

Kinesin superfamily protein 4 (KIF4), a KIF member classified in **Kinesin** 4 has been indicated as a mediator acted in the **tumorigenesis** of human **cancer**. The mechanism of **KIF4A** in **glioma** is yet to be investigated. Zhang et al. explored the potential function and mechanism of KIF4A in gliomas. They analyzed the KIF4A expression and the prognosis in glioma patients using **The Cancer Genome Atlas** (TCGA) databases. KIF4A levels in normal human **astrocyte** cell (NHA) and glioma cell lines were examined by **Western blot**. They studied the function of KIF4A on proliferation, migration, invasion, and cell cycle in glioma cell lines using a series of in vitro and in vivo experiments. Chromatin Immunoprecipitation (ChIP) analysis was applied to search potential KIF4A related downstream in glioma. They identified the significantly up-regulated expression of KIF4A both in glioma tissues and cells. Glioma patients with elevated KIF4A expression have shorter survival. Down-regulation of KIF4A exerted an inhibitory effect on cell proliferation, invasion, and migration. We crucially identified that KIF4A drives glioma growth by transcriptional repression of Rac1/Cdc42 to induce cytoskeletal remodeling in **glioma cells**. **Knockdown** of KIF4A decreased **RohA**, **Rac1**, **Cdc42**, **Pak1** and **Pak2** expression level. The study provided a prospect that KIF4A functions as an **oncogene** in glioma ¹⁾.

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Zhang H, Meng S, Chu K, Chu S, Fan YC, Bai J, Yu ZQ. KIF4A drives glioma growth by transcriptional repression of Rac1/Cdc42 to induce cytoskeletal remodeling in glioma cells. J Cancer. 2022 Nov 21;13(15):3640-3651. doi: 10.7150/jca.77238. PMID: 36606197; PMCID: PMC9809311.

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