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Research report

Urinary cortisol and psychopathology in obese binge eating subjects ☆

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ABSTRACT

Background: Investigations on the relationship between obesity, binge eating and the function of hypothalamic–pituitary–adrenal (HPA) axis have led to inconsistent results. General psychopathology affects HPA axis function. The present study aims to examine correlations between binge eating, general psychopathology and HPA axis function in obese binge eaters. *Methods:* Twenty-four hour urinary free cortisol (UFC/24 h) was measured in 71 obese binge eating women. The patients were administered psychometric tests investigating binge eating, psychopathology and clinical variables. The relationship between binge eating, psychopathology and urinary cortisol was investigated, controlling for age and BMI. *Results:* We found an inverse correlation between UFC/24 h and binge eating, depression, obsessive-compusive symptoms, somatization and sensitivity. In a regression model a significant inverse correlation between urinary cortisol and psychopathology was confirmed. *Conclusions:* Urinary cortisol levels in obese patients with binge eating disorder show an inverse correlation with several dimensions of psychopathology which are considered to be typical of a cluster of psychiatric disorders characterized by low HPA axis function, and are very common in obese binge eating patients. If these results are confirmed, UFC/24 h might be considered a biomarker of psychopathology in obese binge eaters.

Introduction

Binge eating disorder (BED) is characterized by recurrent episodes of binge eating in the absence of purging or other compensatory behaviors (American Psychiatric Association, 2000); it is thought to be a frequent condition in individuals seeking treatment for obesity (Spitzer et al., 1993). The role of HPA (hypothalamic–pituitary–adrenal) axis in obesity and its comorbidities is currently debated (Abraham, Rubino, Sinaii, Ramsey, & Nieman, 2013). Dysfunctions in the HPA axis are thought to play a role in eating disorder psychopathology (Lo Sauro, Ravaldi, Cabras, Faravelli, & Ricca, 2008); in particular binge eating episodes are often preceded by stress and negative affect (Laessle & Schulz, 2009; Levine & Marcus, 1997) and a growing body of research shows that cortisol released during stress might promote hunger and feeding behavior (Tataranni et al., 1996). Indeed, some studies found an augmented cortisol secretion as a result of laboratory stress in obese BED subjects compared to non-BED obese subjects (Gluck, 2006; Gluck, Geliebter, Hung, & Yahav, 2004; Gluck, Geliebter, & Lorence, 2004); in another study patients who developed weight gain after a stressful event have been found to show higher twenty-four hour urinary free cortisol (UFC/24 h) than patients who did not identify a stressful event before the onset of weight gain (Vicennati, Pasqui, Cavazza, Pagotto, & Pasquali, 2009). These data might suggest the presence of higher baseline cortisol levels in some obese BED patients. However, one study examining the HPA in BED found normal cortisol suppression after dexamethasone suppression test (Yanovski, Yanovski, & Gwirtsman, 1993). Some studies that relied on single measurements of evening (Coutinho, Moreira, Spagnol, & Appolinario, 2007) and morning (Monteleone, Di Lieto, Tortorella, Longobardi, & Maj, 2000; Monteleone et al., 2003) cortisol levels in women with BED reported normal levels.

Some authors also reported opposite findings. Two more recent studies in patients with BED suggested a blunted HPA function in obese patients with binge eating disorder (Larsen, van Ramshorst, van Doornen, & Geenen, 2009; Rosenberg et al., 2013).

The difficulty in replicating the anomalies in HPA activity in eating disordered patients with binge eating might be related to other dimensions of psychopathology that affect HPA function.

Dysregulation of the HPA axis in humans has been documented in mood and anxiety disorders (Nemeroff et al., 1999). Particularly, a considerable amount of research has been done in posttraumatic

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Table 1
Baseline demographic data and cortisol values. BMI = body mass index.

	Mean	SD
Age (years)	40.5	12.4
BMI (kg/m^2)	40.8	7.5
Urinary cortisol (μg per L in a 24 h sample)	22.8	17.1

stress disorder (de Kloet et al., 2006; Delahanty, Raimonde, & Spoonster, 2000), chronic fatigue syndrome (Kumari et al., 2009) and depression (Vreeburg et al., 2009); some studies have also been done on psychosis (van Venrooij et al., 2012).

It has been proposed that the alterations of the HPA axis could be specific for different disorders (Handwerger, 2009; Sriram, Rodriguez-Fernandez, & Doyle, 2012) and patients with eating disorders are known to suffer from a variety of psychiatric comorbidities (Hudson, Hiripi, Pope, & Kessler, 2007).

Given the previous findings, it appears relevant to take the effects of comorbid psychopathology into account when studying HPA axis function in obese binge eating subjects.

Urinary free cortisol excretion rate in 24 hours (UFC/24) is an integrated measure of HPA axis function which has been proposed to characterize different phenotypes of obesity (Duclos, Marquez Pereira, Barat, Gatta, & Roger, 2005; Pasquali, Vicennati, Gambineri, & Pagotto, 2008), and it has been used to investigate HPA axis activity in relation to previous stressful events in obese women (Vicennati et al., 2009).

The present study attempts to investigate whether UFC/24 h levels are associated with binge eating and/or general psychopathology in a sample of obese women with BED.

Methods and materials

Of 86 female patients initially recruited from the Center for Eating Disorders of the University of Torino, 4 were excluded from the study because of known endocrine disorders, 7 were excluded because they were diagnosed with a psychotic or bipolar spectrum disorder. Two patients returned incomplete psychometric tests. Two patients had urinary cortisol values above the normal range; they were referred to an endocrinologic assessment and excluded from the study sample. A total of 71 patients met inclusion criteria, accepted to participate in the study and completed the psychometric assessment.

Sociodemographic and psychometric data are reported in Tables 1 and 2.

The BMI of the sample was 40.8 ± 7.5 kg/m^2 (mean ± SD). The diagnostic assessment was made by psychiatrists with experience in the diagnosis and treatment of eating disorders; BED was diagnosed following the criteria proposed by DSM-IV-TR for further study. Patients with illnesses like diabetes or other endocrine disorders were excluded. Patients diagnosed with schizophrenia or related psychoses or bipolar disorder were also excluded; patients diagnosed with anxiety disorder not otherwise specified, dysthymic disorder and major depressive disorder of mild to moderate intensity were included as long as they were not taking psychotropic drugs at the time of assessment. The patients were administered the Binge Eating Scale (BES) and the Symptom Checklist 90 Revised (SCL-90-R) at their first visit to our Centre. The BES is a self-administered questionnaire designed to assess the behavioral and psychological correlates of binge eating (Gormally, Black, Daston, & Rardin, 1982); the SCL-90-R is a self-report tool used to assess several dimensions of psychopathology (see Derogatis, Rickels, & Rock, 1976 for details). HPA activity was assessed dosing UFC per liter of urine in a 24-hour urine sample. UFC (μg/day) was evaluated by CMIA (chemiluminescent microparticle-based immunoassay) automated on Architect i2000 platform (Abbott Diagnostics, Abbott Park, IL, USA). It is a non-invasive, integrated measure that can be collected in the subject's environment. Patients were instructed to collect all urine for 24 hours starting from the next day at 8:00 am, and then were requested to bring the sample to the laboratory of our hospital.

Table 2
Psychometric tests.

		Mean	SD
	BES	22.7	10.7
SCL90R	Somatization	21.2	9.7
	Obsessive-compulsive	14.9	8.6
	Interpersonal sensitivity	13.6	7.5
	Depression	21.0	12.2
	Anxiety	13.2	9.3
	Hostility	6.3	5.0
	Phobic anxiety	4.5	4.8
	Paranoid ideation	7.8	5.9
	Psychoticism	8.6	8.1
	Total	120.3	67.5

SCL-90-R = Symptom Checklist 90 Revised; BES = Binge Eating Scale.

The study has been carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki). Ethical approval for the study protocol was also given by the local Ethics Committee. All the patients provided written informed consent to the study.

Data analysis

For statistical analysis, we used the Statistical Package for Social Sciences (IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.). Partial correlations were calculated between UFC/24 h and psychopathology variables, using age and BMI as confounding variables. The significant correlations were further tested with bootstrapping by creating 1000 bootstrap samples and robust confidence intervals were computed. The variables which were significantly associated with urinary cortisol were then used as predictors in a regression model, where urinary cortisol was the dependent variable, and BMI and age were used as covariates to control for confounding. In case of multicollinearity between the psychological variables, principal component analysis (PCA) was used to obtain uncorrelated principal components to be used in the regression model as predictors.

Results

Partial correlations

Significant correlations were found between UFC/24 h and the following variables: BES (r = –.29; p = .016, 95% CI = –.09, –.45); somatization (r = –.24; p = .041, 95% CI = –.04, –.42); obsessive–compulsive (r = –.32, p = .007, 95% CI = –.11, –.48); sensitivity (r = –.30, p = .012, 95% CI = –.07, –.5) and depression (r = –.30, p = .011, 95% CI = –.47, –.11). Age and BMI were used as covariates; coefficients and confidence intervals were calculated with bootstrapping (1000 bootstrap samples).

The psychometric scales that showed a correlation with urinary cortisol were examined in a correlation matrix as a preliminary step to fitting the regression model (Table 3). We identified a significant degree of correlation between the variables: five out of ten of the pairwise correlations were greater than 0.7 in absolute value, indicating harmful collinearity (Slinker & Glanz, 1985). The other five correlations (0.32–0.59), although smaller, should also be considered problematic for a regression model (Van Steen et al., 2002).