Abstract

Introduction: The reduction in the capacity of insulin to reach its biological effects can lead to a chronic hyperglycemia and hyperinsulinemia, assuming an important role in the pathogenesis of metabolic disorders associated to obesity and diabetes. Insulin resistance is associated to chronic subclinical inflammation, which in part can be mediated by increased plasmatic lipopolysaccharide levels, an endotoxin derived from the membrane of gramnegative bacteria that mainly reside in the gut. Objectives: The aim of this review study is to describe the molecular mechanisms involved in the pathogenesis of insulin resistance due to metabolic endotoxemia and of its connection to obesity and diabetes. Results and discussion: Lipopolysaccharide present in the intestinal lumen can reach the circulatory system causing metabolic endotoxemia. When lipopolysaccharide binds to Toll-like receptor 4, inflammation is activated, changing several stages of insulin signaling. It has been shown that chronic exposure to this endotoxin may contribute to weight gain and type 2 diabetes mellitus manifestation. Obese and diabetic people have increased plasmatic lipopolysaccharide levels. The increase in the number of gram-negative bacteria on gut microbiota, the reduction on gut mucosal integrity, and the consumption of high-fat diets increase the plasmatic lipopolysaccharide levels. Therefore, the type of diet consumed may modulate the composition of gut microbiota and improve gut mucosal integrity, decreasing the occurrence of endotoxemia and its postprandial inflammatory effects, leading to adequate insulin signaling. However, there are very few studies that evaluated the influence of nutrients and/or specific food types on metabolic endotoxemia.

Keywords
Gut microbiota, Lipopolysaccharide, Endotoxemia, Inflammation, Insulin resistance.