Glioma pathogenesis

PKN1 (protein kinase N1), a serine/threonine protein kinase family member, is associated with various cancers. However, 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].

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see Gliomagenesis

Glioma angiogenesis

Glioma angiogenesis

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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.