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"Obesity, TSH and β-cell Function: An Intriguing Relationship"

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Introduction

The prevalence of obesity is increasing dramatically worldwide with the concomitant increase in its associated comorbidities encompassed in the metabolic syndrome such as type 2 diabetes mellitus, cardiovascular and non-alcoholic fatty liver disease, among others [1-5]. In this context, normal thyroid function is necessary to maintain body weight and a healthy metabolic profile. Current understanding of type 2 diabetes is based on a concept of a progressive failure of pancreatic β-cell function with concomitant increased insulin resistance. In this regard, impaired thyroid function has been associated with glucose intolerance by inducing hepatic gluconeogenesis or perpetuating insulin resistance [6]. This actions are mediated by thyroid hormone nuclear receptors (TRs), and in particular, there are two pancreatic isoforms, located in beta-cells, that are regulated by the thyroid, which are the TRa1 and cErb Aa2 [6-8]. Along these lines, high Thyroid-Stimulating Hormone (TSH) levels are often observed in obese individuals and have been postulated as possible mediators of their metabolic syndrome [8]. However, when adjustment by other variables is made, this correlation becomes weaker [9]. In summary, the possible role that thyroid hormones could be playing on insulin secretion from pancreatic β-cells remains unclear [6-8], particularly, when body composition is included in the equation [10]. Therefore, we aimed to determine the relationship between TSH concentrations and beta-cell function in obese subjects when adjustment by body composition is made.

We selected 490 obese subjects >18-year-old in whom thyroid function test and a 75 g oral glucose tolerance test (OGTT) was performed. Insulin sensitivity and beta cell function were assessed by OGTT-derived indexes as follows: Homeostasis model assessment of insulin resistance (HOMA-IR), Quantitative Insulin Sensitivity Check Index (QUICKI) and insulin sensitivity index (ISI) were used to evaluate insulin sensitivity. In the same lines, HOMA-ß, insulinogenic index (IGI) and disposition index (DI) were used to assess beta cell function. Body composition study was conducted, estimating the body density by Air-Displacement plethysmography (Bod-Pod®) and Siri equation was used to calculate body fat percentage (BF%). Abdominal fat content and visceral adipose tissue was determined by bioelectrical impedance analysis using the ViScan system (Tanita Corp). Multiple regression analysis with adjustment by age, gender, fasting plasma glucose, BF% and %visceral fat was performed in order to identify independent variables affecting insulin sensitivity and beta-cell function indexes.

Mean age (SD) was 44.8 (13.7) years and mean BMI (SD) was 37.8 (7.1) kg/m2. 55.7% were women. Serum TSH was not independently correlated with insulin sensitivity after adjustment (HOMA-IR (ß=0.04; p=ns), ISI (ß= -0.07; p=ns), QUICKI (ß= -0.001; p=ns). However, TSH was positively associated with pancreatic β-cell function assessed by the DI (ß=0.338; p=0.013) and IGI (ß=0.095; p=0.013) but not with HOMA-β (ß=1.6; p=0.716) after multivariable adjustment.

In conclusion, within this study we found a positive relationship between TSH and β-cell function even after adjusting by important confounders including body fat content and distribution supporting the potential role of thyroid hormones in the development of beta-cell dysfunction. This association may be partly explained by the increase in glucose-induced insulin secretion by the thyroid receptors located in beta-cells. However, lack of significance was found when evaluating insulin sensitivity, possibly due to the important effect that fat itself has on the pathogenesis of insulin resistance. However, further studies are warranted to explore in depth this relationship and underlying mechanisms.

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