mir 96

MiR-96 has been reported to exhibit a correlation with the Epithelial-mesenchymal-transition (EMT) process. However, the functional role of miR-96 and its mechanism in glioblastoma multiforme (GBM) remains to be completely elucidated. The objective of a study was to investigate the functional role and mechanism of miR-96 in the migration and invasion, in addition to proliferation, apoptosis and cell cycle distribution, of GBM. In the present study, the results suggested that the introduction of miR-96 significantly inhibited the migration and invasion, in addition to proliferation and cell cycle progression, of GBM cells and promoted their apoptosis in vitro, leading to the hypothesis that miR-96 may be a potential tumor suppressor. It was subsequently confirmed that astrocyte elevated gene 1 (AEG-1) was a direct target gene of miR-96, using a luciferase assay and reverse transcription-quantitative polymerase chain reaction analysis, in addition to western blotting. miR-96 was observed to downregulate the expression of AEG-1 at the mRNA and protein levels. Notably, AEG-1 may suppress EMT by increasing the expression levels of E-cadherin, an epithelial marker, and decreasing the expression levels of vimentin, a mesenchymal marker. Therefore, it was concluded that miR-96 may impede the EMT process by downregulating AEG-1 in GBM. Additionally, it was observed that inhibition of AEG-1 led to a similar effect compared with overexpression of miR-96 in GBM. In conclusion, the results of the present study demonstrated that miR-96 may act as a tumor suppressor by regulating EMT via targeting of AEG-1, suggesting that miR-96 may be a potential biomarker and anticancer therapeutic target for GBM in the future [1].

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