Abstract

Increased prevalence of obesity in the world, especially accumulation of abnormal amounts of visceral fat predisposes to insulin resistance, which is the central role of metabolic syndrome (MS). Obesity can deregulate the intracellular signaling of insulin due to the production of inflammatory substances, chemoattractant proteins, adipokines and molecules that trigger hormonal mediator potentials for destabilization of signal transduction, leading to metabolic disorders such as hyperglycemia, hypertension, and dyslipidemia. The complexity of the MS and of the genetic mechanisms involved in its etiology derives from the combination of variants on genes involved and environmental factors that predispose it. The purpose of this paper is to review the effects of obesity in molecular and biochemical responses that trigger insulin resistance and its relation to some candidate genes and the ancestral component of the population.

Keywords

Metabolic syndrome, obesity, insulin resistance, genetics, ancestry.