

Aerobic Glycolysis Pathway Activation by Epstein-Barr virus Latent Membrane Protein 1 in Nasopharyngeal Carcinoma

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ABSTRACT

Glucose is a major source of energy to the cell growth and energy production. During tumors formation and cancer cell growth glycolysis significantly increases as well as glucose uptake and therefore increases biosynthesis, in addition to many more metabolic changes. These transformations are known as “Warburg effect” in presence of adequate oxygen (aerobic glycolysis).

LMP-1 regulates and promotes glycolysis through many pathways, genes, gene regulators, tumor suppressors, and enzymes such as PI3-K/Akt, Ras, mTOR, HIF-1 α , c-Myc, NF- κ B, p53, GSK3 β , FBW7, FGFR1, the M2 splice isoform of pyruvate kinase PKM2, PDHK1, Hexokinase 2 HK2, VHL and mutations of isocitrate dehydrogenase 1 (IDH1), succinate dehydrogenase (SDH) and fumarate hydratase (FH). Adding the capacity of EBV to induce aerobic glycolysis to its relevant role to induce cell proliferation and the inhibition of apoptosis, all these characteristics should prompt current research to improve the vaccination strategies against EBV.

The aim of this study is to analyze the most recent articles on how LMP1 can alter the signaling pathways of glycolysis in the nasopharyngeal carcinoma.

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