

ADIPOSE TISSUE AS A NEUROIMMUNE MODULATOR: THE ROLE OF INFLAMMATORY MEDIATORS IN NEUROINFLAMMATION

André Duarte Bezerra

RESUMO

Introduction: Among the specialized tissues that make up our body is adipose tissue. In addition to performing functions such as thermal regulation and energy storage, it is also capable of acting as an endocrine and immunometabolic organ through the secretion of cytokines and adipokines that have anti- and pro-inflammatory roles, impacting the most diverse tissues present in our body, with nervous tissue being one of the most susceptible to these effects. **Objective:** This work aims to identify the main inflammatory mediators produced by adipose tissue that are capable of affecting nervous tissue and what the possible effects are on the functioning of this tissue. **Methods:** This study consists of a literature review using the PubMed database. Review articles published in the last five years were included. The following descriptors were used for the search strategy: adipose tissue, neuroinflammation, and inflammatory mediators. **Results:** The literature shows that adipose tissue is responsible for the production of inflammatory mediators such as TNF- α (Tumor Necrosis Factor Alpha), IL-6 (Interleukin-6), IL-1 β (Interleukin-1 beta), Leptin, Resistin, MCP-1 (Monocyte Chemoattractant Protein-1), and Fatty Acids, with TNF- α , IL-6, MCP-1, Leptin, and Fatty Acids being able to affect the functioning of the nervous system. These factors can be organized into two main axes: (1) classic pro-inflammatory cytokines, which are activators of pathways such as TLR4 (Toll-like receptor 4), and (2) adipokines and lipid metabolites, which modulate blood-brain barrier permeability and glial cell activation. Articles already indicate the role of these mediators in activating receptors of immune system cells, such as TLR4 (Toll-like receptor 4). This activation is capable of triggering a cascade of reactions that further increase the production of inflammatory mediators, leaving the organism in a state of chronic inflammation. The presence of these cytokines would affect nervous tissue in two ways: firstly, due to the existence of nerve structures that are outside the blood-brain barrier, and secondly, due to the altered permeability of this barrier, which would result in the entry of more and more cytokines into the nervous tissue. The exposure of nerve cells, such as microglia and astrocytes, would lead to the activation of these cells, resulting in the production of more mediators, which would significantly affect this tissue. The presence of inflammatory mediators and the activation of glial cells have been linked to neurodegenerative conditions and also to alterations in neurodevelopment. **Conclusion:** This study demonstrated the importance of adipose tissue as a gland capable of acting as a neuroimmune modulator. When dysfunctional, this modulator impacts the functioning of nervous tissue due to increased secretion of inflammatory mediators. It was shown that systemic inflammation and the circulation of these mediators in the body can impact the functioning of nervous tissue, which is related to some neurodegenerative conditions and alterations in neurodevelopment. Further studies should be conducted to specify how each of these mediators affects nervous tissue so that we can develop therapeutic strategies to mitigate these damages.

Palavras-chave: Adipose Tissue; Inflammatory Mediators; Neuroinflammation.