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Cortisol response and desire to binge following psychological stress: Comparison between obese subjects with and without binge eating disorder

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ABSTRACT

While stress and negative affect are known to precede "emotional eating", this relationship is not fully understood. The objective of this study was to explore the relationship between induced psychological stress, hypothalamic-pituitary-adrenal (HPA) axis activity, and eating behavior in binge eating disorder (BED). The Trier Social Stress Test (TSST) was applied in obese participants with ($n=8$) and without BED ($n=8$), and normal weight controls ($n=8$). Psychological characteristics, eating-related symptoms, and cortisol secretion were assessed. Baseline stress, anxiety and cortisol measures were similar in all groups. At baseline desire to binge was significantly higher among the BED group. While the TSST induced an increase in cortisol levels, a blunted cortisol response was observed in the BED group. In the BED group, a positive correlation was found between cortisol (area under the curve) levels during the TSST and the change in VAS scores for desire to binge. Post-TSST desire to binge and sweet craving were significantly higher in the BED group and correlated positively with stress, anxiety, and cortisol response in the BED group only. These results suggest chronic down-regulation of the HPA axis in participants with BED, and a relationship between psychological stress, the acute activation of the HPA axis, and food craving.

1. Introduction

The obesity pandemic in the Western world with its associated increased morbidity and overall mortality has become a major public health hazard (Flegal et al., 2007). The magnitude of this problem has resulted in substantial research, thus increasing our understanding of the biological factors involved in appetite and eating behavior. A specific area in this field, the relationship between stress regulatory systems and eating behavior, has also received much recent attention.There is increasing evidence that central components of the reward system also play a role in the regulation of the stress response (Ueta et al., 2003). A subset of people who are obese suffer from binge eating disorder (BED), an eating disorder characterized by bouts of uncontrolled binges without the compensatory behaviors that characterize bulimic patients. About 30% of obese people are diagnosed with BED (Yanovski et al., 1993), while the prevalence within the general population ranges from 2 to 3% (Smink et al., 2012) to about 6% (Abebe et al., 2012).

It is known that stress and negative affect often precede "emotional eating" and binge eating (Levine and Marcus, 1997; Pike et al., 2006), but this relationship is not fully understood (Kaye, 2008). Negative emotions have been associated with both increased and decreased food intake (Geliebter and Aversa, 2003), and while this may be related to individual stress reactivity (Cattanach et al., 1988), the mechanisms underlying such opposed behaviors have not been elucidated (Cizza and Rother, 2011). Under conditions of high emotional load, restrained and emotional eaters usually eat more food, specifically sweet and fat foods (Lattimore and Caswell, 2004), perhaps suggesting a lack of responsiveness to satiety signals while under stress (Gibson, 2006).

Neuronal networks that interconnect the hypothalamus and the limbic system suggest the existence of a neural circuit in which mood states strongly influence eating behavior. It is commonly observed that acute stress can induce food restriction. This may be a result of a number of mechanisms, including anorexic signals through the effect of increased central corticotropin-releasing hormone (CRH) secretion with consequent stimulation of α-melanocyte-stimulating hormone (Mastorakos and Zapanti, 2004). Conversely, under conditions of chronic stress and sustained increase in circulating cortisol levels, carbohydrate and fat intake are enhanced (Adam and Epel, 2007), CRH is suppressed and

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neuropeptide Y (NPY) hypothalamic secretion is stimulated (Kyrou and Tsigos, 2007). Hence, It may be hypothesized that contrary to acute stress in which the activation of the hypothalamic–pituitary–adrenal (HPA) axis results in an inhibition of food consumption, under conditions of chronic stress, the activation of the HPA axis may prolong the actions of glucocorticoids in appetite centers, thus causing an orexigenic effect (Kyrou and Tsigos, 2007).

The hypothesis that activation of the HPA axis is involved in the onset of binge eating attacks relies on several lines of evidence: (1) Stress plays a definite role in the initiation of binge eating episodes (Pike et al., 2006; Telch and Agras, 1996); (2) cortisol is a mediator of increased caloric intake (Drapeau et al., 2003), as indicated by the response to infusions of glucocorticoids in both humans and rats (Tataranni et al., 1996; Dallman, 2010); and (3) high cortisol levels are positively related to food intake after laboratory stress stimuli (Gluck, 2006).

In eating disorders such as anorexia nervosa (AN), bulimia nervosa (BN), and night eating syndrome, there is evidence of a hyperactive HPA axis with higher basal cortisol levels (Birketvedt et al., 2002; Pirke et al., 1992) and impaired cortisol suppression by exogenous corticosteroids (Brambilla et al., 1993; Monteleone et al., 1999).

Very few studies have examined the HPA axis in participants with BED. Cortisol suppression after dexamethasone administration has been shown to be normal in this population (Yanovski et al., 1993; Gluck et al., 2004). Single measurement evening cortisol levels (Coutinho et al., 2007), as well as morning cortisol levels, have been reported to be normal in both obese and as well as in non-obese women with BED (Monteleone et al., 2000; Monteleone et al., 2003). In contrast, overall cortisol secretion as reflected by repeated cortisol measurements during the day for two consecutive days was found to be significantly lower in obese women with BED in comparison to obese women without BED (Larsen et al., 2009). Finally, Gluck and colleagues found higher basal cortisol levels in obese women with BED, and a nearly significantly higher cortisol secretion following a cold stress test in comparison to the control group (Gluck et al., 2004). In view of the contradictory results reported in these studies, no conclusions can be drawn as to the HPA axis function in BED subjects. An extensive review of HPA axis and stress involvement in eating disorders was published by Lo Sauro et al. (2008). This review concludes that different HPA axis abnormalities have been observed in BED and obese subjects, but these alterations are considered to be mainly due to excess weight. Thus, the specific association between BED and the HPA axis that is not secondary to obesity still merits study.

To further investigate the involvement of the HPA axis in the eating response to negative emotions, we chose to use a well-standardized psychological stress test, the Trier Social Stress Test (TSST). Cortisol secretion and cognitions related to eating were measured before and immediately after the psychological stress in obese participants with and without BED and a control group of normal weight participants (Rouach et al., 2007). Our hypothesis was that the cortisol response to acute stress in obese BED patients would be higher than in obese non-BED patients, and would be positively correlated with psychological stress and with food craving.

2. Methods

2.1. Subjects

Twenty-four participants, mean age 44.2 ± 15.4 (range 23–70) were recruited from the Obesity Clinic and through advertising among personnel from the Tel Aviv Sourasky Medical Center. The study population comprised the following three groups: (1) Obese (body mass index (BMI) between 30 and 40 kg/m^2) participants with a diagnosis of binge eating disorder (BED group, n=8); (2) Obese (BMI between 30–40 kg/m^2) participants without BED (Non-BED group, n=8); and (3) A control group consisting of normal weight individuals (lean body weight of BMI $>$ 18 kg/m^2 and $<$ 25 kg/m^2), without any diagnosis of eating disorder (NW group, n=8). The diagnosis of Binge The diagnosis of binge eating disorder was made by a certified psychiatrist (NR) according to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th Ed. (American Psychiatric Association, 2000). The sample consisted of 16 females and 8 males. There was a similar male to female ratio in the three groups. Participants diagnosed with anorexia or bulimia nervosa, those with eating disorder not otherwise specified (excluding BED), participants taking any medication affecting the central nervous system or receiving medication known to interfere with cortisol measurements such as contraceptive pills and estrogen replacement therapy were excluded from the study. Ten patients with hypertension, four with hyperlipidemia, three with type II diabetes mellitus, three with hypothyroidism and one with osteoporosis were well controlled with appropriate medical treatment. The investigation was carried out in accordance with the latest version of the Declaration of Helsinki, and it was approved by the Institutional Review Board. All subjects signed an informed consent form after the nature of the procedures had been fully explained to them.

2.2. Study design

Participants were instructed to eat a light breakfast (consisting of a cup of coffee or tea and a slice of bread with cheese) round 07:00 h before arrival to the clinic. Upon arrival, information about the research was given, medical and pharmacological history was taken, and consent was signed. Patients were inquired asked about having eaten breakfast, mainly to ascertain that they were not fasting. The study procedure started at 09:00 h and took a total of 2 h to complete. At first an intravenous catheter was inserted in the subject's forearm, and the subject was then instructed to rest in bed for 40 min before the TSST (Kirschbaum et al., 1993) began. The TSST was conducted by the same examiner (RV). The biological outcome measures included heart rate, blood pressure (systolic and diastolic) and serum cortisol levels, which were determined at 6 consecutive time points: 2 baseline time points before the TSST ($-$30 min and 0) and 4 time points after it (immediately after the completion of the test at 15 min, and at the rest area every 20 min afterwards at 35, 55 and 75 min). The psychological assessment as described below was performed just before the initiation of the TSST and immediately following it.

2.3. Data collection

2.3.1. Trier Social Stress Test (TSST)

The TSST is a standard psychological stress test developed for an induction of moderate psychological stress under laboratory conditions. In the test, participants are asked to deliver a speech for a job application based on personal characteristics, and to perform a mental repetitive arithmetic task in front of two clinical research members while they were under the impression of being photographed on a video camera for subsequent "behavioral analysis". The duration of the psychological stress is about 15 min. After the completion of the TSST, participants were taken to a rest area.

2.3.2. Psychological assessment

We evaluated the participants' subjective psychological state and eating-related symptoms (stress, anxiety, sweet craving and desire to binge) by using visual analog scales (VAS) with a range of 0–100 (not at all-very much, respectively). The following questions were asked:

1) To what extent do you feel an urge for uncontrolled eating (desire to binge) at the present moment?
2) To what extent do you feel craving for sweet foods at the present moment?
3) To what extent do you feel stressed at the present moment?
4) To what extent do you feel anxiety at the present moment?

2.3.3. Laboratory measurements

Cortisol was measured in serum samples by an Electrochemiluminescence Immuno Assay ("Elecsys 2010", Roche). The within-run precision coefficient of variation (CV) was 1.4%, and the between-run CV was 2.1%.

2.4. Statistical analysis

Results are given as mean ± S.D. Basal cortisol levels (baseline) were calculated as the mean of measurements obtained at time points $-$30 and 0. The calculation of area under the curve (AUC) for cortisol levels following stress was computed as the area between the imaginary straight line between each two cortisol measurements and the baseline level. Comparisons of baseline continuous parameters (age, body-mass index, heart rate, cortisol concentrations, VAS measures) between groups (BED and Non-BED) were performed by the non-parametric Mann–Whitney U analysis. The effect of TSST on cortisol was examined by analysis of covariance (ANCOVA) with repeated measures.