Leptin can contribute to evaluate cardiometabolic risks in healthy obese population

Obesity is a major risk factor for the development of cardiometabolic complications and it is proportional to the degree of obesity and, more specifically, the accumulation of visceral fat. The excessive accumulation of fat in adipocytes cells initiates an inflammatory response by secreting some proteins (adipokines), as leptin. Under normal physiological conditions, this hormone reduces food intake, activates the sympathetic nervous system, and contribute with thermogenic signals. It is produced mostly by adipocytes and is expressed in levels proportionate to adipose mass. Leptin has crucial role as a biomarker for cardiometabolic diseases once, at high levels, affects the vascular structure, leading to hypertension, angiogenesis, and atherosclerosis. However, a subgroup of obese individuals know as “metabolically healthy”, Body mass index – BMI ? 30 kg/m^2 without metabolic alterations, seem to be protected or more resistant to develop these complications and it is intriguing why some individuals develop comorbidities and others do not. Therefore, we questioned whether the differences between metabolically healthy and unhealthy obese relied on the alterations in metabolic profile, characterized by serum leptin and adiponectin.

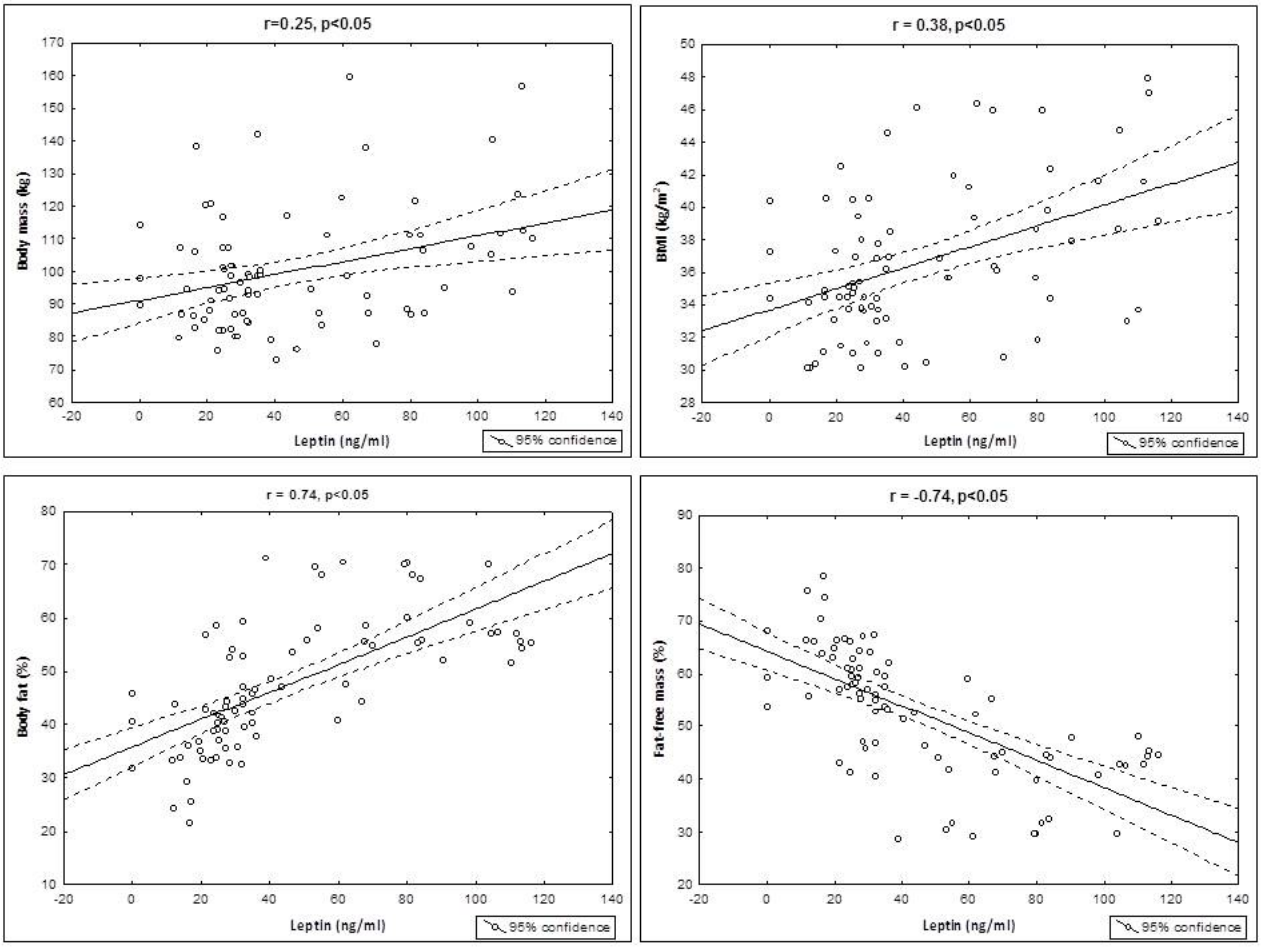

Fig. 1. Correlations between leptin and anthropometric components in obese individuals.

In our study, 142 obese adults were divided into 2 groups – metabolically healthy obese (MHO) or unhealthy obese (MUO) – and they were evaluated for anthropometric measures, body composition, blood pressure, dietary intakes and plasma levels of leptin and adiponectin. Leptin/adiponectin ratio (L/A) was calculated.

According to our results, BMI, blood pressure and leptin were significantly higher in the MUO; and leptin correlated positively with body weight, BMI, and body fat, and negatively with fat-free mass (Fig. 1).

We noticed that although the MHO group showed lower leptinemia than MUO group, these values still indicated a hyperleptinemia state (> 9.4 ng/ml for men and > 27.4 ng/ml for women). Individuals with elevated leptin levels may have a greater risk for the incidence of cardiovascular disease, i.e. our volunteers that showed no metabolic disorders could also have an increased cardiovascular risk, because leptin has a functional receptor in the myocardium leading its structural remodeling. In our analysis, we showed that the individuals' risks raised by 2% for each additional increase in 1 ng/ml of leptin in serum. Besides, both of MHO and MUO groups showed similarly altered adipokines profile with hiperleptinemia and hypoadiponectinemia, suggesting a pro-inflammatory state. Our results confirm that obese individuals with higher leptin levels present an unfavorable metabolic and inflammatory profile, hence, more likely to develop cardiometabolic disorders. Therefore, people with relatively high serum leptin would require further in depth evaluation to identify their likely risk for the development of glucose intolerance and cardiovascular disease. Thus, stratification of obese individuals based on their metabolic phenotype in addition to their inflammatory profile may be important to identify those who are to be prioritized for clinical treatment.

Our findings suggest that leptin is an important cardiovascular disease marker to obese population and can contribute to evaluate metabolic risks in these individuals.

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