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Central Mechanisms of Inflammatory Cytokines in the Initiation and Progression of Metabolic Syndrome

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Abstract This study explored the central-related mechanisms of inflammatory cytokines in the occurrence and development of metabolic syndrome, analyzed the way peripheral inflammatory signals enter the center, the inflammatory response in the hypothalamus, and the participation of glial cells. It also investigated the key central pathways through which inflammatory cytokines affect metabolic regulation and the specific process by which central inflammation promotes the progression of metabolic syndrome. Based on the relevant evidence from animal experiments, human studies and intervention studies, the core role of central inflammatory cytokines was clarified, and the therapeutic methods targeting the central inflammatory pathway and future research directions were prospected. This study aims to provide a basis for understanding the pathogenesis of metabolic syndrome and developing new therapeutic measures.

Keywords Metabolic syndrome; Inflammatory cytokines; Central inflammation; Hypothalamus; Insulin resistance

1 Introduction

Metabolic syndrome (MetS) is a disease characterized by multiple metabolic abnormalities, with its core manifestations being excessive abdominal fat, insulin resistance, dyslipidemia, hypertension and poor blood sugar regulation. These manifestations can serve as diagnostic criteria and will significantly increase the risk of type 2 diabetes, cardiovascular diseases and other conditions. In the past decade, sedentary lifestyle, overnutrition and genetic factors have led to a rapid increase in the prevalence of MetS, affecting a large number of adults and adolescents worldwide (Fahed et al., 2022; Islam et al., 2024), becoming a public health issue that requires high attention (Rossi et al., 2021).

The typical feature of MetS is chronic mild inflammation ("meta-inflammation"), which is different from acute inflammation (such as Fahed, etc.). This persistent inflammation is mainly caused by the dysfunction of adipose tissue. Adipose tissue secretes pro-inflammatory factors such as TNF-α and IL-6, attracting immune cells to aggregate and transform into a pro-inflammatory state, thereby aggravating systemic inflammation (Islam et al., 2024). This is key to the occurrence of MetS and related complications (Rossi et al., 2024).

This study will explore the core issue of the peripheral inflammatory mechanism of MetS: how the brain perceives and integrates peripheral inflammatory signals to regulate metabolic balance or trigger diseases. Pro-inflammatory factors can transmit signals to the central nervous system through pathways such as the blood-brain barrier, activating immune cells like microglia. The resulting central inflammation can disrupt the neural circuits in the hypothalamus that regulate appetite and blood sugar, creating a vicious cycle of metabolic disorders. Clarifying the core mechanism by which inflammatory factors regulate metabolism-related neural regulation is of great significance for formulating new treatment strategies and breaking the mutually reinforcing cycle between inflammation and metabolic diseases.

2 Overview of Inflammatory Cytokines

2.1 Key pro-inflammatory factors

Pro-inflammatory factors are important regulatory factors for the occurrence and development of metabolic syndrome, and they form chronic low-grade inflammation by promoting insulin resistance and metabolic