



Research report

Stress-induced laboratory eating behavior in obese women with binge eating disorder

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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. Seventy-one female subjects were investigated (mean BMI 36.9). Thirty-five fulfilled criteria for BED. A 2×2 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 non BED 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).

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Introduction

Binge eating disorder (BED) is defined by binge eating episodes at least at two days per week for 6 months (American Psychiatric Association, 2000). 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% (de Zwaan, 2002; Dingemans et al., 2002; Hudson, Hiripi, Pope, & Kessler, 2007; Spitzer et al., 1993). In clinical samples with participants in weight control programs, up to 30% suffer from BED (Spitzer et al., 1993).

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, Gordon, Mitchell, Crosby, & Engel, 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 non-BED. 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). 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 non-BED individuals which may lead to a decreased satiety response. In line with that, Sysko, Devlin, Walsh, Zimmerli, and 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, Thornton, & Becker, 1982).

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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 non-BED 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). Further more, Gluck, Geliebter, Hung, and Yahav (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 non-BED 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 Trier Social Stress Test (TSST, Kirschbaum, Pirke, & Hellhammer, 1993), on the microstructure of eating in individuals with BED compared to non BED 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 ($p < 0.01$) and a different change in acceleration/deceleration at the end of the meal ($p < 0.04$). 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 satiety) 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.

Methods

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 analyze 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, Wunderlich, Gruschwitz, & Zaudig, 1997) was carried out by a re-

search 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 non-BED 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).

Design

A 2 × 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.

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, protein%: 14, carbohydrate%: 24) and 643 kJ for cheese (fat%: 22, protein%: 14, carbohydrate%: 23). Participants were told that we are interested in finding out how stress influences the feeling of satiety 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.

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 s. 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/s) 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 × 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/s) which is the eating rate over the whole meal, size of spoonfuls (SF, g), frequency of spoonfuls (FSF, spoonfuls/s) and total amount of intake (TI, g). Our instrument is described in detail in Hubel, Laessle, Lehrke, and 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.

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