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Understanding Metabolic Pathways in Cancer Cells: Implications for Clinical Practice

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Abstract

Metabolic reprogramming is a hallmark of cancer, driving cellular proliferation and survival. This research delves into the intricate metabolic pathways within cancer cells and their implications for clinical practice. Through a comprehensive review of current literature and experimental data, we elucidate the dysregulated metabolic processes that underpin cancer progression. Furthermore, we discuss the potential therapeutic targets emerging from this understanding and their application in clinical settings. This exploration of metabolic pathways in cancer cells provides critical insights for developing innovative strategies for cancer diagnosis, prognosis and treatment.

Keywords: Metabolic pathways • Cancer cells • Metabolic reprogramming

Introduction

LACTB, a unique mitochondrial protein evolved from bacterial penicillin-binding proteins and beta-lactamases, stands as the sole mammalian mitochondrial homolog. This serine beta-lactamase-like protein resides within the confines of the mitochondrial intermembrane space, where it assembles into enduring filaments. These filaments play pivotal roles in maintaining mitochondrial structure and governing lipid metabolism. The modulation of mitochondrial metabolism holds profound implications for comprehending cancer pathophysiology and progression. The metabolic rewiring observed in cancer cells exerts a profound influence on their behavior. In this article, we explore the clinical implications of LACTB in the context of neoplastic cell proliferation, migration, tumor growth, metastasis and responses to chemotherapeutic and immunotherapeutic agents. We also delve into the structural foundations of LACTB's activities and functions, shedding light on key regulatory mechanisms governing its actions.

Furthermore, we investigate the link between aberrant LACTB expression and the efficacy of anticancer medications, along with clinicopathological characteristics of cancer tissues. We provide a comprehensive perspective on the enzymatic properties, polymerization, mutations and epigenetic and post-translational modifications that underpin LACTB's role in cancer pathogenesis. The current pioneering research on LACTB's tumor-suppressing functions, structural basis and regulatory mechanisms presents a fresh perspective on its potential as a metabolic regulator. This perspective offers valuable insights into harnessing LACTB as a target for the development of neoadjuvant medicines and precision cancer therapies [1,2].

Description

The concept of endosymbiosis sheds light on the intriguing biological parallels between mitochondria and Gram-negative bacteria. This includes similarities in DNA organization, core metabolic processes and the double-membrane architectural blueprint. Within mammalian mitochondria, a singular counterpart to penicillin-binding proteins and beta-lactamases (PBP-Ls) exists, known as serine beta-lactamase-like protein (LACTB). Remarkably, LACTB, with its ancestral bacterial lineage, has undergone a remarkable shift from its original role in mediating lipid metabolism to orchestrating tumorigenic processes in mammals. This paradigm shift in LACTB's function has profound implications for our understanding of cancer biology [3]. By dissecting LACTB's influence on diverse cancer types, its involvement in responses to anticancer chemotherapies and immunotherapies and a comprehensive molecular analysis of its role in reprogramming lipid metabolism-a hallmark of cancer this perspective offers a fresh vantage point on LACTB's pivotal role in cancer progression, clinical prognosis and the development of precision cancer therapies and neoadjuvant interventions.

In the intricate realm of tumorigenesis, rogue cells serve as harbingers of tumor growth. The authenticity of a tumor suppressor gene is pivotal in this context. Low LACTB expression emerges as an ominous sign, typically linked to the expansion of rogue cells, while exerting minimal influence on non-tumorigenic counterparts. Notably, LACTB's heightened expression correlates with poor patient survival in the case of pancreatic adenocarcinoma and nasopharyngeal cancer, respectively [4].

Rogue cells inherit traits that align with their aberrant behavior and abilities, arising when they escape the constraints of normal cellular regulation. Drug resistance and malignant transformations are frequently driven by metabolic reprogramming. An enzyme called Phosphatidylserine Decarboxylase, responsible for converting phosphatidylserine into phosphatidylethanolamine and regulating their lyso-forms, shares functional ties with LACTB, thereby influencing phospholipid composition. The balance of lyso-phosphatidylethanolamine plays a pivotal role in regulating the differentiation of cardiac and neuronal PC12 cells. Reinstating LACTB expression curtails PISD activity, reducing PE/LPE levels and establishing a quiescent, tumor-suppressive state. Conversely, silencing LACTB increases PE/LPE levels in mitochondrial membranes, promoting cellular proliferation and upregulating CD44 (a marker for cancer stem cells) [5].

Conclusion

LACTB, a mitochondrial protease, orchestrates lipid metabolism by preventing the accumulation of PE/LPE lipid species. Beyond its influence on chemotherapeutic responses and immunoregulation, this perspective has delved into LACTB's effects on cellular proliferation and epithelial-mesenchymal transition. Typically, LACTB expression is associated with adverse cancer progression outcomes, though certain exceptions exist. The structural foundation of LACTB underpins its enzymatic properties, while upstream signaling pathways govern

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transcriptional, post-transcriptional and post-translational regulations affecting LACTB's expression and activity. These insights imply that comprehending LACTB's role in cancer pathogenesis necessitates a holistic examination, encompassing catalytic activity, functional dynamics, mutation profiles and polymorphism variations. This perspective offers innovative perspectives on LACTB's metabolic involvement in cancer progression and clinical prognosis. For instance, the LACTB-targeted approach could be synergistically employed with the development of drug delivery systems, such as transferrin/-tocopherol modified poly(amidoamine) dendrimers, to enhance tumor targeting precision. Furthermore, combining transferrin and octaarginine modification could create a dual-action strategy to improve therapeutic efficacy.

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Conflict of Interest

None.

References

1. Gray, Michael W. "Mitochondrial evolution." *Cold Spr Harbor Persp Bio* 4 (2012): a011853.
2. Peitsaro, Nina, Zydrune Polianskyte, Jarno Tuimala and Isabella Pörn-Ares, et al. "Evolution of a family of metazoan active-site-serine enzymes from penicillin-binding proteins: A novel facet of the bacterial legacy." *BMC Evolut Bio* 8 (2008): 1-11.
3. Liobikas, Julius, Zydrune Polianskyte, Oliver Speer and James Thompson, et al. "Expression and purification of the mitochondrial serine protease LACTB as an N-terminal GST fusion protein in Escherichia coli." *Prot Exp Purif* 45 (2006):335-342.
4. Keckesova, Zuzana, Joana Liu Donaher, Jasmine De Cock and Elizaveta Freinkman, et al. "LACTB is a tumour suppressor that modulates lipid metabolism and cell state." *Nat* 543 (2017):681-686.
5. Butler, Lisa M., Ylenia Perone, Jonas Dehairs and Leslie E Lupien, et al. "Lipids and cancer: Emerging roles in pathogenesis, diagnosis and therapeutic intervention." *Adv Drug Del Rev* 159 (2020):245-293.

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