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Circulating leptin in patients with anorexia nervosa, bulimia nervosa or binge-eating disorder: relationship to body weight, eating patterns, psychopathology and endocrine changes

Palmiero Monteleone*, Antonio Di Lieto, Alfonso Tortorella, Nicola Longobardi, Mario Maj

Institute of Psychiatry, University of Naples SUN, Largo Madonna delle Grazie, 80138 Naples, Italy

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Abstract

A decreased production of leptin has been reported in women with anorexia nervosa (AN) and has been attributed merely to the patients' reduced body fat mass. The extent to which eating patterns, purging behaviors, psychopathology and endocrine changes may contribute to the genesis of leptin alterations has not been deeply investigated. Therefore, we measured plasma levels of leptin, glucose and other hormones in three groups of eating disorder patients with different body weight (BW), eating patterns and purging behaviors. Sixty-seven women, 21 with AN, 32 with bulimia nervosa (BN), 14 with binge-eating disorder (BED) and 25 healthy females volunteered for the study. We found that circulating leptin was significantly reduced in AN and BN patients, but significantly enhanced in women with BED. In anorexics, plasma glucose was decreased, whereas plasma cortisol was enhanced; blood concentrations of 17β -estradiol and prolactin (PRL) were reduced in both AN, BN and BED patients. In all subject groups, a strong positive correlation emerged between plasma levels of leptin and the subjects' BW or body mass index, but not between leptin and psychopathological measures, plasma glucose, cortisol, PRL and 17β -estradiol. Since leptin was reduced in both underweight anorexics and normal weight bulimics, but increased in overweight BED women, who compulsively binge without engaging in compensatory behaviors, we suggest that factors other than BW may play a role in the determination of leptin changes in eating disorders. © 2000 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Anorexia nervosa; Binge-eating disorder; Bulimia nervosa; Leptin

* Corresponding author. Tel.: +39-081-566-6517; fax: +39-081-566-6523.
E-mail address: monterit@tin.it (P. Monteleone)

1. Introduction

Leptin, the recently discovered adipocyte hormone, is thought to be an important component of the physiological system controlling energy homeostasis. When injected in the experimental animal, leptin behaves as a satiety factor decreasing food intake, increasing caloric expenditure and reducing body weight (BW) (Mantzoros and Moschos, 1998). Therefore, leptin contributes to BW regulation through modulating feeding behavior and energy expenditure. Moreover, leptin affects several neuroendocrine axes (Ahima et al., 1996) and is currently thought to be an important factor triggering the onset of puberty and regulating the menstrual cyclicality in humans (Apter, 1997).

Anorexia nervosa (AN) and bulimia nervosa (BN) are psychiatric disorders characterized by abnormal eating patterns and purging behaviors, aiming to reduce BW in AN and to prevent its increase in BN, because of the patients' pathological fear of becoming fat. Malnutrition-induced metabolic and hormonal abnormalities occur in both syndromes and amenorrhea is a key diagnostic symptom of AN, while oligomenorrhea or amenorrhea may be present in BN. Given this background, the investigation of leptin production is of obvious interest in patients with eating disorders.

It has been recently shown that, in underweight anorexic patients, leptin plasma levels are severely reduced and correlate with decreased body mass index (BMI) and body fat content (Ferron et al., 1997; Hebebrand et al., 1997; Mantzoros et al., 1997; Kopp et al., 1998; Mathiak et al., 1999). During the recovery of BW, leptin concentrations progressively re-increase to reach even higher than normal blood values (Hebebrand et al., 1997). Therefore, the reduction of body fat mass and BW has been maintained to be the major determinant of the lowered leptin secretion in AN.

Some studies have assessed leptin production in BN (Ferron et al., 1997; Kopp et al., 1997, 1998; Mathiak et al., 1999; Nakai et al., 1999). However, most of them lack healthy controls

(Kopp et al., 1997, 1998; Mathiak et al., 1999), one includes patients under different stages of treatment (Ferron et al., 1997), and one, although including normal controls and untreated patients, groups subjects on the basis of their eating behavior score and not by diagnostic categories (Nakai et al., 1999). Hence, the results they provide cannot be conclusive.

Besides AN and BN, DSM-IV includes in appendix B (criteria sets and axes provided for further studies) the category of binge-eating disorder (BED), which is characterized by bingeing episodes and eating-related psychopathology, as in BN, without compensatory behaviors. Therefore, this disorder offers the opportunity to study binge-related alterations without the confounding effects of compensatory behaviors. To the best of our knowledge, only one study has assessed leptin production in obese patients with BED (Karhunen et al., 1997). Therefore, at present, investigations of leptin production in patients with eating disorders are warranted.

In the present study, we measured plasma levels of leptin and other hormones in healthy women and in three groups of patients, consisting of women with AN, women with BN and women with BED, and assessed possible relationships between this hormone and demographic, clinical, psychopathological and biochemical variables of eating disorder patients.

2. Methods

2.1. Subjects

A total of 92 women were recruited for the study. They were 67 outpatients attending the Eating Disorder Center of our Institute and 25 healthy controls. According to DSM-IV criteria, 21 patients fulfilled the diagnosis of AN, 32 the diagnosis of BN, and 14 the diagnosis of BED. Diagnostic assessment was made by a trained interviewer using the Composite International Diagnostic Interview (CIDI) (WHO, 1987). Of the AN women, 14 were exclusively food restrictors

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