Plasma Leptin, Neuropeptide Y (NPY) and Galanin Concentrations in Bulimia Nervosa and in Anorexia Nervosa

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Abstract

OBJECTIVES: It has been reported that leptin and neuropeptide Y (NPY) play a role in the control of appetite and in the regulation of hormonal secretion.

METHODS: Plasma leptin, neuropeptide Y (NPY) and galanin concentrations were estimated in 13 women with bulimia nervosa (BN) 19 women with anorexia nervosa (AN) and in 19 healthy women of the control group (CG).

RESULTS: Plasma leptin concentration in BN was significantly higher than that in AN and it was lower as compared with the control group, despite the same BMI (body mass index) in both the groups. Plasma leptin level in AN was significantly lower as compared with the controls. Plasma galanin concentrations in AN and BN did not differ significantly from the control group. Plasma NPY concentration in AN was lower than that in the control group. However, plasma NPY level in BN was significantly higher as compared with AN and with the control group (CG). The observed increase of NPY in BN was independent of BMI because BMI in bulimia nervosa was normal.

CONCLUSIONS: The data may suggest that other factors than body weight changes may be involved in the modulation of leptin and NPY release in BN. The pathological behaviour of patients with bulimia nervosa may result from disturbed NPY release which is the strongest orexigenic factor.
**Introduction**

The pathogenesis of anorexia nervosa and bulimia nervosa remains poorly understood. Our previous studies showed that the neuropeptides and neurotransmitters modulating eating behaviour play an important role in the neuroendocrine control of hormonal secretion in anorexia nervosa [1, 2, 3, 4].

Neuropeptide Y (NPY) and galanin are orexigenic peptides in the hypothalamic control of feeding behaviour [5, 6, 7, 8, 9]. NPY and galanin may regulate appetite via both central and peripheral mechanisms. The interaction between central and peripheral signals is due to leptin.

Leptin – a peptide secreted by adipocytes may modulate the activity of NPY and other peptides which are known to affect feeding behaviour [10, 11, 12].

The aim of this study is to evaluate the relationship between leptin and NPY, and galanin in bulimia nervosa and in anorexia nervosa.

**Subjects and Methods**

The study subjects were 13 women with bulimia nervosa aged 17–25 yrs (mean 20 yrs), 19 women with anorexia nervosa aged 16–24 yrs (mean 19 yrs) and 19 healthy women of control group aged 17–26 yrs (mean 20 yrs). A diagnosis of bulimia nervosa and anorexia nervosa was made according to the criteria of DSM-IV [13]. Women with anorexia nervosa (AN) were investigated during the weight loss phase of the disease and the duration of clinical symptoms of anorexia nervosa was 20–24 months. All women with AN were amenorrheic. All women with bulimia nervosa (BN) except one normally menstruated. The duration of clinical symptoms of BN was 21–26 months. All healthy women and women with BN were investigated in the follicular phase of the menstrual cycle. No pharmacological or dietetic treatment was introduced before investigations. All gave their informed consent for the study. Blood samples for NPY, galanin, and leptin assays were taken at 8 am from fasting subjects. Plasma NPY, galanin, and leptin concentrations were measured by radioimmunoassay with commercial kits (Peninsula Laboratories, Belmont, CA). Sensitivity of the NPY assay was 2 pg/tube, and the interassay and intraassay coefficients of variation were 8.5% and 7.3%, respectively. Sensitivity of the galanin assay was 13 pg/tube, and the interassay and intraassay coefficients of variation were 7.3% and 6.1%, respectively. Sensitivity of the leptin assay was 0.5 ng/ml, and the interassay and intraassay coefficients of variation were 8.3% and 6.2%, respectively.

The data are presented as means ± SEM. Statistical analyses were performed with nonparametric tests using the program Statistics and Distribution Fitting (Statistica for Windows). The Kruskal-Wallis test and Anova test were also used.

**Results and Discussion**

It has been known that some neurotransmitters and neuropeptides play an important role in the control of appetite and hormone secretion [3, 4, 6, 9].

Multiple endocrine dysfunctions were observed not only in anorexic but also in bulimic eating disorders [14]. The existence of central neurotransmitter disturbances in bulimia nervosa was suggested by some authors [15, 16, 17, 18]. Leibowitz [16] accumulated some evidence that noradrenaline plays a role in the regulation of hunger and satiety as well as in the regulation of many neuroendocrine systems controlling hypothalmo-hypophyseal system. Kay et al [17] found that noradrenaline concentrations in the plasma and in cerebro-spinal fluid (CSF) were significantly lower in patients with bulimia nervosa. It was not a proof of diminution in the noradrenaline turnover because noradrenaline in the CSF is derived not only from the brain but the catecholamines can also come from the blood stream. However, Kay et al [17] observed increases in plasma noradrenaline concentrations during “binge eating”. They concluded that sympathetic nervous system activity decreases due to the periods of intermittent undernutrition. Jimmerson et al [18] suggested that decreased activity of the central serotonin system may be involved in the mechanism of bulimia.

In our paper we presented plasma leptin, NPY and galanin concentrations in women with bulimia nervosa (BN), anorexia nervosa (AN), as compared with the control group (CG) (table I).
We found that the arithmetical mean of plasma leptin concentration in BN was significantly higher than that in AN (p<0.01).

The higher plasma leptin in BN as compared with AN may be explained by higher BMI (body mass index) in BN than that in AN.

However plasma leptin level in BN was lower as compared with the control group, despite the same BMI in both the groups.

Other authors also observed lower leptin concentrations in the normal weight untreated bulimic patients as compared with the controls [19]. They demonstrated that after acute refeeding plasma leptin increased in both the bulimic patients and the controls, however, in the bulimic patients it did not reach the values observed in the normal controls.

The observations may suggest that other factors than body weight changes may be involved in the modulation of leptin production in BN. Plasma galanin concentrations in AN and BN did not differ significantly from the control group. Our previous studies demonstrated the significant positive correlations between leptin, NPY and BMI [2]. We observed a marked increase of both NPY and leptin in the obese patients. However, in the anorectic patients we found low leptin levels and low NPY concentrations.

The results indicated that in patients with anorexia nervosa low production of leptin did not cause any increase of NPY. Our previous data may suggest the existence of disturbances in the feedback mechanism of leptin-NPY in both the obese and anorectic patients [2].

Our present results confirm previous studies. The plasma leptin level in AN was significantly lower (p<0.001) as compared with the controls. The plasma leptin in AN also was significantly lower than that in BN (p<0.01). The plasma NPY concentration in AN was lower than that in the control group. However, the plasma NPY level in BN was significantly higher as compared with AN (p<0.001) and with the control group (CG) (p<0.01). The observed increase of NPY in BN was independent of BMI because BMI in bulimia nervosa was normal.

It may be speculated that other factors than changes in body weight may be involved in the increased production of NPY in bulimia nervosa. The pathological eating behaviour of patients with bulimia nervosa may result from disturbed NPY release which is the strongest orexigenic factor.

REFERENCES


