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Short Communication

Immunometabolic Alterations in Adipose Tissue Macrophages and their Association with Chronic Low-Grade Inflammation in Adults with Obesity

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DESCRIPTION

Obesity is associated with persistent low-grade inflammation that contributes to metabolic dysfunction and several chronic disorders. Among the cellular populations present within adipose tissue, macrophages have received considerable attention because of their involvement in inflammatory signaling and tissue homeostasis. Changes in nutrient availability, lipid accumulation, and cellular stress influence macrophage behavior and alter communication between immune and non-immune cells. This article examines the relationship between adipose tissue macrophages and chronic inflammation in adults with obesity. Particular attention is given to metabolic adaptations within macrophages, interactions with adipocytes, cytokine production, and consequences for systemic health. Understanding these biological processes may contribute to the development of new approaches for reducing inflammation and improving metabolic outcomes [1].

Obesity has become a major public health concern across many regions of the world. Beyond excessive fat accumulation, obesity is characterized by complex biological changes involving endocrine, metabolic, and immune systems. Adipose tissue is no longer viewed as a passive reservoir for energy storage. Instead, it functions as a highly active organ that produces hormones, cytokines, growth factors, and lipid mediators. These substances influence numerous physiological processes, including appetite regulation, glucose metabolism, vascular function, and immune activity [2].

As adipose tissue enlarges, oxygen delivery may become insufficient in certain regions. Reduced oxygen availability can lead to cellular stress and adipocyte injury. Damaged adipocytes release danger-associated molecular signals that attract circulating monocytes from the bloodstream. These monocytes migrate into adipose tissue and differentiate into macrophages. As a result, macrophage numbers increase substantially in individuals with obesity compared with lean individuals.

The accumulation of macrophages is accompanied by significant changes in their metabolic activity. Cellular metabolism influences immune cell behavior by regulating energy production and biosynthetic pathways [3]. In inflammatory conditions, macrophages frequently increase glycolytic activity, allowing rapid generation of energy and metabolic intermediates. This metabolic profile supports production of inflammatory cytokines such as tumor necrosis factor-alpha, interleukin-6, and interleukin-1 beta. Elevated concentrations of these mediators contribute to local tissue inflammation and systemic metabolic disturbances.

Lipids play a central role in shaping macrophage responses within adipose tissue. Excess fatty acids can activate intracellular signaling pathways that stimulate inflammatory gene expression. Certain saturated fatty acids are particularly effective in promoting cytokine production. Continuous exposure to elevated lipid concentrations may also alter mitochondrial function, resulting in increased production of reactive oxygen species. These molecules influence signaling pathways that amplify inflammatory responses and contribute to tissue dysfunction [4].

Insulin resistance represents one of the most significant metabolic consequences associated with chronic inflammation. Under physiological conditions, insulin promotes glucose uptake by skeletal muscle and adipose tissue while suppressing glucose production by the liver. Inflammatory cytokines interfere with components of insulin signaling pathways, leading to impaired glucose regulation. Over time, these alterations increase the likelihood of developing type 2 diabetes and related metabolic complications [5-8].

Recent studies have highlighted the importance of macrophage heterogeneity within adipose tissue. Rather than existing as a uniform population, macrophages display considerable diversity in gene expression, metabolic activity, and biological function. Some macrophage subsets contribute primarily to inflammatory responses, whereas others participate in tissue repair and maintenance. The balance among these populations appears to influence overall tissue health and metabolic status [9,10].

Single-cell analytical techniques have provided new insights into macrophage diversity. Investigators have identified specialized populations associated with lipid handling, extracellular matrix

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regulation, and immune modulation. Certain macrophages form structures around damaged adipocytes known as crown-like formations. These cellular arrangements facilitate clearance of cellular debris but are also associated with increased inflammatory activity. Understanding the factors that determine macrophage specialization may improve knowledge of obesity-related disease mechanisms.

Dietary factors influence immune activity and macrophage behavior. Diets rich in saturated fats and highly processed foods are frequently associated with increased inflammatory markers. In contrast, dietary patterns containing fruits, vegetables, whole grains, and unsaturated fats are linked with reduced inflammation. Bioactive compounds present in plant-derived foods may influence cytokine production, oxidative stress, and cellular metabolism. These observations suggest that nutritional interventions may alter immune responses within adipose tissue. Physical activity also affects inflammatory status. Regular exercise improves insulin sensitivity and modifies immune cell distribution in adipose tissue. Exercise-induced changes in cytokine production may contribute to reductions in chronic inflammation. Furthermore, physical activity enhances mitochondrial function and energy metabolism, factors that influence macrophage behavior and tissue health.

Pharmacological investigations have explored methods for modifying macrophage activity in obesity. Several experimental approaches aim to reduce inflammatory cytokine production, alter immune cell recruitment, or modify metabolic pathways within macrophages. Although some interventions have demonstrated beneficial effects in preclinical studies, translation into routine clinical practice remains an active area of research. Continued investigation is required to determine efficacy, safety, and long-term outcomes.

CONCLUSION

Adipose tissue macrophages occupy a central position in the biological processes linking obesity with chronic low-grade inflammation. Changes in nutrient availability, lipid exposure, cellular stress, and tissue architecture influence macrophage metabolism and inflammatory activity. Interactions between macrophages and adipocytes contribute to systemic metabolic disturbances, including insulin resistance and increased risk of chronic disease. Ongoing research examining macrophage diversity, metabolic regulation, and intercellular communication continues to expand understanding of obesity-related inflammation and may support the development of effective interventions for improving metabolic health.

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