Mir 489

The Glioblastoma cells were isolated and cultured in vitro, and then transfected with miR-489 inhibitor, miR-489 mimics and miR-negative control (NC) or TWIST1-small interfering RNA (siRNA) and TWIST1-NC. The expression levels of miR-489 and TWIST1 gene in the cells were measured via quantitative reverse transcription-polymerase chain reaction (qRT-PCR), and the proliferative capacity of cells in each group was detected by cell counting kit-8 (CCK-8) assay. Besides, the target gene TWIST1 of miR-489 was predicted to construct the luciferase reporter gene vectors of TWIST1 containing miR-489 target sites.

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Results: The expression level of miR-489 in Glioblastoma tissues and Glioblastoma cells isolated and cultured in vitro was remarkably lower than that in normal tissues and cells (p<0.01). The proliferative capacity of Glioblastoma cells was enhanced notably after inhibiting the expression of miR-489 (p<0.01), while it was obviously weakened by overexpressed miR-489 or TWIST1-siRNA (p<0.01). Moreover, the apoptosis rate was increased from $2.3\pm0.4\%$ to $19.6\pm1.2\%$ following miR-489 overexpression. TWIST1-siRNA could markedly down-regulate the expression level of TWIST1 (p<0.01) but evidently up-regulate the protein expression levels of Caspase-3 and Caspase-8 (p<0.01). The results of luciferase reporter assay manifested that miR-489 mimics significantly repressed TWIST1 (p<0.01).

MiR-489 can repress the proliferation and promote the apoptosis of glioma cells by targeting TWIST1 ¹⁾.

Xu et al. identified miR 489 as a direct target of ENST01108 and ENST01108 negatively regulate miR-489 by act as a sponge. SIK1 is verified as the direct target of miR-489 and it is negatively regulated by miR-489. ENST01108 also positively regulate SIKI and it promotes SIKI expression by suppressing miR-489. Taken together, the reciprocal repression of ENST011081 and miR-489 may be served as potential targets for cancer therapeutics in glioma²⁾.

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