PKN1

PKN1, also known as protein kinase N1, is a member of the protein kinase superfamily of enzymes. It plays a crucial role in various cellular processes by catalyzing the transfer of phosphate groups from ATP (adenosine triphosphate) to specific target proteins, a process known as phosphorylation. Phosphorylation can activate or deactivate proteins, thereby regulating their functions and influencing cell signaling pathways.

PKN1 is one of the isoforms of the protein kinase N (PKN) family, and it is classified as a serine/threonine kinase due to its preference for phosphorylating serine and threonine residues on target proteins.

Some of the cellular functions associated with PKN1 include:

Cell proliferation and growth: PKN1 can regulate cell division and proliferation through its involvement in various signaling cascades.

Cell migration: PKN1 is implicated in the regulation of cytoskeletal dynamics, which plays a critical role in cell movement and migration.

Cell survival and apoptosis: PKN1 can contribute to cell survival or cell death (apoptosis) depending on the context and the specific target proteins involved.

Insulin signaling: PKN1 has been shown to interact with components of the insulin signaling pathway, potentially influencing glucose metabolism.

Cancer: PKN1 dysregulation has been associated with various types of cancers, and it may play a role in promoting tumor growth and metastasis.

The functions of PKN1 can vary depending on the cellular context and the specific signaling pathways in which it is involved. Further research is still ongoing to fully understand its precise role in different biological processes and its potential as a therapeutic target in various diseases.

The role of PKN1 in gliomas has rarely been studied. Hao et al. suggest that PKN1 expression in glioma specimens is considerably upregulated and positively correlates with the histopathological grading of gliomas. Knocking down PKN1 expression in glioblastoma (GBM) cells inhibits GBM cell proliferation, invasion, and migration and promotes apoptosis. In addition, yes-associated protein (YAP) expression, an essential effector of the Hippo pathway contributing to the oncogenic role of gliomagenesis, was also downregulated. In contrast, PKN1 upregulation enhances the malignant characteristics of GBM cells and simultaneously upregulates YAP expression. Therefore, PKN1 is a promising therapeutic target for gliomas. Raloxifene (Ralo), a commonly used selective estrogen-receptor modulator to treat osteoporosis in postmenopausal women, was predicted to target PKN1 according to the bioinformatics team from the School of Mathematics, Tianjin Nankai University. They showed that Ralo effectively targets PKN1, inhibits GBM cell proliferation and migration, and sensitizes GBM cells to the major chemotherapeutic drug, Temozolomide. Ralo also reverses the effect of PKN1 on YAP activation. Thus, they confirm that PKN1 contributes to the pathogenesis of gliomas and may be a potential target for Ralo adjuvant glioma treatment [1].

1)

Hao Y, Li Z, Zhang A, Sun L, Wang G, Wang H, Jia Z. The role of PKN1 in glioma pathogenesis and the antiglioma effect of raloxifene targeting PKN1. J Cell Mol Med. 2023 Jul 21. doi: 10.1111/jcmm.17860. Epub ahead of print. PMID: 37480215.

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