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Metabolic Pathway in Clinical Cancer Cells

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Introduction

The only mammalian mitochondrial homolog that descended from bacterial penicillin-binding proteins and beta-lactamases (PBP-Ls) is serine beta-lactamase-like protein (LACTB). The active-site serine protease LACTB forms persistent filaments that are confined to the mitochondrion's intermembrane space (IMS), where they participate in the structure of the mitochondria and regulate lipid metabolism. Changes in mitochondrial metabolism are important for understanding the pathophysiology and development of cancer. The metabolic reprogramming of cancer cells influences their behaviour. The clinical effects of LACTB on neoplastic cell proliferation and migration, tumour growth and metastasis, as well as LACTB's role in chemotherapeutic and immunotherapeutic responses, are demonstrated in this article. Additionally, the structural underpinnings of LACTB activity and function are sketched, and pertinent regulatory mechanisms for LACTB are highlighted. The results of anticancer medicines and the clinicopathological characteristics of cancer tissues have both been linked to the aberrant expression of LACTB. The perspective suggests a great appeal of enzymic property, polymerization, mutation, and epigenetic and post-translational modifications in examining LACTB's role in cancer pathogenesis in light of the current pioneer research on the tumor-suppressed function, structural basis, and regulatory mechanism of LACTB. This viewpoint offers fresh insights into LACTB's potential as a metabolic regulator for developing neoadjuvant medicines or targeted cancer treatments.

Description

The endosymbiont concept clarifies the similar biological characteristics of mitochondria and Gram-negative bacteria, such as DNA organisation, core metabolism, and double-membrane architecture [1]. The sole homolog of penicillin-binding proteins and beta-lactamases (PBP-Ls) in mammalian mitochondria is the serine beta-lactamase-like protein (LACTB) [2]. As a result, the function of LACTB, the bacterial heritage, has been changed from mediating lipid metabolism to mediating tumorigenicity in mammals [2-4]. Through elucidating LACTB's influence on various cancers and involvement in the anticancer chemotherapeutic and immunotherapeutic responses, performing a molecular dissection of LACTB to streamline its role on reprogramming lipid metabolism, a hallmark in cancer [5], and revealing pertinent regulatory mechanisms, this perspective provides novel insights on LACTB in cancer progression and clinical prognosis and developing targeted cancer therapies or neoadjuvant therapeutic interventions.

Rogue cells get the body prepare for tumour growth. There is a difference when a tumour suppressor gene is genuine. Low LACTB expression indicates a poor prognosis since it is typically adversely correlated with the expansion of rogue cells while having little to no impact on non-tumorigenic cells. LACTB is highly expressed and associated with poor patient survival in pancreatic adenocarcinoma (PAAD) and nasopharyngeal cancer (NPC), respectively.

The progenitor traits of a rogue cell, which share its anomalous behaviours or skills, are acquired when it escapes a normal behavioural control. Drug resistance and malignant transformation are driven by metabolic reprogramming. Phosphatidylserine Decarboxylase (PISD), which converts phosphatidylserine (PS) to phosphatidylethanolamine (PE) and controls the amount of their lyso-forms, is functionally associated with LACTB and influences the composition of phospholipids [4]. The differentiation of cardiac and neuronal PC12 cells is regulated by lyso-phosphatidylethanolamine (Lyso-PE, LPE). While restoring LACTB expression decreases PISD activity and the buildup of PE/LPE, resulting in a quiescent-like tumour suppressive state, silencing LACTB increases the quantity of PE/LPE in mitochondrial membranes, promoting cellular proliferation and upregulating CD44 (the cancer stem cell maker) [4].

Conclusion

LACTB, a mitochondrial protease, controls lipid metabolism by preventing the build-up of PE/LPE lipid species. In addition to its impact on chemotherapeutic responses and immunoregulation, the perspective has explored the effects of LACTB on cellular proliferation and EMT. LACTB expression is typically connected with cancer progression adversely, however there are some exceptions. The structural basis of LACTB underpins its enzymatic properties, and upstream signalling displays transcriptional, post-transcriptional, and post-translational regulations on the expression and activity of LACTB. These findings suggest that, rather than focusing solely on its total expression, LACTB's role in cancer pathogenesis will be best understood in the context of its catalytic activity and function, polymerization dynamics, mutation, and polymorphism. In order to develop LACTB-targeted cancer therapies or neoadjuvant therapeutic interventions, this perspective offers novel insights on the metabolic role of the LACTB in cancer progression and clinical prognosis. For example, the LACTB-targeted approach could be combined with developing drug delivery systems, such as transferrin/-tocopherol modified poly(amidoamine) dendrimers to improve tumour targeting or transferrin and octaarginine modified dual.

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