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Hunger and Satiety Signals in Anorexia Nervosa: Neuroendocrinological and Genetic Analyses

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Introduction: Deregulations of peripheral control of food intake in anorexia nervosa (AN), hunger signals (Ghrelin & obestatin) and satiety signals (leptin & insulin), have been reported. However, these differences could be a consequence or a risk factor of the disease. The genetic approach seems to be a good strategy to analyse this issue. Indeed, AN has a high heritability of ~50-80%.

Objectives: The aims of this study are to confirm deregulation of hunger and satiety signals in AN and to analyze these results according to genetic polymorphisms. Furthermore, we search for an endophenotype by the screening of the AN mothers.

Methods: This work recruited 3 populations: 100 anorexic patients recruited at CMME (Sainte-Anne Hospital, Paris), their mothers, and 200 control women, matched with the patients or relatives for age. All subjects were assessed during a morning day at CMME. They arrived at 8:30am, fasting since the day before, to take a blood sample, in order to carry out genetic and physiologic analyses. Then, they were analysed for Body Mass Index and eating behaviors, including a psychiatric interview (DSM-IV-TR criteria) and self-questionnaires. Dosage of ghrelin, obestatin, and leptin were done by EIA or RIA. Single nucleotide polymorphisms were genotyped by Taqman assay.

Results: We have confirmed that peripheral control mechanism of food intake is deregulated in AN patients and we observe difference also between mothers and controls.

Conclusions: Leptin dosage might be an endophenotype for anorexia nervosa.