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Perspective

The Importance of Metabolic Reprogramming in Cancer Metastasis

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DESCRIPTION

Cancer metastasis is the leading cause of death from cancer. It is a complex, inefficient, and multistep process associated with poor prognosis and high patient mortality. Growing evidence suggests that metabolic programming, a recognized cancer hall marker, plays a critical role in cancer metastasis. Changes in glucose, lipid, and amino acid metabolism provide cancer cells with energy and substances for biosynthesis, which maintain bio-functions and have a significant impact on cancer cell proliferation, invasion, and metastasis. Tumor Microenvironment (TME) is a complex system composed of cellular and non-cellular components. Non-tumor cells in TME also undergo metabolic reprogramming or respond to metabolites to promote cancer cell migration and invasion.

To develop new therapeutic strategies to combat cancer metastasis, a comprehensive understanding of the regulatory mechanism in metastasis from the metabolic reprogramming perspective is required. This study describes the metabolic reprogramming and interaction of cancer cells and non-tumor cells in the TME, as well as the development of treatment strategies aimed at altering metabolism. Significant advances in cancer prevention and treatment measures have been made in recent decades. However, the global cancer burden remains high, with over 90% of patients dying from cancer metastasis. Metastasis is the process by which cancer cells spread from their primary sites and grow in distant organs.

Cancer metastasis is a complex and dynamic cascade of multiple parallel overlapping steps that includes invading the circulatory system, surviving during hematogenous transit, penetrating through vascular walls into the parenchyma of a distant target organ, forming dormant or micro metastatic colonies, and finally proliferating into clinically detectable metastatic lesions. Cancer cells undergo complex biological changes during the metastatic cascade, causing them to adapt to an un favorable microenvironment. However, contrary to the traditional linear route that metastasis may occur in the late stage of tumours based on clinical manifestation of metastasis, increasing evidence shows that dissemination of cancer cells from primary to distant sites often occurs much earlier, even before clinical detection and diagnosis of primary tumour.

Although many cancer cells enter the metastatic cascade, only a few cells can reach distant organs and proliferate into micro-metastatic colonies, indicating that metastasis is inefficient. In response to nutritionally deficient environments, tumour cells undergo "metabolic reprogramming," which is a significant switch in a series of critical metabolic modes to meet the needs of biological function. One of the most important characteristics of tumour cells is metabolic reprogramming. Stephen Paget proposed the "seed and soil" hypothesis in 1889, believing that the Tumour Microenvironment (TME) is the "soil" on which the "seeds" (tumour cells) rely for survival. 8 Following research revealed that the formation of metastases necessitates the coordination of "seeds" and "soil."

The TME is a multicellular system made up of stromal cells, inflammatory immune cells, vasculature, and Extracellular Matrix (ECM). The TME acts as a scaffold and has powerful anti and pro-tumor metastasis effects. A growing body of evidence suggests that metabolic reprogramming occurs in a variety of TME cells. To some extent, the metabolic interaction between non-tumor and tumour cells promotes tumour metastasis. In this study, we discuss the metabolic reprogramming of glucose, lipids, and amino acids in cancer cells, as well as their interactions with non-tumor cells in TME. Furthermore, we briefly discuss the development of promising therapeutic strategies for cancer metabolic reprogramming.

In general, normal cells preferentially use the Oxidative Phosphorylation Pathway (OXPHOS) in an oxygen-rich environment and glycolysis when oxygen is scarce. However, in the 1920s, German scientist Otto Heinrich Warburg discovered that Hepatocellular Carcinoma (HCC) cells uptake a lot of glucose for glycolysis and produce metabolites like lactic acid (aerobic glycolysis), which he named the "Warburg effect." Following research, it was discovered that highly malignant or metastatic tumour cells have greater glycolytic ability than those with low metastatic capacity, implying a possible link between abnormal metabolism, invasion, and metastasis characteristics. A

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growing body of evidence suggests that abnormal glucose metabolism caused by micro environmental stimulation and gene dysregulation has a significant impact on cancer metastasis.

In conclusion, abnormal regulation of oncogenes and tumour suppressor genes is the primary cause of cancer cell metabolic reprogramming, which allows them to meet the demand for proliferation, invasion, and metastasis. Non-tumor cells in TME are also metabolically reprogrammed to promote or inhibit cancer metastasis. Targeting metabolic reprogramming is emerging as a malignant tumour prevention and treatment strategy. However, the molecular mechanisms underlying metabolic reprogramming in cancer cells and TME components remain unknown. Because intracellular metabolic modes are dynamic, reversible, time-sensitive, and heterogeneous, existing detection methods occasionally produce inaccurate results, posing challenges to preclinical research and clinical transformation.