BED. Since psychosocial stressors are well-known triggers of binge eating in BN (Hilbert, Vogele, Tuschen-Caffier, & Hartmann, 2011), their role in BED should be investigated more closely. Above all, there is no evidence, which behavioral patterns of eating (i.e., eating rate) characterize the eating behavior of obese BED as compared to obesity without eating disorder. Such patterns could be a moderator between psychosocial stress and binge eating and give important insight into underlying mechanisms involved in the regulation of

food intake in BED. These psychological as well as biological mechanisms are in need of further study, since they are not as well understood as in BN. Psychological theories mainly focus on the role of dietary restraint and negative affect. Biological approaches include the investigation of appetite regulation as well as the Hypothalamic-Pituitary-Adrenal (HPA) axis in BED individuals. Because cortisol is involved in appetite regulation and energy balance (Dallman et al., 2004), a hyperactive HPA axis has been hypothesized as a biological basis of BED, but evidence is rare and inconclusive.

The present thesis addresses the validity of BED as well as underlying mechanisms of BED from three different angles. After a brief introduction to the topic (Chapter1), data discriminating obesity with BED from obesity without BED regarding eating in the natural environment, psychiatric comorbidity, negative affect as well as self reported tendencies in eating behavior are provided by study 1 in chapter 2. Evidence for possible psychological mechanisms explaining increased intake of BED individuals in the natural environment are investigated by analyzing associations of negative affect, emotional eating, restrained eating and caloric intake in obese BED compared to NBED controls. Chapter 3 provides an experimental study (study 2) on the impact of a psychosocial stressor, the Trier Social Stress Test (TSST, Kirschbaum, Pirke, & Hellhammer, 1993), on behavioral patterns of eating behavior in laboratory. Special attention is given to stress-induced changes in variables that reflect mechanisms of appetite regulation in obese BED individuals compared to controls. To further explore by which mechanisms stress might trigger binge eating, Chapter 4 investigates differences in stress-induced cortisol secretion after a socially evaluated cold pressure test (SECPT, Schwabe, Haddad, & Schachinger, 2008) in obese BED as compared to obese NBED individuals.

Taken together, the three studies provide answers to the following questions: 1) Which differences in psychopathological features exist between obese women with and without BED? 2) Which differences in naturalistic eating behavior and eating habits exist between obese women with and without BED? 3) Are there specific relationships between affective symptoms and eating behavior in obese women with BED? 4) Does psychosocial stress change behavioral patterns of eating associated with hunger and satiety specifically in obese women with BED? 5) Is there a higher reactivity of the HPA axis, indicated by heightened stress response of cortisol in obese BED women? Finally, the overall

implications of the results for theory and therapy of BED will then be discussed in Chapter 5. Thereby, also developments that have taken place after the studies had been finished will be considered.

ZUSAMMENFASSUNG

Die Prävalenz von Adipositas steigt weltweit. In den europäischen Mitgliedsstaaten sind derzeit etwa 60% der Erwachsenen und über 20% der Kinder im Schulalter übergewichtig oder adipös (BMI $\geq$ 25, The International Obesity Taskforce, 2012). Etwa 30% der Adipösen in Gewichtsreduktionsprogrammen leiden unter einer Binge Eating Störung (BES). Die BES ist durch das wiederholte Auftreten von Essanfällen gekennzeichnet, bei denen große Mengen an Nahrungsmitteln mit einem einhergehenden Gefühl des Kontrollverlusts verzehrt werden. Im Gegensatz zu Patienten mit einer Bulimia Nervosa (BN) ergreifen BES Patienten nach diesen Essanfällen keine Gegenmaßnahmen. Aktuell ist die BES im Anhang B des Diagnostischen und Statistischen Manuals psychischer Störungen (DSM IV-TR, American Psychiatric Association, 2000) klassifiziert, sie wird aber möglicherweise schon bald als Diagnose im DSM-V erscheinen (American Psychiatric Association, 2012).

Die Validität der BES – als relativ neue Esstörung – sollte weiter belegt werden. Es gibt bisher nur wenig gesichertes Wissen über ihre Entstehung und Aufrechterhaltung. Die vorliegende Arbeit möchte daher einerseits einen Beitrag zur Untermauerung der Validität der BES leisten. Des Weiteren soll die Relevanz einzelner biologischer sowie psychologischer aufrechterhaltender Faktoren der BES empirisch gestützt werden. Dazu wurde eine Stichprobe adipöser Frauen mit und ohne BES sowohl im Labor als auch in der natürlichen Umgebung untersucht. Die Ergebnisse sollen zu einem umfassenderen Verständnis der BES und damit zu einer Verbesserung ihrer Behandlungsmöglichkeiten beitragen.

Es wurde häufig angezweifelt, ob es sich bei der BES wirklich um eine eigenständige Essstörung und nicht vielmehr um ein reines "Epiphänomen der Adipositas" handelt (Tuschen-Caffier, 2005). Insbesondere konnte ein wichtiger Beleg für die Validität der BES, die höhere Auftretenshäufigkeit von Essanfällen in der natürlichen Umgebung im Vergleich zu Übergewichtigen ohne BES, bisher nicht hinreichend erbracht werden (Wonderlich et al., 2009).

Auch ist bisher nicht ausreichend geklärt, welche Stimuli bei Personen mit BES einen Essanfall auslösen. Psychosozialer Stress ist bei der BN als Auslöser für Essanfälle bekannt (Hilbert et al., 2011). Dies legt nahe, seine Rolle bei der BES ebenfalls genauer zu untersuchen. Weiterhin ist nicht bekannt, inwiefern sich adipöse Personen mit einer BES

von Adipösen ohne Essstörung in der Mikrostruktur ihres Essverhaltens (z.B. Essgeschwindigkeit) unterscheiden. Solche Merkmale könnten den Einfluss von psychosozialem Stress auf das Essverhalten moderieren und wichtige Hinweise auf zugrundeliegende Mechanismen in der Regulation der Nahrungsaufnahme bei der BES liefern. Diese psychologischen und biologischen Mechanismen bedürfen der weiteren Untersuchung.

Psychologische Theorien haben sich bisher weitestgehend auf die Rolle des gezügelten Essverhaltens und des negativen Affekts bei der BES fokussiert. Biologische Ansätze untersuchten die Regulationsmechanismen bei der Nahrungsaufnahme sowie der Hypothalamus-Hypophysen-Nebennierenrinden Achse (HHNA) bei Personen mit BES. Da Cortisol eine wichtige Rolle bei der Energieversorgung und Appetitregulation spielt (Dallman et al., 2004), könnte eine hyperaktive HHNA eine biologische Grundlage der BES darstellen.

Die vorliegende Arbeit befasst sich aus drei unterschiedlichen Blickwinkeln mit der Frage der Validität der BES sowie mit den der BES zugrundeliegenden Mechanismen: Nach einer kurzen theoretischen Einführung (Kapitel 1), werden Ergebnisse zur Abgrenzung der BES von der Adipositas vorgestellt. Dazu werden in Studie 1 (Kapitel 2) Befunde zum Essverhalten in der natürlichen Umgebung, zur psychiatrischen Komorbidität und zum negativem Affekt sowie zu selbstberichteten Tendenzen im Essverhalten berichtet. Anhand der Zusammenhänge von negativem Affekt, restriktivem Essverhalten, emotionalem Essverhalten und der Nahrungsaufnahme in der natürlichen Umgebung werden mögliche psychologische Mechanismen, die einer erhöhten Nahrungsaufnahme der Personen mit BES im Vergleich zu Adipösen ohne BES zugrunde liegen könnten, untersucht. Kapitel 3 beschreibt eine experimentelle Studie (Studie 2), die den Einfluss eines psychosozialen Stressors, dem Trier Sozial Stress Test (TSST, Kirschbaum et al., 1993) auf die Mikrostruktur des Essverhaltens im Labor untersucht. Besondere Berücksichtigung finden dabei Stress-induzierte Veränderungen von Variablen, die Mechanismen der Appetitregulation widerspiegeln bei Adipösen mit BES im Vergleich zu Adipösen ohne BES. Studie 3 (Kapitel 4) beschäftigt sich mit den biologischen Mechanismen, die bei der Stress-induzierten Auslösung von Essanfällen aktiv sind. Anhand von Speichelkortisolproben nach einem experimentellen Stressor, einem „sozial-evaluativen

Cold Pressor Test“ (SECPT, Schwabe, Haddad, & Schachinger, 2008), wurde die Stress-induzierte Kortisolsekretion bei den beiden Gruppen untersucht.

Die drei Studien geben Antwort auf folgende Fragen: 1) Welche Unterschiede in Bezug auf psychopathologische Charakteristika bestehen zwischen adipösen Frauen mit und ohne BES? 2) Welche Unterschiede im Essverhalten in der natürlichen Umgebung bestehen zwischen adipösen Frauen mit und ohne BES? 3) Gibt es für die BES spezifische Zusammenhänge zwischen Essverhalten und negativem Affekt in Form von Depressivität und Ängstlichkeit? 4) Gibt es für die BES spezifische Stress-induzierte Veränderungen in der Mikrostruktur des Essverhaltens, die mit Hunger- und Sättigungsmechanismen assoziiert sind? 5) Gibt es eine erhöhte Cortisol-Stressreaktivität bei adipösen Frauen mit BES? Im abschließenden Kapitel werden schließlich die Implikationen der Befunde für Theorie und Therapie der BES zusammenfassend diskutiert.

1. CONCEPTUAL FRAMEWORK

1.1. Introduction

This chapter will outline the conceptual framework underlying the present thesis. The issues addressed here will then be detailed in the three following studies.

In section 2, the distinction of obesity from BED will be addressed, with special regard to laboratory and naturalistic eating behavior, the tendency to eat in response to negative mood states and restrained eating. A definition of the microstructure of eating behavior will then be given in section 3. The influence of stress on the microstructure of eating as well as the relation of eating patterns to physiological mechanisms in the regulation of appetite will be described in this section as well. Section 4 outlines the impact of stress on human eating behavior with regard to the role of psychosocial stressors, types of foods consumed during stress and possible moderating variables. Furthermore, a biological pathway through which stress might influence food intake, the HPA axis, will be described. Evidence for the role of stress as trigger of binge eating in BED will be reviewed in section 5. A short description of important laboratory stressors is given in section 6. Finally, in section 7, evidence for a disturbance in appetite regulation as well as findings on HPA axis functioning in BED will be reviewed. Section 8 summarizes the chapter and gives an outline of the following chapters.

1.2. Distinction of BED from obesity

The core symptom of BED is recurrent overeating with loss of control (binge eating) in the absence of inappropriate compensatory behaviors and/or extreme dietary restraint. Diagnostic criteria for BED from DSM-IV (Appendix B: Criteria Sets and Axes for Further Study) are listed Appendix A. Longitudinal studies suggest that binge eating without compensation in BED leads to weight gain and obesity (Devlin, 2007). Since the prevalence of BED is higher in overweight populations than in the general population and BED has shown to be associated with a higher BMI and obesity (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Hudson, Hiripi, Pope, JR., & Kessler, 2007; Pike, Dohm, Striegel-Moore, Wilfley, & Fairburn, 2001), it has been questioned whether BED is really a distinct eating disorder and not just an “epiphenomenon of obesity” (Tuschen-Caffier, 2005, p. 138). However, Wilfley et al. already concluded in 2003, that “persons with BED

are not simply obese individuals with comorbid psychiatric disorders (…) but are a distinctive subset of the obese population" (Wilfley, Wilson, & Agras, 2003, pp. S97-S98). This is assumed on the basis of following reasons: Compared to obese individuals without BED, obese individuals with BED eat more in laboratory, have increased comorbidities which are accounted for by the severity of binge eating rather than by the degree of obesity, display more eating pathology (i.e., chaotic eating habits, more emotional eating) and report poorer quality of life, as well as impairment in work and social functioning (for review, see also Striegel-Moore & Franko, 2008; Wonderlich et al., 2009). Compared to obese NBED, obese BED subjects furthermore report an earlier onset of overweight, earlier diet attempts and more weight cycling.

1.2.1. Differences in psychopathological characteristics

Obese BED individuals have elevated levels of axis I and II comorbitities. Particularly elevated levels of mood, anxiety and substance-related disorders have been reported (Bulik, Sullivan, & Kendler, 2002; Grilo, White, & Masheb, 2009b; Hudson et al., 2007; Javaras et al., 2008; Specker, Zwaan, Raymond, & Mitchell, 1994; Telch & Stice, 1998; Wilfley et al., 2000; Yanovski, Nelson, Dubbert, & Spitzer, 1993a).

Concerning weight and shape concerns and body image disturbance, which are important symptoms of eating disorders, BED subjects are comparable to subjects with BN and exhibit higher levels than non-binge eating controls (for review, see Hilbert, 2005; Wonderlich et al., 2009).

1.2.2. Differences in eating behavior and eating habits

1.2.2.1. Laboratory eating behavior

Regarding laboratory eating behavior (see Appendix B for a review of studies), a higher food intake of obese BED as compared to obese NBED has been shown especially in studies providing big portion sizes or unlimited amounts of food together with binge instructions (Anderson, Williamson, Johnson, & Grieve, 2001; Bartholome, Peterson, Raatz, & Raymond, 2012; Galanti, Gluck, & Geliebter, 2007; Geliebter, Hassid, & Hashim, 2001; Goldfein, Walsh, LaChaussee, Kissileff, & Devlin, 1993; Gosnell et al., 2001; Guss, Kissileff, Devlin, Zimmerli, & Walsh, 2002; Latner, Rosewall, & Chisholm,

2009; Raymond, Bartholome, Lee, Peterson, & Raatz, 2007; Sysko, Devlin, Walsh, Zimmerli, & Kissileff, 2007; Telch & Agras, 1996a; Yanovski et al., 1992).

1.2.2.2. Naturalistic eating behavior

Data of naturalistic eating behavior have not consistently shown that obese BED individuals engage in more binge eating episodes than obese NBED individuals: Two studies (Greeno, Wing, & Shiffman, 2000; Le Grange, Gorin, & Catley D., 2001) using ecological momentary assessment (EMA[1]) did not find significant differences between obese BED and obese NBED subjects in self-reported binge eating frequency. Hence, a more recent study combining EMA with a computer-based dietary recall measure, found that obese participants with BED exhibited more binge eating episodes than obese controls (Engel et al., 2009). These mixed results question the validity of BED.

1.2.2.3. Emotional eating

Emotional eating describes the tendency to eat in response to negative mood states in order to cope with them (Arnow, Kenardy, & Agras, 1995) and has been shown to be more pronounced in obese BED compared to obese NBED individuals (Eldredge & Agras, 1996; Pinaquy, Chabrol, Simon, Louvet, & Barbe, 2003; Ricca et al., 2009). Besides perceived stress, emotional eating has also been shown to be a significant predictor of BED in obese (Pinaquy et al., 2003), highlighting the importance of these two factors in the maintenance of BED. Regarding the effects of different emotions on eating pathology in BED individuals, negative- interactional emotions (such as anger, feelings of being hurt, guilt and disappointment) have reported to be stronger related to binge eating or desire to binge eat than negative-depressive emotions (Zeeck, Stelzer, Linster, Joos, & Hartmann, 2010).

1.2.2.4. Restrained eating

Restrained eating is defined as the tendency to restrict food intake in order to control body weight (e.g., attempts to follow dietary rules or avoid special types of foods) (Herman &

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[1] A method for the assessment of daily eating behavior and subjective ratings in the naturalistic setting, at periodic intervals, often by random time sampling, using technologies ranging from written diaries and telephones to electronic diaries and physiological sensors (Shiffman, Stone, and Hufford, 2008).

Mack, 1975; Hilbert, 2005). Because the cognitive control over food intake is supposed to override signals of hunger and satiety, restrained eaters usually eat less than unrestrained controls. Under certain conditions such as after alcohol consumption or when feeling depressed or anxious, normal weight restrained eaters eat more than unrestrained controls (Herman & Polivy, 1975; Stunkard & Messick, 1985). It is assumed that under these conditions there is a disinhibition of cognitive control over food intake (Herman & Mack, 1975; Herman & Polivy, 1975). Although restrained eating is considered to be a risk factor in the etiology and maintenance of eating disorders, several studies have assessed dietary restraint in BED and found only moderate restraint scores in obese BED which did not differ significantly from obese NBED (Brody, Walsh, & Devlin, 1994; Marcus, Wing, & Hopkins, 1988; Pinaquy et al., 2003; Wilson, Nonas, & Rosenblum, 1993; Yanovski, 1993). Nevertheless, binge eating in BED might be associated with a general tendency towards eating disinhibition (e.g., eating in response to emotional or external cues, see Hilbert, 2005, p. 156). Eating disinhibition has shown to precipitate binge eating in BED individuals (Castonguay, Eldredge, & Agras, 1995).

1.3. Microstructure of eating behavior

As compared to total food intake, the microstructure of eating can give insight in underlying physiological or cognitive mechanisms regulating food intake. This chapter describes the microstructural approach in measuring eating behavior (1.3.1.) and reviews evidence for differences in microstructural eating patterns in BED as compared to obesity (1.3.2.). Afterwards evidence for stress-induced changes in microstructural eating patterns (1.3.3.) and for an interrelationship between eating style and appetite regulation (1.3.4.) is summarized.

1.3.1. Definition and interpretation

Eating behavior can be studied from a macro as well as from a microstructural perspective (see Laessle & Lehrke, 2012). The macrostructural approach observes energy intake and eating patterns over longer periods of time (e.g., meal patterns over days or weeks). The microstructural approach (Guss & Kissileff, 2000) focuses on intake parameters, or behavioral patterns of eating, within a single meal (e.g. eating rate, size and frequency of spoonfuls), and allows to identify variables, which may promote excessive caloric intake.

The behavioral patterns of eating can be reliably described by the characteristics of cumulative intake curves (CIC) obtained by an universal eating monitor (UEM). The UEM used in the current research consisted of a plate placed on a hidden scale, which records weight changes every 0.5 seconds. The recorded weight-loss data can be reliably described by the CIC: $y = ax^2 + bx + c$ (Kissileff, Thornton and Becker, 1982). In the CIC, y is the amount of food ingested, x is the time, a is the change in the slope of the curve over time which equals change of eating rate (CER), b is the constant slope of the curve over time, which equals the initial eating rate (IER) and c is a constant reflecting food intake at the start of a meal (Kissileff et al, 1982, Westerterp-Platenga et al., 1990, see also Ioakimidis et al., 2011, p. 762).

According to Kissileff et al. (1982) and Guss & Kissileff (2000) IER, reflects facilitatory physiological or cognitive processes in the regulation of food intake, which are related to motivation to eat or hunger. In contrast, CER reflects inhibitory physiological and cognitive processes which control eating over the meal which are related to satiation. Deceleration at the end of the meal reflects satiation, so that less deceleration at the end of the meal points to less satiation.

1.3.2. Microstructure of eating in BED

While normal weight unrestrained subjects show decelerated cumulative intake curves, the curves of obese individuals have been characterized either as linear, which means that there is almost no deceleration at the end of the meal (CER close to zero, Westerterp-Plantenga et al., 1990; Westerterp-Plantenga, Wouters, & Hoor, 1990, Westerterp-Plantenga, Wouters, & Hoor, 1991) or by higher IER (Laessle, Lehrke, & Duckers, 2007). To date, there are no studies on the microstructural patterns of eating behavior in BED using cumulative intake curves. There are some studies on the overall eating rate (total intake (g)/duration of the meal (s)) in BED, which did not find differences as compared to NBED subjects (Geliebter et al., 2001; Telch & Agras, 1996a; Yanovski et al., 1992). The overall eating rate does not give insight in underlying mechanisms of appetite regulation and is rather unspecific. Therefore, more detailed analysis of the microstructural patterns of eating in BED is warranted.

1.3.3. Stress-induced changes in the microstructure of eating

Changes in the microstructure of eating behavior could be a moderator of the stress-eating-relationship. It has been found that after an experimental stress induction, normal weight men and women show less deceleration at the end of a meal as compared to a neutral condition (Dovey, Clark-Carter, Boyland, & Halford, 2009; Frank, 2003). Again, studies are rare, and there are no studies investigating stress-induced microstructural changes in eating behavior of BED individuals.

1.3.4. Impact of eating style on food intake and appetite regulation

Changes in microstructural patterns of eating such as increased speed of eating may put individuals at risk of eating too much (Ioakimidis, Zandian, Bergh, & Sodersten, 2009). It has been shown, that eating rate has an impact on postprandial secretion of PYY and GL-1[2]. Kokkinos et al. (2010) found that over a period of 210 min postprandial PYY and GL-1 secretion was higher in individuals who ate 300ml ice cream in 30 min compared to those who ate the same amount in 5 minutes.

In the other direction weight loss interventions combining standard therapy with a training to slow down eating behavior[3] in obese have shown to improve weight loss and reduce portion-sizes during test meals without reducing the feeling of satiety (Ford et al., 2010; Galhardo et al., 2012) compared to standard therapy. Interestingly, in the study of Galhardo et al (2012) only individuals who trained to slow down their eating rate exhibited changes in gastrointestinal hormone responses, whereas individuals in the standard therapy did not. Changes in gastrointestinal hormones included decreased orexigenic responses in terms of fasting and postprandial ghrelin (an important signal for hunger, which is secreted by the mucosa of the empty stomach) as well as increased anorexigenic responses in terms of fasting and postprandial PYY.

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[2] Peptide tyrosine-tyrosine (PYY), glucagon-like peptide-1 (GL-1) and cholecystokinin (CKK) are short-acting (for instance throughout the day) and anorexigenic intestinal peptides which are thought to act as postprandial satiety signals (Galhardo et al., 2012; Chaudhri, Field, and Bloom, 2008).

[3] The intervention to slow down eating rate was performed with a Mandometer (Ioakimidis, Zandian, Bergh, and Sodersten, 2009). The Mandometer is a method for the control of eating rate based on UEM, which gives subjects feedback from a computer screen on how much and at what rate to eat during a meal.

1.4. The stress-eating-relationship

This chapter reviews evidence of the stress-eating relationship in humans. Firstly, it describes how stress can alter total food intake and how stress can promote consumption of nutrient dense, so-called "palatable" foods. Secondly, it is discussed how the physiological responses to stress may interact with processes involved in appetite regulation.

1.4.1. Effect of stress on eating behavior

Stress can be associated with overall increased as well as decreased food intake (Adam & Epel, 2007; Macht, 2008; Stone & Brownell, 1994). The specific outcome can differ according to the nature of the stressor (physical or psychological), specific types of food, individual differences as well as the duration of the stressor (acute or chronic see 1.4.2.) (Adam & Epel, 2007).

While psychosocial stressors[4] such as ego-threatening, interpersonal and work-related stressors have shown to be associated with increased snacking, physical stressors have found to be associated with decreased snacking (O'Connor, Jones, Conner, McMillan, & Ferguson, 2008).

Stress has been shown to increase intake of more palatable or easily consumed foods like snacks, fast foods, high-fat and high-sugar foods in different study populations when assessed via self-reports (Oliver & Wardle, 1999), EMA (Conner, Fitter, & Fletcher, 1999; O'Connor et al., 2008; Steptoe et al., 1998) as well as after experimental stress-induction (Habhab, Sheldon, & Loeb, 2009; Zellner et al., 2006; Zellner, Saito, & Gonzalez, 2007). Furthermore, several studies show that stress leads to eating patterns that are characterized by a decreased food intake in main meals but an increased between-meal food consumption or snacking (Conner et al., 1999; O'Connor et al., 2008; Sims et al., 2008). For example, in a study of O'Connor (2008), daily hassles were not only associated with increased consumption of high-fat and high-sugar snacks but also with a concomitant reduction in main meals and in vegetable consumption.

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[4] Psychological stress exerts physiological, behavioral, and/or verbal-subjective stress responses as a result of stressful social interactions, which often involve social-evaluative threat (in contrast to stress responses provoked by physical exertion or pharmacological stimulation) (Kudielka & Wüst, 2010).

There are some 'individual differences' (Greeno & Wing, 1994) that moderate the relationship between stress and eating. It is difficult to determine their individual impact on the stress-eating relationship, because these individual differences are interrelated. For example, women are more likely to be restrained as well as emotional eaters (van Strien, Frijters, Bergers, & Defares, 1986) and restrained eaters are more likely emotional eaters (see O'Connor & Conner, 2011). A study investigating the effects of multiple moderators simultaneously of O'Connor (2008) found that the hassles-snacking relationship outlined above was stronger and more positive in females, obese and participants with higher levels of restraint, emotional eating and external eating. Among all moderator variables, emotional eating was the preeminent moderator of the hassles-eating relationship.

Taken together, the results suggest that people appear to use eating palatable foods as coping mechanism (Laitinen, Ek, & Sovio, 2002). This may contribute to excess energy intakes and weight gain, which is supported by longitudinal studies. This suggests that there is an association between chronic life stress and future weight gain (Kivimaki et al., 2006; Mellbin & Vuille, 1989).

1.4.2. Biological stress responses and food intake

Biological responses to stressors can be divided in two important patterns: The "active fight and flight" pattern is induced by activation of the sympathetic-adrenomedullary (SAM) system followed by a secretion of adrenaline and noradrenaline. The "passive" pattern is a result of Hypothalamic-Pituitary-Adrenal (HPA) axis activation and the consequent cortisol secretion (Adam & Epel, 2007; Torres & Nowson, 2007). The activation of the HPA leads to the secretion of corticotropin releasing hormone (CRH) from the hypothalamus, which stimulates the pituitary to release the adrenocorticotropic hormone (ACTH). After ACTH is released into the blood, it triggers the adrenal gland to release glucocorticoid (GC) hormones, especially cortisol, with a negative feedback of the latter on the other two (see Dallman & Hellhammer, 2011). GCs promote the fast release of energy. In the presence of low insulin concentrations, GC are catabolic on tissues high in protein and fat, which leads to increased amino acids and free fatty acids which can be used for glucose production in the liver. Furthermore, they directly promote gluconeogenesis in the liver. Thus, GC "serve a key role in providing the essential glucose" (Dallman & Hellhammer, 2011, p.14).

Whereas noradrenaline and CRH have been reported to suppress appetite, cortisol stimulates appetite during recovery from stress (Torres & Nowson, 2007). There is evidence, that cortisol affects appetite regulation via inhibition of leptin (which is expressed in adipose tissue and inhibits food intake) and stimulation of neuropeptide Y (NPY), one of the most potent central appetite stimulants known (Gutzwiller & Beglinger, 2005; Torres & Nowson, 2007).

The characteristics of the stressor determine, which of the two stress systems is predominantly activated. Activation of the SAM system typically occurs under conditions of acute, short-time stress (Cohen, 2000). On the contrary, chronic stress has been associated with hyper-activation of the HPA axis (Bjorntorp, Holm, Rosmond, & Folkow, 2000). The appraisal of the stressor is also of importance in determining which of the stress systems will be activated over the other: If the stressor is regarded as 'challenge' (a demanding but controllable situation, which one has the resources to cope with (Adam & Epel, 2007), the SAM system is activated. If the stressor is perceived as 'threat' (an uncontrollable, "demanding, situation that one has not the resources to cope well with", Adam & Epel, 2007, p.451), the HPA axis is differentially activated.

1.5. Important laboratory stressors

Consistent with the theoretical considerations above, a meta-analysis has shown that laboratory stressors that combine uncontrollability and social-evaluative components were associated with the largest cortisol and ACTH hormone changes (Dickerson & Kemeny, 2004). The social-evaluative component includes the threat of being negatively judged by others. There are two stress protocols, both of them used in the current research, that have been shown to produce significant cortisol responses, the 'Trier Social Stress Test' (TSST, Kirschbaum et al., 1993) and the 'socially evaluated cold pressor test' (SECPT, Schwabe et al., 2008). The TSST contains a stress anticipation phase and a stress phase with a free speech assignment and subsequent performance of a mental arithmetic. These tasks are performed in front of two clinical research members while participants are under the impression of being videotaped for subsequent behavioral analysis. In the SECPT, subjects are asked to immerse their right hand including the wrist into ice water (0–4 °C) as long as possible. While doing so, they are watched by an experimenter all the time. Additionally,

as in TSST, they are under the impression of being videotaped for subsequent analysis and asked to look into the camera all the time.

1.6. Triggers for binge eating in BED

This chapter briefly describes psychological theories on the maintenance of BED (1.6.1.). Afterwards evidence for the role of negative affect (1.6.3.) and stress (1.6.4.) in the initiation of binge eating episodes in BED is summarized. Since studies on restrained eating are reviewed in chapter 1.2.2.4., they are not summarized again.

1.6.1. Psychological models of binge eating in BED

Current psychological models of initiation and maintenance of binge eating in BED are mostly derived from BN (Munsch, Meyer, Quartier, & Wilhelm, 2012). Thereby, these theories mainly focus on the role of restrained eating and negative affect. According to the restraint theory of BED (for review, see Howard & Porzelius, 1999), the desire for thinness leads to unrealistic dietary restraint, in which normal hunger and satiety regulation is replaced by cognitive control mechanisms. The cognitive control can be interrupted by events such as disobeying a dietary rule, resulting in all or nothing thinking and consequent overeating (abstinence violation effect). Disinhibition hypothesis (Herman & Mack, 1975; Herman & Polivy, 1975) states that the self control of restrained eaters can be temporarily released by disinhibitors like specific cognitions, alcohol or strong emotional states or stress. According to the affect regulation model (Hawkins & Clement, 1984) binge eating episodes are triggered by negative emotions. They function to reduce negative affect by using food for "comfort and distraction" (Haedt-Matt & Keel, 2011c, p. 2), when more adaptive coping mechanisms are not accessible. In turn, binge eating is hypothesized to provide temporary relief from negative affect, reinforcing the behavior. The affect regulation model proposes that negative affect should increase before binge eating and that binge eating is associated with a decrease in negative affect (Haedt-Matt & Keel, 2011c).

A model that integrated these two mechanisms is the dual pathway model of Stice (1994, 2001). In this social cultural model of bulimic behaviour in BN it is proposed that body dissatisfaction leads to binge eating through two pathways, from which either is sufficient: In the restraint pathway, body dissatisfaction results in dietary restraint as a weight control

technique. Dietary restraint in turn is hypothesized produce overeating for metabolic as well as psychological reasons (Polivy and Herman, 1985, van Strien, Engels, van Leeuwe, & Snoek, 2005). In the pathway of negative affect, body dissatisfaction is suggested to lead to negative emotions, such as depression. In turn, binge eating is hypothesized to reduce negative affect as stated by affect-regulation model.

There are no models which explicitly include the role of psychosocial stress in BED.

1.6.2. The role of negative affect

Negative affect was investigated more frequently than stress as a possible trigger for binge eating episodes in BED. Different study approaches have shown, that negative affect is "presumably the most well-established antecedent of binge eating in BED" (Hilbert, 2005, p.156).

Studies in the natural environment using EMA have shown that negative affect preceded binge eating in individuals with BED but not in weight matched controls (Greeno et al., 2000; Le Grange et al., 2001) and that mood was lower prior to binge eating than prior to regular eating episodes (Greeno et al., 2000). A recent meta-analysis (Haedt-Matt & Keel, 2011a), analyzing 36 EMA studies with BED as well as BN participants showed that above all studies, negative affect was a significant precursor of binge eating. Also experimental studies highlight the importance of negative affect in eliciting binge eating episodes. For instance, negative mood induction significantly increased the occurrence of self-defined binges as well as investigator-defined binges in laboratory in individuals with BED (Agras & Telch, 1998). Furthermore, negative affect following mood induction was associated with feeling of loss of control and shifted the perception of an eating episode from overeating to binge eating (Telch &Agras, 1996a). Negative affect might therefore be an important factor that discriminates overeating and binge eating in BED.

1.6.3. The role of stress

It is well known that environmental stressors and daily hassles are associated with the symptom of binge eating in humans (Mathes, Brownley, Mo, & Bulik, 2009; Rosenberg et al., 2012). Not only is stress associated with the initiation of binge episodes, but it also increases the reinforcing efficacy of foods that are commonly binged upon (Goldfield,

Adamo, Rutherford, & Legg, 2008; Greeno & Wing, 1994), highlighting the importance of its investigation in BED.

For bulimia nervosa, it has been experimentally shown that psychosocial stressors induce an increased desire to eat (Cattanach, Malley, & Rodin, 1988; Hilbert et al., 2011; Tuschen-Caffier & Vogele, 1999; Tuschen-Caffier, 2005) and that the desire to binge is specifically linked to interpersonal stress as compared to achievement stress (Hilbert et al., 2011; Tuschen-Caffier, 2005).

In contrast, experimental data for BED are rare. In the study of Hilbert et al. (2011), induction of idiosyncratic interpersonal stress increased the desire to binge eat in individuals with BED as well as in individuals with BN. Additionally, another study showed that after a cold pressure test (CPT), obese BED participants reported greater levels of hunger and desire to binge eat when compared with obese NBED participants (Gluck, Geliebter, Hung, & Yahav, 2004). Concordantly, perceived stress has been shown to emerge as significant predictor of BED status in obese BED and NBED subjects (Pinaquy et al., 2003). Together, these findings point towards the importance of stress as a triggering factor for binge eating episodes in BED, although further studies are needed to confirm the limited evidence.

1.7. Satiety regulation and HPA axis functioning in BED

Disturbances in satiety regulation as well as dysfunctions of the HPA axis are prominent explanations for the aberrant eating pattern in BED. This chapter firstly reviews evidence for a disturbance in the perception of hunger and satiety as well as their biological correlates in BED (1.7.1). Secondly, it summarizes findings on HPA axis functioning in BED (1.7.2.) hypothesizing that a hyperactivity of the HPA could be a moderating biological mechanism.

1.7.1. Evidence for a disturbance of appetite regulation in obese BED

There is evidence for a disturbance in the perception of hunger and/or satiety in individuals with BED. Anderson et al. (2001) and Geliebter et al. (2001) found that obese women with BED, although eating more than obese controls, did not feel more satiated. Sysko et al. (2007) found that although subjects with BED did not experience greater levels of hunger,

they consumed more macaroni and cheese in laboratory. Also, BED subjects consumed more food to reach a similar level of fullness. Guss et al. (2002) found that only BED individuals eat more when instructed to binge eat as compared to the instruction to eat normal, which implies that they might not regulate their intake according to internal cues of hunger and satiation.

Biological studies found correlates in individuals with BED which are related to increased hunger, such as reduced serotonin transporter binding (Wadden, Foster, Letizia, & Wilk, 1993) and increase of the regional cerebral blood flow in the left hemisphere under food exposure (Karhunen et al., 2000). Also increased levels of the orexigen peptide ghrelin have been hypothesized in individuals with BED. In contrast to this assumption, fasting ghrelin levels of obese BED have been reported to be even lower compared to controls (Geliebter, Gluck, & Hashim, 2005, Geliebter, Hashim, & Gluck, 2008; Monteleone et al., 2005). Because ghrelin values in BED have shown to normalize after a combined cognitive behavior and dietary intervention, the lower fasting ghrelin prior treatment in BED suggest that binge eating may downregulate ghrelin (Geliebter et al., 2008), possibly as an attempt of the body to prevent further binge eating. Binge eating despite low ghrelin levels suggests that ghrelin might not function as signal for meal initiation any more. Although also postprandial levels of ghrelin are reported to be lower in overweight or obese BED compared to NBED controls (Geliebter et al., 2005, Geliebter et al., 2008; Munsch, Biedert, Meyer, Herpertz, & Beglinger, 2009), ghrelin has been found to decline significantly less postprandially in obese BED individuals compared to obese controls (Geliebter et al., 2005, Geliebter et al., 2008). If one assumes that the magnitude of the fall in ghrelin may itself be a signal for satiation (Geliebter et al., 2005), the smaller decline in ghrelin could contribute to overeating in BED.

Other studies have suggested a decreased satiety response in BED: A higher gastric capacity in obese binge eaters as compared to NBED individuals has been reported by Geliebter et al. (2004). As gastric capacity is increased, satiety signals from gastric and post-gastric areas are reduced (Hellstrom et al., 2004). On the contrary, similarly to ghrelin, baseline levels of gastrointestinal satiety peptides such as PYY and CCK have been reported to be similar in obese BED as compared to obese controls (Geliebter et al., 2008; Munsch et al., 2009). Concerning meal-related changes of CCK and PYY, no

differences (Geliebter et al., 2004) or a higher increase of CCK and PYY (Munsch et al., 2009) have been found. As the authors concluded, these results may indicate "an initial effort of the system to prevent BED individuals from binge eating (…) [which] might be overwhelmed by the experience of the urge to binge eat by means of deficient self-regulatory processes" (Munsch et al., 2009, p.20).

1.7.2. HPA axis functioning in BED

Studies on HPA axis functioning in BED are rare and inconclusive. In the following section, results from different study approaches are summarized. Studies on basal cortisol levels (1.7.2.1.) and stimulated cortisol levels (1.7.2.2.) give mainly insight in adrenal functioning in BED, whereas the dexamethasone suppression test (DST) investigates the functioning of the whole HPA axis (1.7.2.3.). Furthermore, correlational data (1.7.2.4.) are described, which do not allow specific conclusions on HPA axis functioning in BED. Since results are inconclusive, further studies are needed to clarify the role of the HPA axis in BED.

1.7.2.1. Studies on basal cortisol levels in BED

Results on basal cortisol levels in BED are mixed. Gluck et al. (2004) found higher basal fasting cortisol levels in the morning in obese women with BED compared to obese women without BED. In contrast, another study found no differences in morning cortisol levels between corresponding groups (Monteleone et al., 2003). In both reported studies, there was only a single measurement of morning cortisol.

1.7.2.2. Cortisol stress reactivity in BED

Gluck et al. (2004) investigated the cortisol response of obese women with and without BED after a cold pressor test (CPT). They found, after controlling for contraceptive intake and area under the curve (AUC) for insulin, a nearly significant higher increase in cortisol levels for the BED compared to the NBED group (AUC, $p = 0,057$).

1.7.2.3. Feedback-mechanisms of the HPA-axis in BED

To the best of the writer's knowledge, there are only two studies who applied the dexamethasone suppression test (DST) in participants with BED (Gluck et al., 2004; Yanovski et al., 1993b). Both studies found no differences in cortisol suppression after

dexamethasone administration between obese women with and without BED. Compared to basal cortisol levels, Gluck et al. (2004) found a stronger relative decrease in cortisol levels in the BED group. Both studies applied the regular dose of 1 mg dexamethasone.

1.7.2.4. Correlative findings

There is evidence for a positive correlation between severeness of BED, measured by the Binge Eating Scale (BES, Gormally, Black, Daston, & Rardin, 1982), and cortisol levels in the evening in obese women with BED (Coutinho, Moreira, Spagnol, & Appolinario, 2007). This might point towards a disruption of the normal circadian rhythm of cortisol secretion by binge eating episodes which lead to higher cortisol levels in the evening.

1.8. Aims of the study

Based on the conceptual framework outlined above, the following studies aim to further contribute to the validity of BED and to support the significance of factors included in psychological as well as biological models of BED.

Chapter 2 presents results referring to the validity of BED diagnosis by discriminating obesity with BED from obesity without BED regarding eating in the natural environment, psychiatric comorbidity, negative affect as well as self reported tendencies in eating behavior. Evidence for affect regulation model and restraint theories of binge eating in BED is investigated by analyzing associations of negative affect, emotional eating, restrained eating and caloric intake in obese BED compared to NBED controls.

Chapter 3 provides experimental data on the impact of a psychosocial stressor on microstructural patterns of intake in BED as compared to obesity. Changes in objective parameters that reflect motivation to eat (hunger) as well as inhibitory processes (satiation) are investigated. Additionally, self-reported perceptions of hunger and satiety are investigated. Chapter 4 investigates changes in stress-induced cortisol secretion as possible mechanism of stress-induced changes in eating in BED.

Chapter 3 and 4 aim to investigate possible maintenance factors of BED which are included in the following hypothetical model (see figure 1): Stress changes the microstructural eating patterns (i.e., higher IER, less deceleration at the end of the meal)

specifically in obese women with BED. This relationship is moderated by stress-induced changes in cortisol levels (higher stress-reactivity of the HPA). Microstructural changes in eating promote increased food intake or binge eating, whereas this relationship is moderated by disturbances in the regulation or perception of hunger and satiety signals, which in turn lead to increased food intake or binge eating.

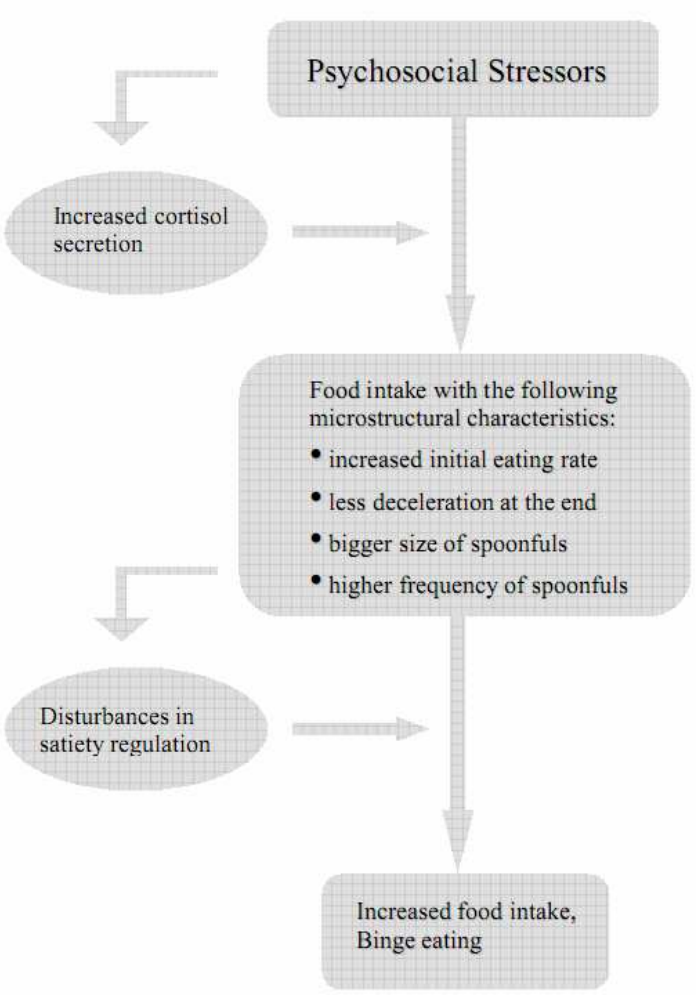

Figure 1: Hypothetical model of stress-induced eating in BED

2. ASSOCIATIONS OF NEGATIVE AFFECT AND BINGE EATING BEHAVIOR IN OBESE WOMEN WITH AND WITHOUT BINGE EATING DISORDER

2.1. Abstract

The present study was planned to investigate differences in psychopathological features, eating behavior and eating habits between obese women with and without BED. It also aimed to identify specific relationships between affective symptoms and eating behavior in obese women with BED.

84 obese women were studied (40 with BED, 44 NBED). Psychiatric comorbidities were assessed with the structured diagnostic interview for DSM IV (SCID). Depressive symptoms were measured with the Beck Depression Inventory (BDI) and anxiety with the state-trait anxiety inventory (STAI). Eating habits (emotional and restrained eating) were assessed by the Dutch eating behavior questionnaire (DEBQ). Food diaries were used for assessing naturalistic eating behavior (food intake) and mood before and after food intake.

BED subjects exhibited higher levels of comorbidity (in particular mood disorders, anxiety disorders and substance-related disorders), higher depressive symptoms, trait anxiety, external and emotional eating scores than NBED subjects. Regression analyses revealed that anxiety and emotional eating were significant predictors for BED status. In the BED group, depressive symptoms were significantly related to emotional eating and food intake and negatively related to restraint. Anxiety was significantly related to emotional eating. In general, food intake significantly enhanced mood. Mood was worse on the days with self-reported binge eating episodes than on nonbinge days.

These results are discussed with regard to etiological models for BED and for BED being a distinct diagnostic category separate from obesity.

2.2. Introduction

Binge eating disorder (BED) is associated with obesity. Community surveys have estimated the prevalence of BED between 2 and 5%. In overweight women seeking treatment the prevalence is about 30% (Spitzer et al., 1993a; Stunkard, 1959).

A number of studies have investigated characteristics associated with binge eating disorder. Regarding comorbidity, most studies found higher levels of lifetime Axis I

comorbidity in individuals with BED than in NBED individuals (Bulik et al., 2002; Grilo et al., 2009b; Hudson et al., 2007; Javaras et al., 2008; Specker et al., 1994; Telch & Stice, 1998; Wilfley et al., 2000; Yanovski et al., 1993a), whereas studies comparing non-treatment seeking obese BED and NBED individuals are underrepresented (Bulik et al., 2002; Yanovski et al., 1993a). Therefore, one aim of our study was to compare the psychiatric comorbidity of a non-treatment seeking sample of obese women with BED with NBED controls.

There is a discourse about whether BED is a clinically significant condition in terms of disordered eating. It is questioned whether BED is distinct from typical obesity or if individuals with BED do not eat differently than an obese control group but simply express more distress about the same eating patterns. Because of its importance for the validity of the BED construct, another aim of the study was to investigate eating behavior (in terms of emotional and restrained eating and naturalistic eating behavior) of obese BED compared to NBED individuals.

To date, little is known about the mechanisms driving the binge eating in BED. Theories have mainly focused on the role of negative affect and restraint in BED.

According to the restraint theory of BED (for review, see Howard & Porzelius, 1999), the desire for thinness leads to unrealistic dietary restraint. Hence, binge eating occurs to compensate for the excessive nutritional deprivation. Psychologically the inevitable violation of extreme restraint heightens negative mood, activates all-or-none thinking and disinhibits attempts to control what one eats which ultimately leads to a binge eating episode (abstinence-violation effect, Grilo & Shiffman, 1994).

Assuming that restraint is the cause for binge eating behavior, persons with BED should have moderate to high scores on measures of dietary restraint and the scores should be higher than in individuals without binge eating problems. Several studies have assessed dietary restraint in BED. Most of them found only moderate restraint scores which did not differ significantly from NBED controls (Marcus et al., 1988; Masheb & Grilo, 2000; Pinaquy et al., 2003; Wilson et al., 1993; Yanovski, 1993; Yanovski & Sebring, 1994; Zwaan, 2001).

Affect-related models derived from BN (i.e. the trade-off theory Kenardy et al., 1996, escape from self-awareness model Heatherton & Baumeister, 1991, masking theory Polivy et al., 1988) propose that in individuals with BED there should be a tendency toward overeating in response to negative emotions in order to cope with them, which is referred to as emotional eating (Arnow et al., 1995).

Yet, little is known about the relations among eating habits (in terms of emotional and restrained eating) and negative affect (in terms of anxiety and depression) in BED as well as about the associations of these variables with eating behavior in a naturalistic environment. Notwithstanding, these relations are important to understand the mechanisms driving binge eating in BED.

The present study was designed to answer the following questions: 1) Which differences in psychopathological features exist between obese women with and without BED? 2) Which differences in eating behavior and eating habits exist between obese women with and without BED? 3) Are there specific relationships between affective symptoms and eating behavior in the obese women with BED?

2.3. Materials and Methods

2.3.1. Participants

The sample consisted of 84 obese female participants (BMI higher or equal to 30 kg/m²) who were recruited via advertisement in newspapers for a larger study on stress induced laboratory eating (Laessle & Schulz, 2009), offering 100€ for participation. 257 women applied for the study. 84 of these women did not participate because of time limitations or personal reasons. 88 women were excluded because they met the following exclusion criteria: (a) age out of the established range of 18-48 (n = 9), (b) BMI < 30 (n = 31), (c) bulimic behaviors or EDNOS other than BED (n = 7). (d) gastrointestinal disorders, thyroid disorders without medical substitution or other medical issues influencing weight or eating behavior (n = 41). These criteria were checked by a physician in a general hospital in Trier, where all interested participants underwent a medical examination before taking part in the study.

For the diagnosis of binge eating disorder the German version of the structured diagnostic interview for DSM IV (SKID-I, Wittchen, Wunderlich, Gruschwitz, & Zaudig, 1997) was carried out by a research psychologist. According to this procedure, 44 obese women were assigned to the NBED and 40 to the BED group. In the BED group, 8 individuals had a subthreshold variant of BED, defined as having a minimum of one binge episode per month, while fulfilling all other DSM-IV criteria (Friederich et al., 2007; Striegel-Moore et al., 2000).

The BED and the NBED group did not differ significantly concerning mean age (BED 31.9 ± 7.9; NBED 34.4 ± 8.8), mean weight (BED 104.2 ± 15.1, NBED 105.5 ± 17.1), mean height (BED 170cm ± 10, NBED 170cm ± 10), BMI (BED 36.7 ± 5.2, NBED 36.9 ± 5.9) and % body fat (BED 37.6 ± 8.7, NBED 37.4 ± 8.0)

2.3.2. Materials

Depressive symptoms were assessed with the German version of the Beck Depression Inventory (BDI-II, Beck & Steer, 2006). Trait anxiety was assessed using the trait scale of the German version of the state-trait anxiety inventory (STAI-T, Laux & Schaffner, 1981). Eating habits (emotional and restrained eating) were assessed using the emotional eating and restrained eating scale of the German version of the Dutch eating behavior questionnaire (DEBQ, Grunert, 1989; van Strien et al., 1986).

For the naturalistic eating behavior, participants completed food diaries on two days at intervals of two weeks. On one day, there was a stress induction with the Trier Social Stress Test (TSST, Kirschbaum et al., 1993) in the morning. The TSST is a standardized protocol to induce social stress and has been shown to provoke psychobiological stress responses. On the other day, there was a neutral condition (reading magazines for the same time as the TSST lasts) in the morning. In the food diaries, food intake was recorded from approximately ten o'clock in the morning until the end of the day. The participants did not have breakfast before starting the food diaries. Mean intake (in KJ) was calculated using a professional german nutrition software (EBISpro, Erhard, 2008) for the stress day and for the neutral day. In addition the mean value of these days was calculated. Mood was assessed directly prior to and immediate after each food intake on 100mm visual analog

scales (VAS), with higher values indicating better mood. The repeated measure of mood allows to investigate changes in mood after food intake.

This study was reviewed and approved by the ethics committee of the University of Trier.

2.3.3. Data analysis

Comparisons between BED and NED groups for the SCID-I variables used chi-squared (or Fisher exact where cell sizes were small) testing and U-tests. Bonferroni adjusted alpha levels were applied. A multivariate analysis of variance (MANOVA) was conducted to compare the means of the two groups (obese BED group vs. obese NBED group) on depression, anxiety and eating habits. For univariate post tests, the Bonferroni was adjusted to an alpha level of $p < 0.01$. The statistical power for the MANOVA, assuming a medium effect size, was .95. Correlation analyses were conducted using Pearson correlation. All analyses were carried out using the Statistical Package for the Social Sciences (SPSS Statistics 17).

2.4. Results

2.4.1. Structured Clinical Interview for DSM-IV

In the BED group there were significant more additional diagnoses than in the NBED group (U-test, $p < .01$). Compared to the NBED group, the BED group revealed more mood disorders (U-test, $p < .01$), more anxiety disorders (U-test, $p < .01$) and more substance-related disorders (U-test, $p < .05$)[5]. Frequency of additional lifetime DSM-IV Axis I diagnosis in the BED and NBED group are depicted in table 1. Particularly, more participants in the BED group had lifetime Major Depressive Disorder (χ^2-test, $p < .01$).

[5] ***Author´s note: When applying the Bonferroni adjusted alpha of .0125, this difference was only tendentially significant (see table 1).***

Table 1: Frequency of additional lifetime DSM-IV Axis I diagnosis in the BED and NBED group

	NBED (n = 43)	BED (n = 41)	Chi^2 *P*- Value
at least one additional disorder	20 (46.5%)	34 (82.9%)	0.000*
at least one mood disorder	14 (32.6%)	26 (63.4%)	0.004*
at least one anxiety disorder	14 (32.6%)	26 (63.4%)	0.004*
at least one substance use disorder	2 (4.7%)	9(22%)	0.019
Major Depressive Disorder (MDE)	14 (32.6%)	25 (61.0%)	0.008**
Dysthymia	1 (2.3%)	4 (9,8%)	0.16
Depressive Disorder NOS (DDNOS)	1 (2.3%)	1 (2.4%)	0.74
Panic Disorder	4 (9.3%)	8 (19.5%)	0.15
Specific Phobia	7 (16.3%)	16 (39.0%)	0.018
Social Phobia	3 (7%)	3 (7.3%)	0.64
Generalized anxiety disorder (GAS)	2 (4.7%)	2 (4.9%)	0.67
Post Traumatic Stress Disorder (PTSD)	3 (7%)	6 (14.6%)	0.22
Obsessive-compulsive disorder	1 (2.3%)	1 (2.4%)	0.74
Alcohol use or dependence	1 (2.3%)	6 (14.6%)	0.048
Illicit drug abuse or dependence	1 (2.3%)	6 (14.6%)	0.048
Somatoform disorders	1 (2.3%)	5 (12.2%)	0.09

*significant on the Bonferroni adjusted alpha of .0125
**significant on the Bonferroni adjusted alpha of .017

2.4.2. Depressive symptoms, anxiety, eating habits and naturalistic eating behavior

The data for depressive symptoms, anxiety and eating habits are depicted in table 2. The overall MANOVA was significant testing all variables simultaneously ($F_{1,75}$ = 8.04, p < .001). Obese women with binge eating disorder were significantly more depressed ($F_{1,79}$ = 11,08, p < .001) and experienced significantly higher levels of anxiety ($F_{1,79}$ = 16.07, p < .001) than women without binge eating disorder.

Concerning eating habits, obese women with binge eating disorder scored higher on emotional eating ($F_{1,82}$ = 35,69, p < .001), whereas no significant differences were detected for the dietary restraint scale.

To identify the most important factors associated with the presence of BED two logistic regressions considering the presence of BED as the dependent categorical variable were conducted. The first one was performed on the independent variables depressive symptoms and trait anxiety. Only trait anxiety emerged as significant predictor of BED (R^2 = .22, p < 0.01). The second one was performed on the independent variables assessing eating behavior (restrained eating and emotional eating). Emotional eating emerged to be of predictive value for BED (R^2 = 0.32, p < 0.01).

Data of the three measures of naturalistic eating behavior are depicted in table 1.2. The overall MANOVA testing all variables simultaneously was significant ($F_{2,73}$ = 6,00, p < .01). Woman with BED consumed significant more food (KJ) on the day after the neutral condition ($F_{1,74}$ = 6.75, p < .05), after the stress condition ($F_{1,74}$ = 8.15, p < .01) and on average of the two days ($F_{1,74}$ = 12.06, p < .001). In general, they reported significantly more binge eating episodes (U-Test, p < .01). In order to test the influence of mood, an ANOVA with mood prior to food intake as dependent variable was conducted for BED/NBED and binge eating over the day (yes/no). For both groups, mood prior to food intake was significantly worse on days with self reported binge eating compared to nonbinge days ($F_{1,77}$ = 6.9, p < .01). Mood before intake did not differ between groups and the interaction effect was not significant. Food intake significantly enhanced mood ($t_{df = 80}$ = -.4283, p < .001). Changes in mood did not differ between the groups or between binge and nonbinge days.

Table 2: Depressive symptoms, anxiety, eating habits and naturalistic eating behavior (Means, standard deviations and p values) for the BED and NBED group

	NBED n = 43		BED n = 41		F-Test
	Mean	SD	Mean	SD	p
Depressive symptoms (BDI)[1]	10.9	9.4	18.2	10.2	.001*
Anxiety (STAI) [1]	41.6	11.5	51.8	11.2	< .001*
Emotional Eating	27.0	7.5	36.9	7.6	< .001*
Restrained Eating	28.8	7.1	27.8	6.8	.615
food intake on the neutral day[2]	7517.92	3417.72	9841.17	4346.69	.011**
food intake on the stress day[2]	7377.25	2571.98	9694.58	4329.56	.006**
average of both days[2]	7447.58	2146.80	9767.87	3544.35	001**
mood prior to food intake on binge day	4.31	.81	4.52	.73	
mood prior to food intake on nonbinge day	5.08	.70	4.78	.81	

[1] n = 42 in the NBED group and n = 39 in the BED group for these variables
[2] n = 39 in the NBED group and n = 38 in the BED group for these variables
*significant on the Bonferroni adjusted p level of .01
** significant on the Bonferroni adjusted p level of .0.017

2.4.3. Relation of depressive symptoms, anxiety and eating habits in BED

In the BED group depressive symptoms were significant related with restrained eating (r = -.52, p < .001) but not with emotional eating (r = .27, n.s.[6]). In the NBED group, depressive symptoms were significantly related to emotional eating (r = .47, p < .01) but

[6] ***Author´s note: The p value was lower than .1 which can be regarded as tendentially significant.***

not with restrained eating (r = .08, n.s). In both groups, anxiety was significantly related to emotional eating (for the BED group: r = .34, p < .05, for the NBED group r = .53, p < .01). There was no significant correlation between anxiety and restrained eating in the two study groups.

To determine whether the relationships between depressive symptoms and restrained eating or between depressive symptoms and emotional eating differed significantly between the BED and NBED group, two models of moderated linear regression were tested. Restrained eating and emotional eating respectively was used as dependent variable and depression, BED/NBED group and the interaction term (depression x BED/NBED group) as the factors of the analysis. For restrained eating, the interaction term depression x BED/NBED group was a significant factor (p < .01) exhibiting that the link between depressive symptoms and restrained eating differed between the two groups. The interaction term was not significant for emotional eating as dependent variable, suggesting that the relation among depressive symptoms and emotional eating did not differ significantly according to the presence of BED[7].

2.4.4. Relation of depressive symptoms, anxiety and naturalistic eating behavior

In the BED group but not in the NBED group, depression was significantly related to caloric intake after stress (r = .37, p < .05 vs. .r = .03, n.s.). In a subsequent moderated linear regression model with caloric intake after stress as dependent variable the interaction term depression x BED/NBED group was tested. There was a tendency for a stronger link between depressive symptoms and intake after stress in the BED group than in the NBED group (p = .065).

Anxiety was significantly related to caloric intake after stress in the whole study group (r = .23, p < .05) but not in the BED (r = .26, n.s.) or NBED subgroups (r = -.06, n.s.). In a moderated linear regression model, the interaction term anxiety x BED/NBED group was tested (with caloric intake after stress as dependent variable). There was a tendency for a

[7] ***Author´s note: In a respective analyses with emotional eating as dependent variable, the interaction term axiety x BED/NBED group was not significant (p = .412).***

stronger link between anxiety and intake after stress in the BED group than in the NBED group (p = .065).

2.5. Discussion

Major findings are as follows: 1) As expected, the BED subjects in our population exhibited higher levels of comorbidity (in particular mood disorders, anxiety disorders and substance-related disorders, ***see footnote 5***), higher depressive symptoms, trait anxiety, external and emotional eating scores than NBED subjects. Among these variables, regression analyses based on correlational data revealed that anxiety and emotional eating were significant predictors for BED status. 2) In the BED group, depressive symptoms were ***not***[8] significantly related to emotional eating. The relationship between depressive symptoms and restrained eating differed significantly between the two groups according to the presence of BED. Only in the BED group, depressive symptoms were negatively related to restraint. 3) In the BED group, depressive symptoms were significantly related to food intake on the stress day. This relationship tended to be stronger in BED than NBED. 4) Anxiety was significantly related to emotional eating. The relationship between anxiety and food intake on the stress day also tended to be stronger in the BED group. 5) Food intake significantly enhanced mood. Mood was worse on the days with self-reported binge eating episodes than on nonbinge days.

Before interpreting the results some methodological limitations of the present study should be considered. For some analysis (i.e. correlation analysis in the subgroups of BED and NBED individuals) our sample size was relatively small. This requires a replication with larger samples. The findings also seem to be restricted to females, because only women were included in the sample. On the other hand, Tanofsky et al. (1997) did not find sex differences for BED with regard to eating patterns and psychological characteristics. Therefore, our results might also be transferred to a male population, although this should be investigated in further studies.

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[8] ***Author´s note. In the original version, this important "not" was mistakenly missing. As mentioned in the results section, this correlation was not significant, although a statistical tendency was observed.***

The finding of a higher degree of comorbidity (in particular mood disorders, anxiety disorders and substance-related disorders, ***see footnote 5***), higher depressive symptoms and trait anxiety in BED compared to NBED individuals is in line with earlier studies (for depressive symptoms and anxiety, see Allison, Grilo, Masheb, & Stunkard, 2005; Antony, Johnson, Carr-Nangle, & Abel, 1994; Fassino, Leombruni, Piero, Abbate-Daga, & Giacomo Rovera, 2003; Gladis et al., 1998; Isnard et al., 2003; Pinaquy et al., 2003; Ricca et al., 2009; Telch & Stice, 1998). Higher prevalence of lifetime Axis I comorbidity in individuals with BED than in NBED individuals were also found in treatment seeking groups and/or normal weight populations (Grilo et al., 2009a; Hudson et al., 2007; Javaras et al., 2008; Specker et al., 1994; Telch & Stice, 1998; Wilfley et al., 2000) and in non-treatment-seeking samples (Bulik et al., 2002; Yanovski et al., 1993a) but not in a recent study in overweight binge eaters in a treatment seeking sample (Ricca et al., 2009). Our results on emotional eating support the view that the tendency to eat in response to negative mood states is not related to obesity per se but is an important feature of BED. This has been confirmed in earlier studies on emotional eating in overweight/obese subjects with BED (Eldredge & Agras, 1996; Pinaquy et al., 2003; Ricca et al., 2009). The findings that in the regression analyses anxiety and emotional eating were significant predictors for the presence of BED underline the significance of these characteristics in BED. Our results on emotional eating and eating behavior in the natural environment provide further support for the validity of the BED construct. In contrast to le Grange (2001) and in accordance to Engel et al. (2009), BED participants in our study reported more binge eating episodes than NBED participants. Also they consumed more food (in KJ) on both assessment days.

Affect-related theories state that binge eating is used for coping with negative affect, e.g. by providing comfort and/or distraction from negative affect (Heatherton & Baumeister, 1991; Kenardy et al., 1996; Polivy et al., 1988). Binge eating has therefore been hypothesized to be a dysfunctional attempt to compensate the frequent confrontation with negative mood states (Engel et al., 2009). Data of our food diaries support this view: Mood was significantly worse on the days with self reported binge eating compared to nonbinge days. Food intake significantly enhanced mood.

Beyond their individual significance for BED, anxiety and emotional eating were strongly related. There was a significant relationship between anxiety and food intake on the stress day in the BED group which tended to be stronger than in the NBED group. This gives support for affect-related theories on BED.

Concerning restrained eating, we did not find differences between obese binge eaters and obese non- binge eaters, which is in line with other studies (Marcus et al., 1988; Masheb & Grilo, 2000; Pinaquy et al., 2003; Wilson et al., 1993; Yanovski, 1993; Yanovski & Sebring, 1994; Zwaan, 2001). Nevertheless, our data show that the co-occurrence of depressive symptoms and restrained eating has particular adverse effects for individuals with BED. Only in the BED group, depressive symptoms were negatively related to restraint. This relationship differed significantly between the two groups according to the presence of BED. These results are in line with the disinhibition hypothesis (Herman & Mack, 1975; Yanovski & Sebring, 1994) and suggest that the self control of BED individuals can be temporarily released by depressive symptoms, which in turn increases the likelihood of binge eating. This conclusion is corroborated by our results on naturalistic eating behavior where we found a positive relationship between depressive symptoms and food intake on the stress day in the BED group, which was significantly stronger than in the NBED group.

We have shown that obese women with BED differ substantially from women with the same weight problems in the absence of an eating disorder. It is clear from our results that BED is a clinically significant condition that is distinct from typical obesity. This supports the existence of BED as a specific subtype in obesity, which deserves to be considered a form of psychopathology.

For BED treatment, it is important to note that increased restraint is not related to binge eating disorder in BED. Treatment should focus on emotional eating behavior. Especially eating in response to depression and anxiety should be explored carefully. Comorbidities must be considered in the treatment of BED.

3. STRESS-INDUCED LABORATORY EATING BEHAVIOR IN OBESE WOMEN WITH BINGE EATING DISORDER

3.1. Abstract

Aim of the study was to compare the microstructural eating behavior of obese patients with and without binge eating disorder (BED) after stress induction in laboratory. 71 female subjects were investigated (mean BMI 36.9). 35 fulfilled criteria for BED. A 2 x 2 factorial design with repeated measurement (stress vs. no stress) on the second factor was applied. Stress was induced by the Trier Social Stress Test (TSST) and chocolate pudding served as laboratory food. Variables of eating behavior were measured by a universal eating monitor (UEM). Only in participants with BED stress was associated with an increase in the initial eating rate and a diminished deceleration of eating at the end of the meal. Generally, BED subjects ate with larger size of spoonfuls during the laboratory meal than NBED controls. The eating behavior of obese patients with binge eating disorder seems to be significantly affected by stress. The stress-induced eating behavior of BED patients is characterized by a stronger motivation to eat (indicated by a fast initial eating rate) as well as by a lack of satiety perception (indicated by less deceleration of eating rate).

3.2. Introduction

Binge eating disorder (BED) is defined in the fourth edition (text revision) of the Diagnostic and Statistical Manual (DSM-IV-TR, 2000) by binge eating episodes at least at two days per week for 6 months. Binge eating episodes are characterized by the intake of large amounts of food in a discrete time period and loss of control over eating. In addition, binge eating episodes are associated with behavioral changes in eating such as eating much more rapidly than normal, eating until feeling uncomfortably full or eating large amounts of food when not physically hungry. In contrast to bulimia nervosa, BED subjects do not practise compensatory behaviors such as vomiting or the misuse of laxatives after a binge episode.

Although in day-to-day clinical practice a generally accepted category (Dingemans, Bruna, & van Furth, 2002), BED is included in the appendix for diagnostic categories requiring further study in DSM-IV. When applying DSM-IV-TR criteria, the lifetime prevalence of BED in community samples is about 2-5% (Dingemans et al., 2002; Hudson et al., 2007;

Spitzer et al., 1993c; Zwaan, 2002). In clinical samples with participants in weight control programs, up to 30% suffer from BED (Spitzer et al., 1993c).

BED is associated with obesity. Although a causal link has not been established, longitudinal studies suggest that BED leads to weight gain and obesity (Devlin, 2007). However, there is evidence that individuals with BED differ from individuals who are just obese (Hilbert, 2005). Laboratory studies can give objective support to the discrimination of BED from obesity and therefore to the validity of the BED diagnosis (Wonderlich et al., 2009). They can also give insight in underlying mechanisms of the eating disorder.

Many laboratory studies show that individuals with BED tend to eat more calories than weight matched individuals without BED in a variety of different paradigms (Wonderlich et al., 2009). It remains unclear from these studies, why individuals with BED consume more calories than NBED. Biological studies found correlates in individuals with BED which are related to increased hunger, such as reduced serotonin transporter binding (Wadden et al., 1993) and increase of the regional cerebral blood flow in the left hemisphere under food exposure (Karhunen et al., 2000). Other studies have suggested a decreased satiety response in BED. Geliebter and Hashim (2001) found a higher gastric capacity in obese binge eaters compared to NBED individuals which may lead to a decreased satiety response. In line with that, Sysko, Devlin, Walsh, Zimmerli, & Kissileff (2007) reported that BED was associated with less fullness following food intake.

Increased hunger or a disturbance in the satiation process should be observable in characteristics of the eating style (e.g., a higher eating rate at the beginning of the meal or a less pronounced slowing down of the eating rate at the end of the meal (Kissileff et al., 1982).

Although not explicitly investigated, in a study of Yanovski et al. (1992) the rate of caloric consumption per minute (measured as total amount consumed / duration of the meal) was not higher in obese participants with BED compared to obese NBED individuals when they were either asked to "eat normal" or to “binge eat and let themselves go and eat as much as they could". It remains unclear if this finding can be replicated or if it is due to the specific instructions given. Also, the overall eating rate does not give insight into processes

of hunger and satiety. Therefore it is necessary to investigate the microstructure of eating in more detail with variables that reflect these processes.

There is also a lack of information, which stimuli will induce an aberrant eating pattern in BED patients. The most well established antecedent for binge eating is negative mood (Hilbert, 2005). There is also evidence that experience of daily stress is followed by episodes of binge eating (Crowther, Sanftner, Bonifazi, & Shepherd, 2001). Furthermore, Gluck et al. (2004) showed that after a physical stressor (cold pressure test), obese BED participants reported higher levels of hunger and stronger desire to binge eat than obese NBED participants. Empirical evidence supporting the selfish brain theory point to a close relationship between psychosocial stress and food intake (Peters, Kubera, Hubold, & Langemann, 2011). The role of psychosocial stress and food intake in Binge Eating Disorder is, however, unclear.

In a previous study we investigated the role of a psychosocial stressor, the Trierer Social Stress Test (TSST, Kirschbaum et al., 1993), on the microstructure of eating in individuals with BED compared to NBED under the instruction "to eat as much and as long as you like" (Laessle & Schulz, 2009). From a non stress to a stress condition, individuals with BED showed a greater increase in average eating rate ($p < .01$) and a different change in acceleration/deceleration at the end of the meal ($p < .04$). Unfortunately, the sample size in the above mentioned study was relatively small.

The present study therefore aimed to further investigate the effect of a psychosocial stressor on the microstructure of eating behavior (in particular variables which indicate hunger and satiation) in obese women with and without BED with a greater sample size. Stress was hypothesized to change the microstructural characteristics of eating behavior specifically in obese women with BED.

3.3. Methods

3.3.1. Participants

The sample consisted of 71 obese female participants (BMI higher or equal to 30 kg/m²) who were recruited via advertisement in newspapers, offering 100€ for participation. 257

women applied for the study. 84 of these women did not participate because of time limitations or personal reasons. 88 women were excluded because they met the following exclusion criteria: (a) age out of the established range of 18-48 (n = 9), (b) BMI < 30 (n = 31), (c) bulimic behaviors or EDNOS other than BED (n = 7), (d) gastrointestinal disorders, thyroid disorders without medical substitution or other medical issues influencing weight or eating behavior (n = 41). These criteria were checked by a physician in a general hospital in Trier, where all interested participants underwent a medical examination before taking part in the study. 14 participants were excluded because it was not possible to analyse the UEM data in at least one condition. All participants stated to like chocolate pudding before taking part in the study.

For the diagnosis of binge eating disorder the German version of the structured diagnostic interview for DSM IV (SKID-I, Wittchen et al., 1997) was carried out by a research psychologist (Dipl. Psych., equivalent to master's degree in psychology) and supervised students who were all thoroughly trained. According to this procedure, 36 obese women were assigned to the NBED and 35 to the BED group. In the BED group, 6 individuals had a subthreshold variant of BED, defined as having a minimum of one binge episode per week, while fulfilling all other DSM-IV criteria (Striegel-Moore et al., 2000).

3.3.2. Design

A 2 x 2 factorial design was applied. Factor I was "comparison group" (BED vs. NBED). Factor II was a repeated measurement factor (stress vs. no stress). The repeated laboratory tests took place within 4 weeks, with at least one week between the two investigations.

3.3.3. Procedure

Before participating, subjects received detailed written information about the study and signed written consent. Subjects arrived in the laboratory of the University of Trier between 9.00 and 10.00 a.m. after overnight fast. On the morning of each test session, participants consumed a standardized food of half a salami or cheese sandwich to make stomach fullness equal. The energy of the sandwich half was about 650 kJ for salami (fat%: 22, proteine%: 14, carbohydrate%:24) and 643 kJ for cheese (fat%: 22, proteine%: 14, carbohydrate%: 23). Participants were told that we are interested in finding out how stress influences the feeling of satiation while eating a chocolate pudding. For this purpose,

after a stressor or a neutral condition they can eat from the pudding as much and as long as they would like. Stress was induced by the Trier Social Stress Test (TSST, Kirschbaum et al., 1993), a standardized protocol which contains a stress anticipation phase and a stress phase with a free speech assignment and subsequent performance of a mental arithmetic. The TSST is a validated tool to provoke psychobiological stress responses. In the neutral condition reading newspapers was provided for the same time as the TSST lasts. The sequence of stress and neutral condition was counterbalanced. Before and after the TSST and the neutral condition respectively, participants made subjective ratings of feeling stressed on 100 mm visual analogue scales (VAS, 0: not at all, 100: maximum).

This study was reviewed and approved by the ethics committee of the University of Trier in may 2006.

3.3.4. Measurement of eating behavior

The eating behavior was measured using a universal eating monitor (UEM), based on Kissileff, Klingsberg and van Itallie (1980). The UEM consists of a desk that is equipped with a hidden electronic scale which records weight changes on the plate every 0.5 seconds. A special software to compute the signals from the scales produces a cumulative intake curve (x-axis: time; y-axis: amount eaten in g), but also secondary measures, which can be used as descriptors of intake behavior (Kissileff et al., 1982). These measures are: initial eating rate (IER, g/sec) which is the eating rate at the beginning of the meal and reflects the motivation to eat or hunger, change of eating rate (CER, g/sec² x 1000) which is the change of the eating rate at the end of the meal (deceleration reflects satiation, less deceleration at the end of the meal points to less satiation), average eating rate (AER, g/sec) which is the eating rate over the whole meal, size of spoonfuls (SF, g), frequency of spoonfuls (FSF, spoonfuls/sec) and total amount of intake (TI, g). Our instrument is described in detail in Hubel et al. (Hubel, Laessle, Lehrke, & Jass, 2006) and is of proven reliability (Laessle & Geiermann, 2012). We modified the original technical equipment with a more refined electronic scales and a desk which has been constructed to be free of any vibration.

Yanovski et al. (1992) found that participants with BED ate more dessert than the obese control group. For this reason and because of specific demands of the UEM on the

consistency of food, the laboratory food consisted of chocolate pudding (energy/100 g: 658 kJ; fat%: 9.1, protein%: 3.1, carbohydrate%: 15.8). The initial quantity in a bowl on the balance was 500 g, but all participants were free to take further helpings. Before the start of the meal, participants were asked to make subjective ratings of hunger and after the meal of hunger and palatability on 100 mm VAS.

3.3.5. Statistical analysis

Following Kissileff et al. (1982) and Bobroff and Kissileff (1986) 2 sets of variables derived from the eating monitor data were defined and analysed by MANOVA for repeated measurement (O'Brien & Kaiser, 1985).

The first set includes initial eating rate (IER) and change of eating rate (CER). It covers continuous variables related to motivation to eat and to aspects of satiety regulation.

The second set includes total intake (TI), size of spoonfuls (SSF), and frequency of spoonfuls (FSF) which reflect non continuous characteristics of eating behavior related to palatability of food.

For each set of variables a global test of the group X stress interaction for all dependent variables simultaneously was conducted. Repeated measurement was done on the second factor stress (stress vs. neutral condition). If the global test was significant, tests for each variable separately were interpreted. All analyses were adjusted for age differences between the comparison groups.

In a previous study significant stress dependent changes in eating rate, frequency of spoonfuls, and change of eating rate were found (Laessle & Schulz, 2009). These results were used to generate directed hypotheses for these variables in the present study, which were tested by one-tailed significance tests according to Bortz & Doering (2006).

The analyses were carried out using the Statistical Package for the Social Sciences (SPSS 17.02).

3.4. Results

3.4.1. Anthropometric characteristics

Anthropometric characteristics of the two groups are depicted in table 3. For these data, no significant group differences were detected.

Table 3: Anthropometric characteristics (means and standard deviations) of the sample

	BED (n = 35)	NBED (n = 36)
age (years)	32.4 (8.0)	35.6 (8.4)
weight (kg)	104.0 (16.1)	105.0 (17.7)
BMI	37.0 (5.5)	36.9 (6.1)

3.4.2. Subjective ratings

The TSST induced a significant increase in the feeling of being stressed in both groups ($F_{1,69}$ = 139.27 p < .001). The groups did not differ significantly in their self-reported stress level after the stress or the neutral condition.

Furthermore, the comparison groups did not differ significantly regarding their ratings of hunger neither in the stress nor in the neutral condition. Mean hunger ratings were 3.75 (*SD* = 2.3) in the BED and 3.19 (*SD* = 1.9) in the NBED group.

There were no significant main effects or interaction effects regarding the ratings of satiety after the test meal. Mean satiety ratings were 6.8 (*SD* = 1.61) in the BED group and 6.68 (*SD* = 1.53) in the NBED group.

3.4.3. Eating behavior

Data for the continuous variables of eating behavior in the laboratory are depicted in table 4.

Table 4: Means and standard deviations of continuous eating characteristics for obese women with and without BED dependent on a laboratory stressor

	BED		NBED	
	No Stress	Stress	No stress	Stress
CER (g/sec^2 x 1000)	2.1 (6.0)	.04 (3.5)	.17 (2.3)	.74 (3.2)
IER (g/sec)	.33 (1.8)	.53 (1.6)	.53 (1.5)	.47 (1.6)
AER (g/sec)	1.4 (.6)	1.4 (.7)	1.4 (.6)	1.4 (.7)

CER: change of eating rate: the lower the value, the less deceleration at the end of the meal
IER: initial eating rate
AER: average eating rate

The global test for the group x stress interaction testing all variables simultaneously was significant. ($F_{3,62}$ = 2.81, p < .05, *partial eta²* = .12). Univariate analysis revealed a significant interaction effect for IER ($F_{1,64}$ = 2.99, p < .05, *partial eta²* = .05) and CER ($F_{1,64}$ = 5.22, p < .02, *partial eta²* = .08).

Participants with BED showed an enhanced initial eating rate (IER or motivation to eat) from the non-stress to the stress condition, while participants without BED showed a reduced initial eating rate in the stress compared to the non-stress condition.

BED participants showed less deceleration in the stress than in the non-stress condition, while participants without BED showed a stronger deceleration in the stress than in the non-stress condition.

Data for the non continuous variables of eating behavior in the laboratory are depicted in table 5.

Table 5: Means and standard deviations of non continuous eating characteristics for obese women with and without BED dependent on a laboratory stressor

	BED		NBED	
	no stress	stress	no stress	stress
FSF (per sec)	.11 (.05)	.12 (.05)	.12 (.05)	.13 (.05)
SSF (g)	13.2 (4.6)	12.6 (4.3)	11.3 (2.8)	10.8 (2.7)
TI (g)	282.6 (130.2)	241.4 (122.5)	276.7 (112.6)	243.4 (136.7)

FSF: frequency of spoonfuls
SSF: size of spoonfuls
TI: total intake

The global test for the group x stress interaction testing all variables simultaneously was not significant, indicating that there are no stress-specific differences between obese women with and without BED in total intake, frequency of spoonfuls and total intake.

Although observed on the descriptive level, both groups showed enhanced FSF and lower SSF and TI under stress, the main effects of stress were not significant for these variables.

For size of spoonfuls, a significant main effect of diagnostic group ($F_{1,68} = 5.5$, $p < .02$, *partial eta²* = .08) emerged, whereas patients with BED ate in both conditions with larger spoonfuls.

3.5. Discussion

The current study found stress-induced changes in microstructural characteristics of eating behavior specifically for obese subjects with BED when asked to eat as much and as long as they like. Only in participants with BED, stress was associated with an increase in the initial eating rate and a diminished deceleration of eating at the end of the meal. Independent of being stressed, BED subjects showed a larger size of spoonfuls during the laboratory meal.

According to Kissileff et al. (1982) and Guss & Kissileff (2000) the initial eating rate reflects underlying physiological processes which facilitate eating and hunger as its associated cognitive correlate. Change of eating rate or the rate of deceleration reflects inhibitory processes which control eating over the meal and its cognitive correlate, satiation. Our data point to a stress-induced disturbance in both processes specifically in obese women with BED compared to obese women without BED.

These disturbances would be in line with studies which found biological correlates in individuals with BED related to increased hunger (Karhunen et al., 2000; Wadden et al., 1993) or diminished satiety (Geliebter & Hashim, 2001). In addition, the results are in line with psychological studies using self-report measures. Latner et al. (2009) assessed subjective ratings on variables, that are related to a stronger motivation to eat or hunger. Women with BED reported a greater desire to eat, prospective consumption, excitement about eating, and desire for a meal and dessert before a buffet-style test-lunch compared to weight-matched controls. In a study of Sysko et al. (2007) participants made ratings of hunger and fullness on visual analogue scales after every 75-g increment of food. They found, that in comparison to obese or normal-weight controls, patients with BED consumed significantly more food to reach a similar level of fullness or hunger. The authors conclude that the process of eating in BED appears to be characterized by a diminished increase in fullness particularly during the latter portion of the meal, which is in line with the current study.

Another study (Samuels, Zimmerli, Devlin, Kissileff, & Walsh, 2009) found no differences between obese BED subjects and obese controls neither in hunger ratings nor in ratings of fullness. These results do not necessarily argue against our interpretation, because the study did not use objective measurement of intake characteristics such as a universal eating monitor or the induction of stress. Interestingly, in the current study, there is discrepancy between the objective measurement of the UEM and subjective ratings of hunger and satiety, which did not differ between the groups or conditions. The UEM is a tool which is proved to give reliable and adequate measures for an integrated characterization of eating behavior during a meal in humans (Hubel et al., 2006; Laessle & Geiermann, 2012; Westerterp-Plantenga, 2000). Therefore it can be concluded from our data that there is a stress-induced disturbance regarding the motivation to eat and satiation processes. The lack

of correspondence in the subjective ratings might point to a deficit in the perception of hunger and satiety signals in individuals with BED.

The finding of a stress X group interaction effect in change of eating rate is in line with our previous study (Laessle & Schulz, 2009). On the contrary, the interaction effect in initial eating rate could not be found in the former study, presumably due to lack of statistical power. In the current study, there was no stress-induced change in the average eating rate for the BED or NBED group. This is contradictory to our previous finding but in line with a study of Yanovski et al. (1992) in which the rate of caloric consumption per minute (measured as total amount consumed / duration of the meal) was not higher in obese participants with BED compared to obese NBED individuals. As mentioned above, the overall eating rate does not give insight into processes of hunger and satiety and might be less appropriate to indicate disturbances in these processes.

A somewhat unexpected finding is the lack of a main effect of group (BED vs. NBED) on total meal intake. Many studies found that obese individuals with BED ate more than weight-matched controls without BED under both binge and non-binge instructions (Walsh & Boudreau, 2003). However, there are studies who also did not find significant differences between these groups concerning the amount of food intake (Gosnell et al., 2001; Lattimore, 2001). There are some factors which might account for the lack of a main effect of group in the current study. These are variety of food/number of dishes presented, amount of food presented and meal instruction. Previous studies have shown that increasing the variety and amount of food presented in laboratory meals increases the amount eaten (Cohen, 2008; Kral & Rolls, 2004). Rolls, Morris, & Roe (2002) showed that portion size of food affects energy intake in normal-weight and overweight men and women. In their study, subjects consumed 30% more energy when offered the largest portion (1000g) than when offered the smallest portion (500g) of macaroni and cheese. Compared to this study, the initial amount of 500g chocolate pudding in the current study is a small portion size. Furthermore, in the current study, there was no variety in the presented food. Most studies demonstrating differences in meal intake between individuals with BED and NBED controls used multiple-item-meals (Telch & Agras, 1996a, Telch & Agras, 1996b), or macaroni and cheese in much bigger amounts (Sysko et al., 2007) or a single-item meal with unlimited amount (Anderson et al., 2001). The few studies that

found no difference in total intake between BED and NBED individuals used either a single-item meal of 250g ice cream (Lattimore, 2001) or one or two binge foods of individual amount (Gosnell et al., 2001). The initial amount might serve as a cue for an appropriate meal size, providing expectations of when the meal would end. Therefore it is quite possible, that the initially relatively small single-item test meal in the current study was not sufficient to reveal the predicted differences between the groups. Another limitation of the laboratory setting is related to the fact that diagnostic criteria of BED include eating alone due to embarrassment of overeating. In a single-item test meal, the amount eaten is more obvious than in a multiple-item test meal. This might also have contributed to the lack of a main effect of group concerning intake. It is also possible that the instruction "to eat as much and as long as you like" contributes to the non-significant differences between the groups (BED vs. NBED). Participants in the current study did not receive instructions such as "to binge eat", "to eat as much as you can" or "to eat normal". These instructions, used in the above mentioned studies, can provide a priori expectations of what amount is appropriate (Samuels et al., 2009). The results in the current study might be less confounded by these instructions.

There are further limitations that should be considered, when interpreting the results. The study was undertaken in a study laboratory, therefore the artificial laboratory situation must be considered. Although this is also an advantage, because we were able to control and standardize all variables of interest, ecological validity might be questioned. However, there are hints that eating monitor data from the laboratory correspondent well to everyday eating behavior in patients with binge eating disorder (Guss et al., 2002). Our sample was exclusively female and was drawn from the community. Caution is therefore warranted in generalising from this community sample to patients with binge eating disorder and to men with the condition. The sample size is relatively small and although significant effects were obtained, there is need for replication.

In summary, this study found a stress-induced increase in the initial eating rate and a diminished deceleration of eating at the end of the meal particularly in participants with BED. This points to a stress-related disturbance in physiological processes contributing to the development of hunger and satiety among patients with BED, but additional studies are needed to better understand the role of these processes in the maintenance of binge eating.

Subjective ratings suggest there may be a deficit in the perception of hunger and satiety signals in individuals with BED. This view is supported by the perspective that BED individuals become "disconnected" from internal experience and show desensitization to internal cues which are related to normal satiety processes (Kristeller & Wolever, 2011). Therefore our results support the utility of clinical interventions, who focus on the development of an awareness of hunger and satiety cues during a meal, such as mindfulness-based eating awareness training for treating binge eating disorder (MB-EAT, Kristeller & Wolever, 2011).

4. NO EVIDENCE OF INCREASED CORTISOL STRESS RESPONSE IN OBESE WOMEN WITH BINGE EATING DISORDER

4.1. Abstract

Increased cortisol levels after stress have been associated with excessive food intake during binges in obese women with binge eating disorder. The present study tried to replicate these findings in a sample of obese women with compared to obese women without binge eating disorder. 20 women with a mean BMI of 37,75 kg/m^2 were studied. Salivary cortisol was sampled before and after a socially evaluated cold pressure test at 8 time points. Both comparison groups showed the expected cortisol increase after the stressor ($p < .03$), but no significant differences between groups emerged ($p = .96$). These results do not support an increased stress reactivity of HPA in binge eating disorder. Instead they would favour psychological explanations of binges, focusing on diminished perception of satiety.

4.2. Introduction

Obesity, defined as a BMI > 30 has become a major health problem in the US but also in Germany. A subset of obese individuals have binge eating disorder (BED) which is characterized by the consumption of objectively large amounts of food and a sense of loss of control over eating. Cortisol is involved in appetite regulation and energy balance (Dallman et al., 2004). Therefore, as a biological basis of BED, hyperactivity of HPA indicated by heightened stress response of cortisol has been hypothesized (Gluck, 2006). To date, there is only one study supporting this assumption (Gluck et al., 2004), using a cold pressure test as stressor. The present study tried to replicate these results with a comparable methodology in obese women with and without binge eating disorder.

4.3. Methods

4.3.1. Sample characteristics

20 patients were recruited for cortisol assessment after stress during a follow-up study of another research project dealing with laboratory eating behavior in binge eating disorder (Laessle and Schulz, 2009). During this project, diagnostic assessment of BED and NBED had been made using a structured clinical interview (SKID, Wittchen et al., 1997). Mean age was 32 ± 9,1years for BED and 38 ± 10,3 years for NBED and was not significantly different between groups.

Mean BMI was 37,9 ± 5,8 kg/m^2 for BED and 37,6 ± 7,4 kg/m^2 for NBED, and did not differ between groups.

The laboratory test took place between 15,00 and 16,00 p.m.. To exclude low blood glucose, which would prevent an adequate cortisol response, all patients were instructed to eat a standardized "Muesli" bar 1hour before the test. The bar contained 50g, 217 kcal, 21,5g carbohydrate , 8,5g fat, 3,5g protein.

4.3.2. Stressor

Stress was induced by a socially evaluated cold pressure test (SECPT), described in detail by Schwabe, Haddad and Schächinger (2008).

Saliva cortisol was sampled by Salivetten (Sarstedt, Nümbrecht) at 8 measurement points: before stress, after stress, +10min, + 20min, + 30min, + 45min, + 60min, + 90min.

4.3.3. Statistical analysis[9]

Multivariate analysis of variance for repeated measurement (MANOVA) was calculated, using cortisol values after stress as within subjects factor. We used the statistical package for the social sciences (SPSS 17.0).

4.4. Results

The time course of salivary cortisol after the stressor is depicted in table1[10].

For both comparison groups there was a significant increase of cortisol after the stressor (MANOVA: $F_{3,12} = 5{,}9$; $p < .03$), adjusted for pre-stress values.

However, cortisol secretion was not different between groups (MANOVA: Interaction Diagnosis X cortisol secretion: $F_{7,12} = .26$; $p = .96$).

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[9] ***Author´s note: Statistical analysis were carried out by the co-author.***

[10] ***Author´s note: See also the figure in Appendix C.***

Table 6: Mean values of cortisol after stress in obese women with (BED) and without (NBED) binge eating disorder (means ± SD in nmol/l)

measurement time	BED (n = 7)	NBED (n = 13)
before stress	2,8±1,4	2,4±1,0
after stress	2,6±1,3	2,4±1,0
+ 10min	2,8±1,0	2,9±1,7
+ 20min	3,1±1,4	3,5±2,4
+ 30min	2,6±1,3	2,8±1,7
+ 45min	2,2±1,1	2,2±1,2
+ 60min	2,0±1,0	1,9±0,8
+ 90min	1,6±0,8	1,5±0,7

4.5. Discussion

This study has limitations, that warrant caution, when interpreting the data. The sample size was very small, so that we should replicate the nonsignificant results for differences in HPA activity with more patients. Stress response of cortisol is only one indicator of HPA activity and does not provide information on disturbed feedback regulation of HPA in BED. The study included only women and therefore does not allow a generalisation to men with BED. Keeping these limitations in mind, our preliminary data do not support a heightened stress reactivity of HPA in women with BED.

An alternative explanation of binges in BED is a disturbance in the perception of satiety, due to specific alterations in the microstructure of eating. After stress patients with BED eat faster with a greater frequency of spoonfuls (Laessle & Schulz, 2009). Both characteristics of eating behavior have influence on the process of satiation and promote a too late perception of fullness, which in turn results in the consumption of large amounts of

food. This hypothesis is further supported by Gluck et al. (2004), showing higher hunger ratings for BED patients during stress.

5. GENERAL DISCUSSION

5.1. Overview of the chapter

This chapter will address overall conclusions of the current research as well as implications for further research and treatment of BED. Because it might be helpful to have study limitations and strength in mind before reflecting overall conclusions and implications, they will be mentioned first.

5.2. Limitations of the present research

Our sample was exclusively female and was drawn from the community. Caution is therefore warranted in generalising from this community sample to patients with BED and to men with the condition. As mentioned before, Tanofsky et al. (1997) did not find sex differences for BED with regard to eating patterns and psychological characteristics. Therefore, our results might also be transferred to a male population, although this should be investigated in further studies. Recruitment was carried out via advertisements in newspapers and flyers, offering payment for participation. This procedure might have lead to a selection of people reading local newspapers and possibly in need for money. Since it is very difficult to recruit individuals fulfilling BED criteria from the community and because the study was relatively time-consuming for participants, this way of recruitment was nevertheless inevitable. Exclusion criteria (possible participants were excluded if they had medical conditions influencing weight or eating behavior) further reduce the generalizability of our findings, but can also be considered as strength of the study, because possible confounding variables were eliminated.

For some analyses, especially for analysis of differences in cortisol levels and correlational analyses, our sample size was relatively small. This should be kept in mind, particularly when interpreting non significant results. I.e., for correlational analyses of emotional eating and depression in the BED group, power (1-β) was only .53. This means, the accepted type 2 error (β) was .47 and thus more than nine times higher than the accepted type 1 error (α) of .05. Future research should therefore investigate larger samples. In case the a priori calculated sample size exceeds the available resources, the affordable maximum sample size should be specified and an α level with which the ratio of type 2 to type 1 error is

more appropriate should be calculated (compromise power analyses, Faul, Erdfelder, Buchner & Lang, 2009).[11]

In chapter 2, data from a cross sectional correlative study are reported. It is important to consider that this methodology cannot determine causality. Therefore it cannot be determined if i.e. depressive symptoms reduce restraint as stated by disinhibition hypothesis, if reduced restraint leads to increased depressive symptoms or if there is no causal relation at all. Because it was the aim of the current thesis to provide support for specific maintenance factors of BED, interpretations related to these factors or mechanisms were focused. Also predictions made by regression analyses (i.e. that anxiety and emotional eating were significant predictors of BED status) were based on cross-sectional data and do not imply causality. It is also important to consider that the measures of eating behavior (emotional and restrained eating) rely on one's perception of the own behavior and may not be an accurate representation of the actual behavior (Epel et al 2007). Further more it should be kept in mind that anxiety was measured as trait. Trait anxiety reflects the tendency to respond with state anxiety in the anticipation of threatening situations (Laux & Schaffner, 1981). Therefore results should not be misinterpreted in the sense that an actual feeling of anxiety is i.e. related to increased food intake in the natural environment.

Eating behavior in the natural environment was only studied on two days after the laboratory session. Therefore, the food log data provide only a 'snapshot' of naturalistic eating behavior. We decided not to include more assessment days because the laboratory study was demanding and therefore additional, time consuming recording of daily food intake would have probably reduced the number of participants in this study, response rates or quality of the food logs. We did not assess stress or daily hassles in the food diaries for the same reasons. These data would be of interest for the investigation of the stress-eating relationship in the natural environment, but a longer time period would be needed to make valid interpretations. Further, self-reported food intake via food logs is a retrospective assessment method and might be subject to memory and reporting bias. We tried to reduce

[11] For correlation of emotional eating and depression in the BED group (n = 41) of the current thesis, a more balanced β/α ratio of 3 would have resulted in an accepted α level of .01 and a power of .68. Certainly, compromise analyses should not be used post hoc and therefore the interpretation of the current research is based on the conventional α level of .05.

memory bias by instructing participants to fill out the food log immediately after every food intake.

Some disadvantages of the laboratory setting in study 2 and 3 have to be mentioned: First of all, ecological validity must be questioned (see below for further details for study 2). Furthermore, laboratory studies are limited in studying the effects of acute stressors on eating behavior, whereas chronical stressors might have a greater impact on eating behavior (Torres & Nowson, 2007). Additionally, individuals may not be sufficiently stressed in this artificial laboratory environment as compared to stress that is experienced in real world (Torres & Nowson, 2007).

Limitations of our laboratory setting in study 2 are as follows: 1) The UEM can only be used with liquids or semi-solid homogenous food items. Thus, we did not use a multiple-item test buffet which displays natural eating behavior more adequately than chocolate pudding. It has been shown that in contrast to BN, who eat preferably dessert food when binge eating, BED participants eat a full range of foods (Wilfley et al., 2003). 2) The amount of pudding provided might have been too small to reveal the predicted differences between the groups as discussed before (see page 40 ff). 3) The instruction "to eat as long and as much as you like" might have also contributed to the lack of difference in intake, because most of the earlier studies which found such differences used binge instructions (as discussed in detail on page 40 ff). On the other hand the instruction in the current study is more likely to produce natural responses and this might also be considered as strength of the study. Further, binge instructions are not reasonable in our context, because of possible confounding with the effect of the stressor. 4) One of the defining characteristics of binge eating is eating alone because of embarrassment. Therefore, it is possible that women with BED did not eat naturally in our laboratory.

5.3. Strength of the present research

The questions of validity and maintenance of BED are of real clinical importance and also of high relevance with the release of DSM-V in May 2013 in mind. The current research approached these questions from a psychological as well as from a biological perspective. Eating behavior was assessed in laboratory as well as in the naturalistic environment. The novelty of this paper is the investigation of stress-induced changes in characteristics of eating behavior using objective eating monitor data and well validated stressors in the laboratory.

The current research comprised naturalistic as well as laboratory studies on eating behavior of obese BED and non BED. The assessment of eating behavior in the natural environment in study 1 has the advantage of high external validity and generalizability of the findings. The laboratory studies performed in study 2 and 3 have the advantage that all variables of interest could be controlled and that they therefore allow the inference of causal relationship. Study 2 made use of reliable feeding laboratory methodology (Laessle & Geiermann, 2012), which can be considered as strength of the research.

Another strength is the use of validated stress protocols in study 2 (TSST) and study 3 (SECPT). Both stressors included a social-evaluative component (see page 10), which contains the threat of being negatively judged by others. Stressors including this component have shown to elicit the strongest psychological stress responses as well as profound activation of the HPA axis (Dickerson & Kemeny, 2004). Studies with comparable stressors on BED are rare. The only previous study of stress-induced cortisol secretion used a standard CPT without such an evaluative element (Gluck, 2004).

Concerning methodology, there are some further advantages: 1) The within-subjects design in study 2 can be considered as strength because it has the advantage of greater power and reduction of error variance associated with individual differences (Bortz & Doering, 2006). 2) Study 3 used MANOVA, which has more power than area under the curve (AUC), used in a similar study (Gluck, 2004).

5.4. Conclusions of the present research

The current thesis aimed to further contribute to the validity of BED and to support the significance of psychological as well as biological maintenance factors of BED. This was done in order to provide a better understanding for the syndrome of BED, which is of importance to improve treatment.

In summary, the following conclusions can be drawn:

Obese women with BED differ substantially from women with the same weight problems in the absence of an eating disorder in different aspects: 1) BED individuals exhibited higher comorbidity (particularly anxiety disorders and major depression), higher trait anxiety and more depressive symptoms, indicating that these factors are not associated with obesity per se but are important features of BED. 2) Importantly, the eating behavior in the natural environment of obese women with BED differed significantly from that of obese women without BED, with BED individuals showing higher food intake and exhibiting more binge eating episodes. 3) BED subjects differed also in microstructural characteristics of eating behavior from obese NBED: Independent of being stressed, they showed a larger size of spoonfuls during the laboratory meal.

Negative mood precedes binge eating in the natural environment: Mood ratings prior to food intake were lower on binge days compared to non-binge days.

Eating in response to negative affect in order to cope with them (emotional eating) discriminates BED from obesity: 1) Women with BED had significantly higher levels of emotional eating. 2) Emotional eating was a significant predictor of BED diagnosis.

In BED subjects, negative affect is associated with increased caloric intake in the natural environment: 1) Only women in the BED group ate more on the stress day when reporting high levels of depressive symptoms, and this relationship tended to be stronger in the BED group. The correlation coefficient in the NBED group was close to zero. 2) There was a significant positive relationship between anxiety and caloric intake on the stress day in the whole study group. This relationship tended to be stronger in the BED group. The correlation coefficient in the NBED group was close to zero.

Together, these findings support the view that binge eating in the natural environment is used to cope with negative affect as supposed by affect regulation models (Hawkins & Clement, 1984).

The association of negative affect and emotional eating is not stronger in BED subjects[12]. 1) In both BED and NBED group, higher trait anxiety was significantly associated with higher self-reported tendency to eat in response to negative affect. Statistically, the strength of this association did not differ between the two groups. 2) More depressive symptoms were related to higher self-reported tendency to eat in response to negative affect. The correlation was only significant in the NBED group (in the BED group this relationship was only tendentially significant) but statistically, this relationship was not stronger in the NBED group.

Taken together, in the NBED group negative affect was only associated with the self reported tendency to eat in response to negative affect but not with real food intake in the naturalistic environment. A positive association between negative affect and actual food intake was only observable in BED individuals. These differences might be due to the different levels of emotional eating in the two groups, with much lower scores in the NBED group. Thus, although there may be a tendency to eat in response to depression and anxiety in NBED subjects (indicated by the values of emotional eating which were still relatively high in the NBED group[13] and the correlation of emotional eating with anxiety and depression in NBED), this tendency might not as likely result in increased actual intake as in BED individuals.[14]

Restrained eating does not discriminate BED from obesity. Mean restraint scores did not differ between the two study groups. Compared to normal, mean group scores of restrained

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[12] Descriptively, correlations were even higher in the NBED group.

[13] The mean score of emotional eating in the NBED group was higher than 75% (and the mean score in the BED group was higher than 95%) of the scores in a nonclinical reference sample, (van Strien, Frijters, Bergers, and Defares, 1986).

[14] **In line with that, the association between emotional eating and caloric intake on the stress day was descriptively stronger in the BED group ($r = .1$ vs. $r = .05$). The correlation was significant in the whole study group ($r = .219$, $p < .05$) but not in the BED or NBED group alone.**

eating were relatively high[15]. Data of the current research support the view that *the self control of BED subjects can be released by depressive symptoms and stress* as stated by disinhibition hypothesis: 1) Higher levels of depressive symptoms were associated with lower levels of restrained eating only in obese individuals with BED but not in the obese control group. 2) As mentioned above, only in the BED group there was a positive relationship between depressive symptoms and food intake on the stress day[16]. 3) Descriptively, in the non-stress condition BED subjects decelerated even more at the end of the meal than NBED controls. Importantly, after the laboratory stressor, BED subjects almost showed no deceleration at the end of the meal, which points to a disturbance in inhibitory control of food intake in BED or disinhibition (see definition of CER, page 5 and Zandian, Ioakimidis, Bergh & Södersten, 2009). The current results can therefore be interpreted as giving objective behavioral support for stress-induced disinhibition in BED subjects.

With respect to the hypothetical model of stress-induced eating in BED (see figure 1), the following conclusions can be drawn:

Psychosocial stress induces microstructural changes in the eating behavior of obese women with BED: Only in participants with BED, stress was associated with an increase in the initial eating rate and a diminished deceleration of eating at the end of the meal. While BED individuals decelerated their eating rate in the non-stress condition, they ate at an almost linear rate (CER was close to zero) after the stress induction. These findings are of importance because they clearly demonstrate that a psychosocial stressor promotes an adverse eating pattern specifically in BED subjects.

The findings point to a stress-related disturbance in processes contributing to the development of hunger and satiety among patients with BED: 1) The stress-induced increase of the initial eating rate points to an increased motivation to eat or hunger in BED

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[15] The mean restraint score in both the BED and NBED group was higher than 75% of the scores in a nonclinical reference sample (van Strien, Frijters, Bergers, and Defares, 1986).

[16] The relationship between restraint eating and kcal intake on the stress day also differed descriptively between the two groups. In the BED group, there was a negative correlation (r = -.2), whereas in the NBED group there was slightly positive correlation (r = .05).

subjects. 2) The stress-induced diminished deceleration of eating at the end of the meal points to less satiation or less inhibitory control over food intake in individuals with BED.

There is evidence for a deficit in the perception of hunger and satiety signals: 1) Both increase of IER and less deceleration at the end of the meal promote a late perception of fullness in BED subjects. 2) There was a lack of correspondence of subjective hunger and satiety ratings with objective UEM measures, which also suggests a deficit in the perception of hunger and satiety signals in individuals with BED.

Insufficient satiety perception might point towards a lack of interoceptive awareness in BED subjects (van Strien et al., 1986). Lack of interoceptive awareness could also explain the high levels of emotional eating, which may occur in individuals who confuse emotional distress with hunger (Bruch, 1974; van Strien et al., 2005).

Microstructural changes in eating did not lead to increased food intake in the laboratory. We did not find stress-related or general differences between total intake of obese BED and NBED individuals in the laboratory. This finding might be due to limitations of the laboratory setting (see page 55 f. for details), since in the natural environment, we could clearly show that obese women eat more throughout the day and engage in binge eating episodes more frequently. Behavioral characteristics of eating such as CER and IER might have been less affected by these limitations, i.e. because subjects were not aware of this measurement.

There is no evidence for higher cortisol stress-reactivity in BED: Because obese women with BED did not show a higher stress-reactivity in cortisol levels, it is unlikely that cortisol moderates the impact of stress on eating behavior in BED as compared to obesity. Descriptively, there was even a slightly blunted stress-induced cortisol secretion in BED as compared to NBED women in our study (see figure in Appendix C), which is in line with a recent study of Rosenberg et al. (2012), which was published after study 2 was finished (see also page 60). These authors suggest, that instead of a hyperactivation of the HPA there might be even a down-regulation of the HPA in obese BED compared to NBED and normal weight controls as a result of chronic stress.

5.5. Directions for future research

In the naturalistic environment we have shown that in the BED group, depressive symptoms were significantly related to food intake on the stress day. There was also a moderate correlation ($r = .3$) between anxiety and food intake on the stress day. This association was not significant ($p = .12$), which might have been due to a lack of statistical power, since the correlation was significant in the whole study group but close to zero in the NBED group. Further studies should therefore examine the relationship of BED diagnosis, anxiety and depression and food intake with bigger sample sizes. In order to determine the directionality of these relationships, statistical approaches such as the testing of different models through path analysis should be considered. Tested models should also include emotional eating and restraint to determine mechanisms through which negative affect influences food intake and binge eating in BED. Concerning the direction of the relationship between negative affect and food intake in BED, a recent laboratory study using path analysis, which was published after study 1 was finished, suggests that symptoms of depression and anxiety influence binge eating diagnosis which in turn influences food intake (Peterson, 2012).

We have shown that binge eating frequency was higher in obese BED compared to obese NBED individuals and that mood prior to food intake was generally lower on binge days as compared to nonbinge days. This finding may point towards the importance of negative affect for the initiation of binge eating episodes in BED. A more detailed analysis of affect prior to and also after binge eating would be of some interest, especially compared to BN. Treatment of BED is traditionally based on interventions developed for BN (Munsch, 2012). There is, however, evidence for differences in the initiation of binge eating episodes in BED and BN. For example Hilbert & Tuschen-Caffier (2007) reported that BED individuals experience less intense negative mood than BN patients and engage in binge eating also when feeling only moderately negative. There is recent evidence, that binge eating in BED is not triggered by an accumulation of negative affect as suggested in BN but a rather an abrupt breakdown in emotion regulation indicated by a sudden increase in negative affect (Munsch et al., 2012). Obviously, such differences are important for treatment. Therefore, a more detailed analysis of antecedents and also of consequences of binge eating would be beneficial. For instance, different facets of negative affect (i.e.

depression, anxiety, anger, guilt, shame) and also positive affect should be investigated, and assessment should occur at different time points (i.e. at different time points prior to food intake, during binge eating, and at different time points after food intake). This would also help to identify mechanisms that reinforce binge eating behavior in BED.

We have shown that a laboratory stressor can induce changes in the microstructure of eating behavior specifically in obese BED individuals. Further studies should investigate if this finding can be replicated.

Our data on IER and CER pointed to a stress-induced disturbance in psychological or biological mechanisms underlying both motivation to eat and eating inhibition. These mechanisms should further be elaborated. For example, stress-induced secretion of hormones involved in the short-time regulation of food intake, such as ghrelin and CCK secretion should be investigated in BED, in order to better understand the role of physiological appetite regulation under stress in BED. Additionally, reward pathways could also contribute to a lack of inhibitory control of food intake. These could be of interest in BED, because it has been hypothesized that "some obese subjects may experience a greater reward value from food that overrides inhibitory control of food intake and rendering such individuals easily disinhibited" (Guss et al., 2002, p.1027).

Obese BED and obese NBED did not show a higher stress-reactivity in cortisol levels. It is possible, that different HPA axis abnormalities that have been observed in BED and obese subjects are mainly due to excess weight (Lo Sauro, Ravaldi, Cabras, Faravelli, & Ricca, 2008). Therefore, further studies should include normal weight control groups and bigger sample sizes in order to test if possible alterations are related to BED or to obesity per se. These studies should use stressors with social-evaluative components. The above mentioned study of Rosenberg et al. (2012) included a normal weight control group and used the TSST but did not have a much bigger sample size (n = 24). Therefore additional studies are needed. Also longitudinal studies would be useful to test HPA axis-adaptations to chronical stress in BED in more detail.

5.6. Implications for treatment

Based on the current findings, the following conclusions for treatment can be drawn:

Treatment should consider psychiatric comorbidity, specifically anxiety and depression. Since women with BED exhibit more negative affect in terms of depressive symptoms and trait anxiety and show a strong tendency to eat in response to negative affect, treatment should furthermore include affect-regulation techniques, as provided by dialectical behavior therapy (DBT, Linehan, 1996). DBT focuses on the development of adaptive emotional regulation skills and their implementation in daily life and has already been adapted for treatment in BED (Telch, Agras, & Linehan, 2001).

The current research points towards a stress-induced deficit in the regulation and perception of hunger and satiety signals in individuals with BED, which should also be considered in treatment. Stress-reduction and stress coping techniques should be included in the treatment of BED. Also techniques to improve satiety regulation and perception should be trained in order to enable BED individuals to apply them in stressful situations. These techniques include "putting fork down between mouthfuls, chewing thoroughly before swallowing, preparing one portion of food at a time, leaving some food on the plate, pausing in the middle of the meal, doing nothing else while eating" (Laessle, 2005, p.77). These techniques could help patients to gain control over food intake in stressful situations and should therefore be taken into account in the treatment of BED. Furthermore, mindfulness-based treatment approaches such as MB-Eat (Kristeller & Wolever, 2011) or DBT should be considered to improve interoceptive awareness of hunger and satiety while eating in stressful situations. Such treatment could also support the patient to distinguish more sharply between emotional stress and hunger and might therefore result in a reduction of emotional eating.

Restrained eating was not higher in obese women with BED, suggesting that restraint does not necessarily has to be targeted in BED treatment. Yet, the mean restraint score in both the BED and NBED group was higher than 75% of the scores in a nonclinical reference sample (van Strien et al., 1986). Further, our data point to the importance of disinhibition of cognitive control under negative affect and stress in BED subjects. Therefore, decreasing inhibition of cognitive control should be considered in the treatment of BED.

Since disinhibition of cognitive control is expected to occur less under flexible than under rigid restraint[17] (Vögele, 2005), the approach of increasing flexible restraint and decreasing rigid restraint, as included in obesity therapy (Pudel, 2003) should also be considered in the treatment of BED.

[17] **"Rigid Control is characterized by a dichotomous, all-or-nothing approach to eating, dieting, and weight. Flexible Control, on the other hand, is characterized by a more graduated approach to eating, dieting, and weight, in which "fattening" foods are eaten in limited quantities without feelings of guilt" (Westenhoefer, Stunkard, and Pudel, 1999, p.54).**

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APPENDIX

Appendix A: DSM-IV Diagnostic criteria for Binge Eating Disorder

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

1. eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances

2. a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

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

1. eating much more rapidly than normal

2. eating until feeling uncomfortably full

3. eating large amounts of food when not feeling physically hungry

4. eating alone because of 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. The binge eating occurs, on average, at least 2 days a week for 6 months.

Note: The method of determining frequency differs from that used for Bulimia Nervosa; future research should address whether the preferred method of setting a frequency threshold is counting the number of days on which binges occur or counting the number of episodes of binge eating.

E. The binge eating is not associated with the regular use of inappropriate compensatory behaviors (e.g., purging, fasting, excessive exercise) and does not occur exclusively during the course of Anorexia Nervosa or Bulimia Nervosa.

Appendix B: Laboratory-Based Studies on Eating Behavior in BED

Reference	Sample	BED diagnosis	Methods/Instructions	Results
Anderson, Williamson, Johnson, & Grieve, 2001	24 women 8 obese BED 8 obese non-BED 8 normal weight non-BED	DSM-IV interview	single item (chocolate ice cream) to let yourself go and eat as much as you can" in Lutealphase (kcal +)	→ TMI: BED > NBED = normal weight → BED: stronger feeling of loss of control → no differences in enjoyment, postmeal hunger, time to eat the test meal → BED eat faster when instructed to binge eat → Although they eat more, they don't feel more satiated
Bartholome, Peterson, Raatz, & Raymond, 2012	Community sample 32 obese women 15 BED 17 normal weight	DSM-IV interview	multiple-item simulate a binge:"let yourself go and eat as much as you like"	→ TMI: BED > NBED → TMI + dietary recall (24h): BED > NBED → proportion of fat and carbohydrates: BED = NBED
Galanti, Gluck, & Geliebter, 2007	Community sample, men and women (BMI ≥ 27) 22 BED 21 BE* 36 non-BED	QEWP	liquid test meal instruction: "to ingest the test meal until you feel extremely full"	→ TMI: BED and BE > non-BED → Impulsivity and depression: BED and BE > non-BED →.Impulsivity is the best predictor for test meal intake

Geliebter, Hassid, & Hashim, 2001	Outpatient sample 85 overweight (24 males, 61 females) 30 BED 55 without BED	QEWP	liquid test meal continue until you feel extremely full"	→TMI: BED > NBED, men > women → no differences in hunger or fullness ratings after the meal and in duration of test meal or rate of intake → men eat faster than women → depression scores. BED > NBED → depression scores unrelated to TMI → Although they eat more, they don't feel more satiated
Goldfein, Walsh, LaChaussee, Kissileff, & Devlin, 1993	Community sample 20 obese women 10 BED 10 non-BED	QEWP	single item test meal + instruction to eat normal vs. multi-item test meal + binge instruction ("let yourself go and eat as much as you can")	→ BED greater food intake single item test meal + instruction to eat normal: 743 kcal vs. 781 kcal multi-item test meal + binge instruction 1515 kcal vs. 1115 kcal → Only in multiple item test meal with instruction to binge eat : BED > NBED
Gosnell et al., 2001	Community sample 10 women 5 BED 5 non-BED 130% of their ideal body weight	DSM-IV interview	one or two food items "let yourself go and eat as much as you can" variation of number and amount of binge foods	→ TMI: BED = non BED across all conditions (but tendency) → Number and amount of food presented influenced significantly the amount of food consumed. → Data obtained across feeding laboratories and/or feeding paradigms are not comparable unless amount and number of foods are equivalent.

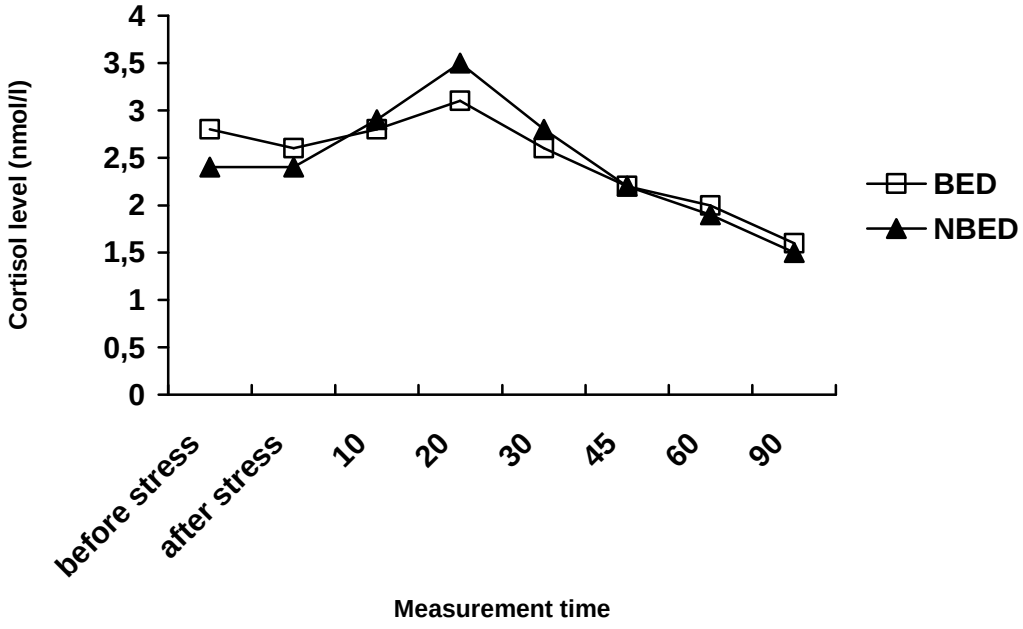
Guss, Kissileff, Devlin, Zimmerli, & Walsh, 2002	Community sample 42 women 12 obese BED with BMI>38 9 BED with BMI 28 to 32 6 non-BED with BMI>38 8 non-BED with BMI 28 to 32 7 normal weight non-BED	DSM-IV SCID	multiple-item meal "Let yourself go and binge eat" vs. instruction to eat normal	→ TMI: BED > NBED: under both instructions → TMI: BED with higher BMI > BED with lower BMI only when asked to binge eat. → TMI: binge meals > normal meals only in BED → kcal of fat: obese > normal weight → BED higher satiety ratings after binge meal than after normal meal → Meal size of BED individuals proportional to body size but only when asked to binge eat
Latner, Rosewall, & Chisholm, 2009	Community sample 30 women 15 BED 15 NBED mean BMI 28	DSM-IV EDE interview	multiple item test meal "Relax and let yourself eat as much or as little as you feel like eating,.." high vs. low preload	→ TMI: BDE > NBED in both conditions → BED higher desire to eat, prospective consumption, excitement about eating, desire for meals and desserts
Lattimore, 2001	female undergratudtes who are currently dieting to loose weight 20 women 11 binge eaters according to question-naire, 9 non-binge eaters	BULLIT-R	single item (ice cream) to eat as much as they wanted" (taste test) Stroop test	Binge eaters consumed more ice cream (mean difference of 29 g) Both Binge Eaters and non Binge Eaters consumed more icecream after the Stroop test

(Raymond, Bartholome, Lee, Peterson, & Raatz, 2007	Community sample 20 obese women 12 BED 8 NBED	DSM-IV SCID	multiple-item buffet (6-10 different kinds of food) instruction: "let yourself go and eat as much as you like"	→ TMI: BED > NBED → kcal of fat: BED > NBED kcal kcal of fat → no differences in the proportion of calories from any macronutrient
Sysko, Devlin, Walsh, Zimmerli, & Kissileff, 2007	Trestment seeking sample 36 women 12 obese BED 12 obese NBED 12 normal weight controls	DSM-IV interview	single-item (macaroni and cheese) let yourself go and binge eat" ratings of hunger and fullness after every 75g increment of food	→ TMI: BED > NBED = normal weight → BED consumed more food to reach a similar level of fullness or hunger → Although subjects with BED did not experience greater levels of hunger, they consumed more on the test meal under both binge and non binge instructions
Telch & Agras, 1996a	Community sample 60 females 30 obese BED 30 obese non-BED	QEWP	multi item buffet "Feel free to let yourself go and eat as much as you want, there's no time limit" negative or neutral mood	→ TMI: BED > NBD → No difference in food intake between negative and neutral mood → negative affect was associated with eating episodes labeled as binges and associated with loss of control
Telch & Agras, 1996b	Community sample 26 BN (DSM III-R) 26 BED (DSM-IV) 26 overweight, no eating disorder (NED)	QEWP	Multi-item buffet, "Feel freee to let yourself go and eat as much as you want, there´s no time limit" 1 vs. 6 h food deprivation, number of bites was observed, food diaries	→ BN = BED = NED inTMI kcal: buffet + food diary Eating rate (bites/minute) → No evidence for difference in BED vs. non-BED and BN in food intake

Yanovski et al., 1992)	Community sample 19 women BMI < 30 10 BED 9 non-BED	DSM-IV interview	multicourse meal instruction to binge eat ("let yourself go and eat as much as you can" or to eat normal	→ TMI: BED > NBED in both binge and normal instruction → BED greater percentage of energy as fat only in binge meal → positive correlation between TMI and BDI score

= means no significant difference,
TMI: Test meal intake,
QEWP: Questionnaire on Eating and Weight Patterns {Spitzer 1992 #1811},
***** consumption of a large amount of food, with a sense of loss of control, while not meeting the full diagnostic criteria for BED (i.e., lower frequency of binge episodes, fewer than three behavioral indicators, or lesser degree of distress about their overeating or loss of control)

Appendix C: Mean values of cortisol before and after stress in obese women with (BED) and without (NBED) binge eating disorder (in nmol/l)



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INDEX OF PUBLICATIONS

The experimental data presented in chapters two; three and four of this work were published in international peer-reviewed journals.

Chapter 2 Schulz, S., & Laessle, R. G. (2010). Associations of negative affect and eating behaviour in obese women with and without binge eating disorder. *Eat Weight Disord*, 15(4), e287-293.

Chapter 3 Schulz, S., & Laessle, R. G. (2012). Stress-induced laboratory eating behavior in obese women with binge eating disorder. *Appetite*, 58(2), 457-461.

Chapter 4 Schulz, S., Laessle, R., & Hellhammer, D. (2011). No evidence of increased cortisol stress response in obese women with binge eating disorder. *Eat Weight Disord*, 16(3), e209-211.

ERKLÄRUNG

Hiermit versichere ich, dass ich die vorliegende Arbeit selber verfasst und keine außer den angegebenen Hilfsmitteln und Referenzen benutzt habe. Die Arbeit wurde bisher weder im Inland noch im Ausland in gleicher oder ähnlicher Form einer anderen Prüfungsbehörde vorgelegt.

Dipl. Psych. Simone Schulz Trier, im Januar 2013