miR 26b

Previous studies have established the role of microRNA (miR)-26b in various tumors, including breast cancer, lymphoma and glioma. Its function and mechanism in neuroglioma, however, remains to be elucidated. In the present study, in vitro cultured U87 glioma cells were randomly divided into miR-26b mimic, miR-26b inhibitor and respective control (NC) groups. MTT assay was performed to detect the effect of miR-26b on cell proliferation, while a cell invasion assay detected its effects on cell invasion. Caspase-3 activity was also quantified to test cell apoptosis, followed by reverse transcription-quantitative polymerase chain reaction and western blotting to detect the variation of Bcl-2 expression under the effect of miR-26b. miR-26b mimics transfection upregulated its expression in U87 cells, which had significantly reduced Bcl-2 mRNA and protein expression levels and higher casapse3 activity, and inhibited cell proliferation and invasion compared with the control group. The transfection of miR-26b inhibitor, in contrast, facilitated U87 cell proliferation and invasion, inhibited caspase-3 activity and elevated Bcl-2 mRNA/protein expression. In conclusion, miR-26 could facilitate apoptosis and inhibit proliferation/invasion of neuroglioma cells via downregulating Bcl-2 expression and potentiating caspase-3 activity [1)]

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microRNA miR-26b may act as a tumor suppressor in glioma. Low level expression of miR-26b has been found in glioma cells. The level of miR-26b is inversely correlated with the grade of glioma. EphA2 is a direct target of miR-26b. Over-expression of miR-26b in glioma cells represses the endogenous level of EphA2 protein. Ectopic expression of miR-26b inhibits the proliferation, migration, invasion and vasculogenic mimicry of human glioma cells [2)].

A study was done to evaluate the expression and molecular mechanisms of COX-2 and miR-26b in human GBM tissues and GBM cell lines T98G, U87 and U251. In the present study, Jiang et al., found that expression of miR-26b was markedly downregulated in GBM cell lines and human GBM tissues, compared to matched non-tumor associated tissues. Furthermore, miR-26b expression was inversely proportional to that of COX-2 mRNA and protein. Ectopic expression of miR-26b dramatically reduced the proliferation, colony formation, and proliferation/apoptosis-related proteins in GBM cells. Flow cytometry analysis showed that ectopic expression of miR-26b significantly decreased the percentage of S phase cells and increased the percentage of G1/G0 phase cells. Finally, luciferase reporter assay revealed that miR-26b inhibited the expression of COX-2 by binding to the 3'-UTR of COX-2 in GBM cells. Taken together, our results suggest that miR-26b plays an important role to inhibit the proliferation and invasion of GBM cells, and presents a novel mechanism for direct miR-26b-mediated suppression of COX-2 in GBM. Thus, miR-26b/COX-2 may have an important role in treatment of GBM patients [3)].

1)
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