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Sodium consumption and its association with serum leptin in volunteers without cardiovascular disease

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Leptin is a hormone responsible for regulating food intake by inhibiting hunger; its release is proportional to the adipose tissue mass⁽¹⁾. Several studies in mice have found higher concentrations of leptin in those fed with a high-sodium and fat diet compared to those fed only with a high-fat diet⁽²⁾. An increase in the adipocyte volume in white fat tissue associated with a high sodium diet was also observed⁽²⁾. Thus, these data suggest a relationship between dietary sodium consumption and plasma leptin concentrations. It has been hypothesized that this association could contribute to the development of obesity in humans by a leptine resistance-mediated mechanism⁽³⁾. The purpose of this study was to determine the association between sodium consumption and serum leptin concentrations in participants without cardiovascular disease.

One hundred and ninety-seven participants (127 women and 70 men), between 20 and 50 years old, living in Mexico City without cardiovascular disease, high blood pressure or diabetes were randomly selected from the participants recruited in the Tlalpan 2020 cohort from January to August 2017⁽⁵⁾. The Tlalpan 2020 study was approved by the Institutional Bioethics Committee of the National Institute of Cardiology Ignacio Chavez, under number 13–802.

Participants were stratified by body mass index (BMI) (BMI < 18, 18 ≤ BMI < 25, and BMI ≥ 25 kg/m²), and gender; their levels of plasma leptin, 24-hour urinary sodium excretion (the gold standard to determine sodium consumption), were also measured. The linear association between urinary sodium and plasma leptin, was evaluated by a multiple linear regression analysis, stratified according to gender and BMI, adjusting for age.

Participants mean age was 37.61 ± 8.95 years old. Serum leptin was significantly higher in women compared to men (6.00 ± 4.02 vs. 1.64 ± 1.35, p < 0.001) and in overweight or obese women compared to women within the normal range of BMI (p = 0.006 and p < 0.001, respectively). A positive and significant correlation between plasma leptin and urinary sodium excretion was found only among women with obesity (IMC ≥ 30 kg/m²), (β = 0.396, p = 0.035).

The differences in leptin concentrations between women and men could be attributed to differences in body composition, since women have a larger percentage of fat mass, especially subcutaneous fat. In addition, it is known that obesity and higher leptin levels frequently coexist and that high sodium intake is also associated with obesity. The interplay between all these factors had not been studied in humans. As an association between urinary sodium excretion and leptin was found only among women with obesity, we suggest that high sodium intake could be responsible for decreasing leptin sensitivity, reducing the inhibitory effect of leptin on appetite and facilitating weight gain, and being women more susceptible to this mechanism.

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