Brief Report

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Cellular & Molecular Medicine: Open Access
ISSN: 2573-5365

2025
Vol. 11 No. 1:1

Decoding the Crosstalk between Inflammation and Cellular Metabolism in Chronic Diseases

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Received date: February 01, 2025, Manuscript No. ipcmm-25-20874; **Editor assigned date**: February 03, 2025, Pre QC No ipcmm-25-20874(PQ); **Reviewed date**: February 19, 2025, QC No ipcmm-25-20874; **Revised date:** February 26, 2025, Manuscript No.ipcmm-25-20874(R); **Published date:** March 5, 2025, DOI: 10.21767/2573-5365.11.1.1

Citation: Thompson S (2025) Decoding the Crosstalk between Inflammation and Cellular Metabolism in Chronic Diseases. J Cell Mol Med Vol.11 No.1:1

Introduction

Chronic diseases such as diabetes, cardiovascular disorders, obesity, and cancer are increasingly recognized as conditions driven not only by genetic and environmental factors but also by intricate interactions between inflammation and cellular metabolism. The immune and metabolic systems are tightly interconnected, each influencing the other through a network of signaling molecules, metabolic intermediates, and transcriptional regulators. Inflammatory responses alter metabolic pathways to meet the energetic and biosynthetic demands of immune cells, while metabolic dysfunction can in turn trigger or amplify inflammation. This dynamic interplay often referred to as immunometabolism has emerged as a critical determinant in the onset and progression of chronic diseases [1].

Description

Inflammation is an essential physiological process that protects the body against infection and injury; however, when sustained or dysregulated, it becomes a key contributor to chronic disease. Immune cells such as macrophages and T lymphocytes undergo profound metabolic reprogramming during activation, switching from oxidative phosphorylation to glycolysis to meet increased energy demands and support inflammatory functions. Cytokines such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6) act as metabolic modulators, influencing glucose uptake, lipid metabolism, and mitochondrial function In metabolic tissues including adipose tissue, liver, and skeletal muscle persistent low -grade inflammation disrupts insulin signaling and lipid balance, contributing to conditions like insulin resistance and fatty liver disease [2].

Moreover, oxidative stress generated during chronic inflammation exacerbates mitochondrial dysfunction; further impairing cellular energy homeostasis and perpetuating the inflammatory cycle. This continuous feedback loop between inflammation and metabolism forms the molecular foundation of much chronic pathology.

At the molecular level, several signaling pathways act as key regulators of the inflammation-metabolism interface. The Nuclear Factor-Kappa B (NF-κB) and hypoxia-inducible factor 1-alpha (HIF-1α) pathways link metabolic changes to inflammatory gene expression, while AMP-Activated Protein Kinase (AMPK) and mechanistic Target of Rapamycin (mTOR) serve as central metabolic sensors that influence immune responses. For instance, activation of AMPK promotes anti-inflammatory effects by enhancing mitochondrial biogenesis and fatty acid oxidation, whereas mTOR activation supports pro-inflammatory responses by stimulating anabolic metabolism [3].

The discovery of these metabolic checkpoints has reshaped our understanding of chronic disease pathophysiology, emphasizing that immune cell activity and metabolic state are inseparable processes. Therapeutic strategies targeting metabolic regulators such as AMPK activators, mTOR inhibitors, or antioxidant compounds are being explored to restore the balance between inflammation and metabolism, offering promising interventions for diseases like type 2 diabetes, atherosclerosis, and neurodegeneration [4,5].

Conclusion

The crosstalk between inflammation and cellular metabolism represents a fundamental biological mechanism underlying many chronic diseases. This intricate relationship determines how cells respond to stress, injury, and metabolic imbalance, ultimately influencing disease progression and outcome. By decoding this bidirectional communication, researchers are uncovering new targets for therapeutic intervention that go beyond symptom management to address root molecular causes. Integrative approaches combining metabolic modulation with anti-inflammatory strategies hold immense potential for transforming the treatment landscape of chronic diseases.

Acknowledgement

None

Conflicts of interest

None

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