KIF15

Kinesin family member 15 is a protein that in humans is encoded by the KIF15 gene.

This gene encodes a motor protein that is part of the kinesin superfamily. KIF15 maintains half spindle separation by opposing forces generated by other motor proteins. KIF15 co-localizes with microtubules and actin filaments in both dividing cells and in postmitotic neurons.

Kinesin family member 15 (KIF15) promotes proliferation in several cancers, but its effect on glioblastoma is unclear. In this study, differentially expressed gene analysis and network analysis were performed to identify critical genes affecting glioma progression. The samples were divided into a KIF15 high-expression group and KIF15 low-expression group, and the association between FIK15 expression level and clinical characteristics was summarized and analyzed by performing medical data analysis; the effect of KIF15 on glioblastoma cell proliferation was detected by employing colony formation and MTT assays. The effect of KIF15 on tumor growth in mice was determined. It was found that KIF15 was a potential gene affecting the progression of glioblastoma. In addition, KIF15 was highly expressed in glioblastoma tumor tissues, and KIF15 was correlated with tumor size, clinical stage and other clinical characteristics. After the KIF15 gene was knocked out, the proliferation ability of glioblastoma was significantly inhibited. KIF15 also contributed to the growth of glioblastoma tumors in mice. Therefore, we found KIF15 to be a promising clinical therapeutic target [1)].

In the present study, the role of kinesin-12 (KIF15) in glioma was revealed. Immunohistochemical staining and western blot analysis were used to detect the protein expression. An MTT assay was performed to evaluate cell proliferation. Flow cytometric analysis was utilized to assess cell apoptosis and the cell cycle. A mouse xenograft model was constructed for in vivo study. The results indicated that KIF15 was significantly upregulated in glioma tumor tissues and positively correlated with pathological staging, recurrence risk and poor prognosis. Silencing of KIF15 could inhibit cell proliferation and stemness of glioma cells, arrest cells in the G2 phase and induce cell apoptosis. The in vivo study verified the inhibitory effect of KIF15 knockdown on tumor growth. The mechanism study demonstrated the regulation of apoptosis- and cycle-related proteins in the KIF15 KD-induced inhibition of glioma. KIF15 was revealed to function as a tumor promoter in the development and progression of glioma. KIF15 also served as a prognostic indicator for glioma and may be a therapeutic target for glioma therapy [2)].

Stangeland et al. compared gene expression in GSCs to that in neural stem cells (NSCs) from the adult human brain, using microarrays. Bioinformatic filtering identified 20 genes (PBK/TOPK, CENPA, KIF15, DEPDC1, CDC6, DLG7/DLGAP5/HURP, KIF18A, EZH2, HMMR/RHAMM/CD168, NOL4, MPP6, MDM1, RAPGEF4, RHBDD1, FNDC3B, FILIP1L, MCC, ATXN7L4/ATXN7L1, P2RY5/LPAR6 and FAM118A) that were consistently expressed in GSC cultures and consistently not expressed in NSC cultures. The expression of these genes was confirmed in clinical samples (TCGA and REMBRANDT). The first nine genes were highly co-expressed in all Glioblastoma subtypes and were part of the same protein-protein interaction network. Furthermore, their combined up-regulation correlated negatively with patient survival in the mesenchymal Glioblastoma subtype. Using targeted proteomics and the

COGNOSCENTE database we linked these genes to Glioblastoma signalling pathways.Nine genes: PBK, CENPA, KIF15, DEPDC1, CDC6, DLG7, KIF18A, EZH2 and HMMR should be further explored as targets for treatment of Glioblastoma [3].

1)
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