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Commentary

Cancer Metabolism and Bioenergetics: Strategies for Therapeutic Targeting

Abela Hachemi*

Department of Oncology, Harvard University of Cambridge, Massachusetts, United States of America

DESCRIPTION

Cancer metabolism and bioenergetics are pivotal areas of study in oncology, providing new insights into how cancer cells fuel their rapid growth and survival. Unlike normal cells, cancer cells exhibit unique metabolic adaptations that allow them to thrive in challenging environments. By understanding these metabolic pathways, researchers are developing therapeutic strategies to disrupt cancer cell metabolism, potentially offering new avenues for cancer treatment. This article explores the fundamentals of cancer metabolism and bioenergetics and examines emerging strategies for therapeutic targeting.

Cancer metabolism

Cancer cells undergo numerous genetic and biochemical changes to sustain their high energy demands, evade the immune system, and promote growth. One hallmark of cancer metabolism is the "Warburg effect," where cancer cells rely on aerobic glycolysis even in the presence of ample oxygen. This metabolic reprogramming allows cancer cells to produce energy more quickly, supporting their rapid division, though it is less efficient than mitochondrial respiration.

Beyond the Warburg effect, cancer cells also display altered lipid, amino acid, and nucleotide metabolism. By reprogramming these pathways, cancer cells can efficiently generate the biomolecules they need for cell division and repair. For instance, increased glutamine metabolism supports nucleotide and amino acid synthesis, helping to sustain rapid proliferation. Understanding these pathways provides a foundation for developing therapies that specifically target cancer cell metabolism.

Bioenergetics in cancer cells

Bioenergetics focuses on how cells produce and use energy. In cancer cells, bioenergetics is often reconfigured to meet the demands of continuous growth and division. While normal cells rely heavily on oxidative phosphorylation in the mitochondria for efficient ATP production, cancer cells frequently shift to glycolysis, even under oxygen-rich conditions. This shift allows for rapid ATP generation but also provides essential building blocks for cell growth.

Cancer cells often exhibit increased mitochondrial biogenesis and alterations in mitochondrial dynamics, which support both energy production and cell survival. Additionally, hypoxic conditions within tumors where oxygen is scarce further promote metabolic reprogramming, increasing reliance on anaerobic pathways. This hypoxic adaptation not only aids survival in low-oxygen environments but also contributes to the aggressive nature of certain tumors.

Strategies for therapeutic targeting

Given the metabolic vulnerabilities of cancer cells, researchers are developing therapies that target these specific pathways. Some potential strategies include:

Inhibiting glycolysis: Since cancer cells depend heavily on glycolysis, drugs that inhibit glycolytic enzymes are being investigated as potential therapies. For instance, inhibitors of Hexokinase 2 (HK2), which catalyzes the first step of glycolysis, have shown promise in preclinical studies. Additionally, targeting Lactate Dehydrogenase (LDH), an enzyme involved in converting pyruvate to lactate, may disrupt the Warburg effect, impairing cancer cell growth.

Targeting glutamine metabolism: Many cancers rely on glutamine as a carbon and nitrogen source, making glutamine metabolism another attractive target. Inhibitors of glutaminase, an enzyme that converts glutamine to glutamate, have shown efficacy in disrupting cancer cell growth in glutamine-dependent tumors. These inhibitors may be particularly effective in cancers such as triple-negative breast cancer and glioblastoma, where glutamine metabolism plays a significant role.

Exploiting mitochondrial dysfunction: Mitochondrial metabolism remains essential for many cancer cells, especially those that shift between glycolysis and oxidative phosphorylation. Drugs that target mitochondrial enzymes, such as Pyruvate Dehydrogenase Kinase (PDK) inhibitors, can disrupt this metabolic flexibility, making cancer cells more vulnerable to treatment. Additionally, therapies that increase Reactive Oxygen

Correspondence to: Abela Hachemi, Department of Oncology, Harvard University of Cambridge, Massachusetts, United States of America, E-mail: hachemi_abela@gmail.com

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Species (ROS) within mitochondria can induce oxidative stress, triggering cell death in cancer cells.

Combining metabolic inhibitors with other therapies: Combining metabolic inhibitors with traditional treatments like chemotherapy or immunotherapy holds promise for enhancing treatment efficacy. For example, metabolic inhibitors can make cancer cells more sensitive to chemotherapy by disrupting their bioenergetic balance. Additionally, by altering the tumor microenvironment, these inhibitors may enhance immune responses, improving the effectiveness of immunotherapies.

CONCLUSION

Therapeutic targeting of cancer metabolism and bioenergetics represents an exciting frontier in oncology. By understanding the unique metabolic dependencies of cancer cells, researchers can develop treatments that selectively disrupt cancer growth while sparing normal cells. Although challenges remain, such as minimizing toxicity and overcoming resistance, these strategies offer a promising path toward more effective cancer therapies. With continued research, metabolic targeting could become an integral part of personalized cancer treatment, providing hope for improved outcomes in the fight against cancer.