miR 16

miR-16, or microRNA-16, is a small RNA molecule involved in the regulation of gene expression. It is part of the microRNA family, which plays a crucial role in post-transcriptional gene regulation. miR-16 is found in various species, including humans.

Key points about miR-16 include:

Biological Function: miR-16 functions as a regulator of gene expression. It binds to the 3' untranslated region (UTR) of messenger RNA (mRNA) molecules and can lead to mRNA degradation or translational repression. By doing so, miR-16 can fine-tune the levels of specific proteins in the cell.

Tumor Suppressor: miR-16 is known to act as a tumor suppressor in various types of cancer. It can target and downregulate genes associated with cell proliferation, angiogenesis, and anti-apoptotic pathways. Dysregulation of miR-16 has been implicated in the development and progression of cancer.

Disease Associations: Besides cancer, miR-16 has been linked to other diseases, including cardiovascular diseases and neurological disorders. It can influence the expression of genes involved in these conditions.

Diagnostic and Therapeutic Potential: Because of its role in cancer and other diseases, miR-16 has attracted attention as a potential diagnostic biomarker and therapeutic target. Researchers are exploring its use in early cancer detection and the development of miRNA-based therapies.

miRNA Network: miR-16 is part of a complex regulatory network involving multiple microRNAs and their target genes. This network plays a vital role in maintaining cellular homeostasis and controlling various biological processes.

Research and Studies: Many studies have investigated miR-16 and its role in different physiological and pathological contexts. Research on miRNAs like miR-16 has expanded our understanding of gene regulation and its implications for health and disease.

In summary, miR-16 is a microRNA that plays a significant role in the regulation of gene expression, with particular relevance in cancer as a tumor suppressor. Its involvement in various biological processes and its potential as a diagnostic and therapeutic tool make it an important subject of study in molecular biology and medical research.

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miRNA-16, miRNA-143, and miRNA-200 showed statically significant higher expression among cases with cerebral aneurysms in comparison to controls. Thus, these preliminary results of miRNAs biomarkers are promising future tool to be used for aneurysmal screening [1)]

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miR-16 plays a significant role in tumors of various origins. This microRNA has been linked to various aspects of carcinogenesis, including cell apoptosis and migration. However, the molecular functions of miR-16 in gliomagenesis are largely unknown.

Neural stem cell-derived extracellular vesicles-mediated alleviation on neuronal injury by carrying miR-16-5p to target MYB was highly likely one of the mechanisms by which NSC-EVs mediated miR-16-5p in neuroprotection of depression rats [2].

The expression of miR-16 in human brain glioma tissues was lower than in non-cancerous brain tissues, and that the expression of miR-16 decreased with increasing degrees of malignancy.

The data suggest that the expression of miR-16 and nuclear factor (NF)-κB1 was negatively correlated with glioma levels. MicroRNA-16 decreased glioma malignancy by downregulating NF-κB1 and MMP9, and led to suppressed invasiveness of human glioma cell lines SHG44, U87, and U373.

The results also indicated that upregulation of miR-16 promoted apoptosis by suppressing BCL2 expression. Finally, the upregulation of miR-16 in a nude mice model of human glioma resulted in significant suppression of glioma growth and invasiveness. Taken together, the experiments have validated the important role of miR-16 as a tumor suppressor gene in glioma growth and invasiveness, and revealed a novel mechanism of miR-16-mediated regulation in glioma growth and invasiveness through inhibition of BCL2 and the NF-κB1/MMP-9 signaling pathway. Therefore, the experiments suggest the possible future use of miR-16 as a therapeutic target in gliomas [3].

In a study, qRT-PCR revealed that miR-16 was significantly downregulated in 23 glioma tissue specimens compared to 7 normal brain tissue specimens. Moreover, its levels were markedly lower in the glioma samples at stages T2-T4 compared to those at stage T1. The overexpression of miR-16 significantly suppressed the proliferation, migration and invasion of U251 and U87 glioma cells. Luciferase reporter assay identified Sal-like protein 4 (SALL4) as a target gene of miR-16, and its protein levels were found to be decreased in miR-16-overexpressing U251 and U87 cells. Furthermore, the overexpression of SALL4 significantly reversed the suppressive effects of miR-16 on the proliferation, migration and invasion of U251 and U87 cells, suggesting that miR-16 playsa tumor suppressor role in glioma by inhibiting cell proliferation and invasion through the targeting of SALL4. Finally, we found that SALL4 was significantly upregulated in glioma tissues compared to normal brain tissues, and its levels were markedly higher in the glioma tissues at stages T2-T4 compared to those at stage T1. In addition, the expression levels of SALL4 inversely correlated with the miR-16 levels in glioma tissues, suggesting that the downregulation of miR-16 contributes to the upregulation of SALL4 in glioma. On the whole, the findings of this study indicate a role for the miR-16/SALL4 axis in glioma. This data may also provide a potential therapeutic target for the treatment of glioma [4].

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