Assessment of biochemical parameters and characterization of *TNFα* -308G/A and *PTPN22* +1858C/T gene polymorphisms in the risk of obesity in adolescents

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Received August 3, 2015; Accepted October 16, 2015

DOI: 10.3892/br.2015.534

Abstract. Obesity is currently considered an inflammatory condition associated with autoimmune diseases, suggesting a common origin. Among other factors, candidate genes may explain the development of this disease. Polymorphisms in the tumor necrosis factor α (*TNFα*) and lymphoid protein tyrosine phosphatase (*PTPN22*) genes lead to an increased risk to development of immune and inflammatory diseases. The aim of the present study was to analyze the biochemical parameters and the effect of the *TNFα* -308G/A and *PTPN22* +1858C/T polymorphisms in the susceptibility of adolescents to obesity. A group of 253 adolescent subjects were recruited and classified as obese, overweight or normal weight according to their nutritional status. Anthropometric measurements, clinical and biochemical data were analyzed. DNA was extracted from peripheral blood samples by the phenol-chloroform method, and *TNFα* -308G/A and *PTPN22* 1858C/T polymorphisms were determined by polymerase chain reaction-restriction fragment length polymorphism assays. Clinical, genetic and biochemical parameters were analyzed to determine the existence of a possible association with the development of obesity. Statistically significant differences in body mass index, insulin, triglyceride levels and homeostatic model assessment for insulin resistance (HOMA-IR) index were observed among the three groups analyzed (P≤0.05). The studied polymorphisms did not confer a risk for developing obesity in the analyzed population (P>0.05); however, significantly low levels of insulin and decreased rates of HOMA-IR were observed in the 1858 CT genotype carriers of the *PTPN22* gene. In conclusion, no association between the *TNFα* -308G/A and *PTPN22* +1858C/T polymorphisms and the risk to development of obesity in the adolescent population analyzed was observed. However, the 1858 CT genotype of the *PTPN22* gene was associated with variations of certain biochemical parameters analyzed.

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Key words: obesity, autoimmune diseases, inflammatory, Mexican population, PCR-RFLP, *TNFα* -308G/A, *PTPN22* +1858C/T

Introduction

Obesity is a chronic disease characterized by hyperplasia and/or hypertrophy of adipose tissue as a result of a positive energy balance, which occurs when energy intake exceeds energetic expenditure (1). Although the 'obesogenic environment' contributes to the development of obesity, this does not explain the inter-individual variability that occurs in the susceptibility of this pathology (2). It has been reported that this disease contributes to an increasing number of pathologies, such as diabetes mellitus type 2 (DM2), cardiovascular disease, cancer, hyperlipidemia and metabolic syndrome (3,4), hence the importance of their study. Obesity is established indirectly by the body mass index (BMI), as this corporal value correlates with fat (5).

Obesity is considered a complex and multifactorial disease, as it is the result of an interaction between genetic, behavioral and environmental factors that may influence individual response to diet and daily physical activity (6,7).

Evidence indicates that the accumulation of body fat has a strong genetic background for monogenic and polygenic forms