

IMBALANCE BETWEEN PROTECTIVE AND INFLAMMATORY ADIPOKINES IN THE METABOLIC HOMEOSTASIS OF TYPE 2 DIABETES

Maria Clara Sangueta

Emanuelli Salvador Presa; Márcia Rocha Gabald

RESUMO

INTRODUCTION: Adipose tissue functions as an active endocrine organ in regulating insulin sensitivity. Through the secretion of adipokines, it establishes a complex systemic communication that shapes insulin signaling in peripheral tissues, such as muscle and liver. There is growing evidence that endocrine dysfunction in this tissue, characterized by a low-grade chronic inflammatory state, is fundamental to the failure of glycemic homeostasis in Type 2 Diabetes Mellitus (DM2). Thus, the transition from a protective secretory profile to a pro-inflammatory phenotype directly contributes to systemic metabolic collapse. **OBJECTIVE:** To analyze the evidence on the impact of adipokine dysregulation on metabolic homeostasis in DM2, exploring the molecular mechanisms that link adipocyte dysfunction to insulin resistance and pancreatic beta cell failure. **METHODOLOGY:** This investigation constitutes an integrative literature review, with a qualitative and descriptive approach. Data collection was carried out through a survey of scientific evidence indexed in the PubMed and SciELO databases, covering the period from 2024 to 2026. The search strategy included articles in Portuguese and English, using MeSH/DeCS descriptors such as "Adipokines", "Type 2 Diabetes", "Asprosin", and "Homeostasis". Inclusion criteria focused on studies that addressed in detail the cell signaling mechanisms and biomarkers of metabolic dysfunction strictly associated with the type 2 Diabetes Mellitus phenotype, allowing a comparative analysis of the results found. **RESULTS:** The results demonstrate that DM2 should be understood as a consequence of endocrine signaling failure in adipose tissue. In this sense, the severe reduction of adiponectin is directly linked to the failure in insulin receptor phosphorylation, while the increase in pro-inflammatory adipokines, such as resistin and RBP4, induces endoplasmic reticulum stress in pancreatic beta cells. Recent evidence highlights that modulating novel adipokines, such as asprosin, a glycogenic adipokine that acts in the liver stimulating the release of glucose into the circulation, correlates with peaks of fasting hyperglycemia at elevated levels, making it a promising target for the development of new antidiabetic drugs aimed at restoring insulin sensitivity without causing episodes of hypoglycemia.

CONCLUSION: It is concluded that type 2 diabetes should be understood as a consequence of failure in endocrine signaling in adipose tissue and not just a disease of the pancreas or insulin receptor. Restoring the balance between inflammatory and protective adipokines is a crucial approach for glycemic control and the prevention of long-term microvascular complications. Understanding these molecular mechanisms allows for the development of personalized and effective therapeutic strategies for managing the treatment of the disease.

Palavras-chave: Adipokines Type 2 Diabetes Asprosin Homeostasis.