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Child Obesity 2018: Associations of polymorphisms of LEPr and FTO genes with food consumption in severely obese individuals- Jaqueline Driemeyer C Horvath- Universidade Federal do Rio Grande do Sul, Brazil

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Genetic factors play an important role in the pathogenesis of obesity. This study aimed at assessing the associations of polymorphisms of LEPr and FTO genes with food consumption in obeses. Thus, a cross-sectional study of 222 obeses, adult patients was carried out. Two SNPs of LEPr (rs1137101 and rs8179183), and one SNP of FTO (rs9939609) were genotyped and analyzed. The food consumption was measured through a three-day diet diary; nutrients were calculated using Nutribase software. We used generalized linear models (GLMz) in SPSS 18.0 to analyze the additive effect of each SNP to caloric intake and macronutrients. The main effect was based on the interaction LEPr rs1137101 and rs8179183; being the caloric consumption associated to these genes ($p<0.037$-GLMz). The mean (±standard deviation) of the caloric intake in the LEPr rs1137101 (AA) genotype was 2780.2 (±1147.9) kcal/day; while in the LEPr rs8179183 (GC) genotype was 2811.2 (±1012.6) kcal/day. In both cases, the caloric intake was higher than the other genotypes. Regarding the macronutrients, only protein intake was associated with the SNPs evaluated ($p=0.023$-GLMz). Removing SNP rs9939609 of FTO from the model had no impact on the statistical significance. SNPs of the LEPr gene seem to play a potential role in the stratification of obese patients, as they may help to predict those individuals who are at higher risk for upper calories intake, and, therefore, for poorer outcomes. These findings have to be confirmed in prospective studies

Obesity prevalence has increased during the past century and the World Health Organization (WHO) estimates that the number of overweight/obese young children will reach 70 million in 2025 . Obesity is a chronic disease with multifaceted etiology . Socioeconomic changes during the last decades have contributed to these phenomena, including the increased availability of high-fat foods and generalized adoption of sedentary lifestyles. Furthermore, there is evidence that genes play an important role in the rise of obesity . Heritability is estimated to account for 40–90% of the population adiposity variation. Seemingly, the presence of single nucleotide polymorphisms (SNPs) offer a protective factor in the development of non-communicable diseases, such as obesity related diseases . With the development of high-throughput genotyping techniques, new approaches such as genome-wide linkage and genome-wide association studies (GWAS) have been used to understand genetic influences in obesity . However, the majority of identified SNPs have unknown biological functions and some of these studies yielded contradictory results, suggesting a need for further examination into the functions of identified SNPs related to obesity.

Peroxisome proliferator−activated receptor-gamma (PPARG) is another gene that has an important role in obesity. PPARG is a member of the nuclear hormone superfamily, which is involved in adipocyte differentiation and glucose metabolism . There are evidences that PPARG deficiency results in increased leptin levels . The PPARG rs1801282 variant is positively associated with obesity and has been extensively examined in epidemiological studies .The aim of the present study was to assess the independent contributions of LEPR (rs1137101), FTO (rs9939609), MC4R (rs2229616 and rs17782313), and PPARG-2 (rs1801282) polymorphisms for clinically overweight or obesity phenotypes and endocrine-metabolic traits in prepubertal children.

Discussion

Ethnicity and environmental factors (i.e., modifying the gene expression but not it’s structure) may affect specific genetic variants under specific conditions, which may distinctly affect obesity-related phenotypes. Obesity can be associated with different metabolic phenotypes of atherogenic lipid profiles and insulin resistance and several studies have investigated the links between obesity, biochemical traits, and polymorphisms to establish possible mechanisms of action . This genetic information could be useful to identify children at risk, plan early interventions, and reduce the life-long burden of obesity-related diseases. However, most studies of obesity-SNP associations have yielded controversial results, and the mechanisms underlying the increased risk of obesity conferred by specific alleles remain unclear.

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