Abstract

Introduction: The peroxisome proliferator-activated receptor gamma 2 (PPAR2) is an adipogenic transcription factor that influences insulin resistance (IR) in the presence of agonists such as polyunsaturated fatty acids (PUFA). Objective: Evaluate the influence of dietary fat in glicidic metabolism in morbidly obese women with Pro12Pro genotype in the gene PPAR2. Methods: Were selected 25 women with genotype Pro12Pro. The fat intake was estimated by food records, being used for the division of groups, GA (until 30% of the total energy expenditure (TEE)) and GB (greater than 30% of the TEE). Biochemical and anthropometric evaluations were conducted in fasting, following the test meal high in n-6 PUFA and postprandial biochemical evaluations. IR and insulin sensitivity (IS) were assessed by HOMA-IR (Homeostasis Model Assessment) and QUICKI (Quantitative Insulin Sensitivity Check Index), respectively. Results and discussion: GA presented normal HOMA-IR and QUICKI. GB presented higher body mass index (BMI), HOMA-IR, saturated fatty acids (SFA) and monounsaturated (MUFA) intake higher, compared with GA (p < 0.05). In GA, the MUFA intake was negatively correlated with HOMA-IR, fasting glucose and insulin, and positively with QUICKI. The fat and SFA intake contributed to the increase in body mass and IR. However, MUFA intake may have reduced the impact of high fat diet in glicidic metabolism. It is suggested that obese women with Pro12Pro genotype in the PPAR2 gene avoid high fat and SFA diets, prioritizing MUFA for controlling obesity and improving the IS.

Keywords

Morbidly obesity, Insulin resistance, PPAR2, Dietary fats.