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Metabolic Reprogramming of Immune Cells in HIV Infection and its Implications for Treatment

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DESCRIPTION

The Human Immunodeficiency Virus (HIV) presents a unique challenge to the immune system, driving profound changes in immune cell metabolism that significantly impact disease progression and treatment outcomes. HIV exploits host metabolic pathways to enhance its replication, evade immune responses, and establish persistent infections. Understanding the metabolic reprogramming of immune cells during HIV infection can provide insights into potential therapeutic strategies and improve the management of the disease. HIV primarily targets CD4+ T cells, a crucial component of the adaptive immune response. Upon infection, these cells undergo dramatic metabolic changes, shifting from a quiescent state to an activated one. This activation is characterized by increased glycolysis, a process that converts glucose into energy rapidly, allowing the cells to meet the heightened energy demands associated with viral replication. Glycolysis generates adenosine triphosphate (ATP) and metabolic intermediates necessary for nucleotide synthesis and the production of biosynthetic precursors. As a result, HIV-infected CD4+ T cells exhibit elevated levels of lactate and other glycolytic byproducts, contributing to a metabolic environment that favors viral replication. The metabolic reprogramming in HIV-infected cells also affects their functionality and longevity. Activated CD4+ T cells typically transition to a memory phenotype, which is essential for long-term immunity. However, in the context of HIV, this transition is altered. The chronic activation of these cells leads to a state of exhaustion characterized by impaired proliferation, decreased cytokine production, and increased expression of inhibitory receptors like PD-1. This exhausted phenotype is associated with persistent viral replication and contributes to the progression toward AIDS. Furthermore, the metabolic alterations extend beyond CD4+ T cells to other immune cell types, including CD8+ T cells, natural killer (NK) cells, and monocytes. For instance, CD8+ T cells, which are crucial for killing HIV-infected cells, also experience metabolic shifts during infection. These cells initially increase their glycolytic activity to respond effectively to viral loads but may become dysfunctional over time, leading to reduced antiviral activity. This dysfunction is exacerbated by the inflammatory environment created by chronic HIV infection, which can further impair immune cell metabolism and function. HIV treatment strategies have evolved to target not only the virus but also the underlying metabolic disturbances in the immune system. Antiretroviral therapy (ART) effectively suppresses viral replication, allowing for partial restoration of immune function and metabolic homeostasis. However, even with successful viral suppression, many patients experience persistent immune activation and metabolic dysregulation, indicating that ART alone may not fully restore immune health. Recent research has begun to explore adjunctive therapies that target the metabolic pathways altered by HIV infection. Approaches that enhance oxidative metabolism, promote mitochondrial function, or modulate glycolytic flux show promise in improving immune cell function. For example, compounds that stimulate mitochondrial biogenesis or enhance fatty acid oxidation may improve the quality of CD8+ T cell responses and enhance their ability to control HIV replication. Additionally, dietary interventions and exercise are being investigated for their potential to ameliorate metabolic dysfunction in HIV-infected individuals. Nutritional strategies that promote a balanced intake of macronutrients and micronutrients could support immune cell metabolism and overall health.

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CONFLICT OF INTEREST

The author declares there is no conflict of interest in publishing this article.

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