Evaluation of the role of 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase 3 in the Glioblastoma progression

Poster

Celular/Molecular, Neurona y glía - Cellular/Molecular, Neurons and glia

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Glioblastoma (GBM) is the most aggressive glioma affecting the central nervous system. Several features of this lethal tumor have been extensively studied. During GBM progression, cellular metabolism becomes dysregulated, with upregulation of proteins associated with high glycolytic flux. Among these, 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase 3 (PFKFB3) is a key enzyme involved in the regulation of glycolysis, acting as an allosteric activator of phosphofructokinase-1. Beyond its metabolic role, PFKFB3 has been widely characterized in cancer, where its increased expression is associated with enhanced malignant phenotypes. However, its specific role in GBM cells remains not fully understood. In this study, we evaluated the function of PFKFB3 during GBM progression using an in vitro approach. To investigate its role, we employed PFK15, a potent inhibitor of PFKFB3, and our results showed that inhibition of PFKFB3 significantly reduced cell viability (MTT assay) and proliferation (BrdU assay) after 24h and 48h of treatment in cultured GBM cells. Furthermore, wound healing assay results indicated decreased cell migration following 24h of

treatment with the inhibitor. Cell survival analyses, including TUNEL assay and flow cytometry, demonstrated increased apoptosis in PFK15-treated cells. Overall, our findings indicate that inhibition of PFKFB3 reduces cell viability and proliferation while increasing apoptosis in GBM cells in vitro. Understanding the role of PFKFB3 in the context of GBM may contribute to the identification of novel therapeutic targets for this disease.

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