Maternally Expressed Gene 3 (MEG3)

Maternally Expressed Gene 3 (MEG3) is a gene that plays a crucial role in regulating various cellular processes. It is an imprinted gene, meaning that it is expressed or active only from one parent, specifically from the maternal allele. Imprinting is an epigenetic phenomenon where the expression of a gene depends on its parental origin.

MEG3 belongs to a class of genes known as long non-coding RNAs (lncRNAs). Unlike protein-coding genes that produce functional proteins, lncRNAs produce RNA molecules that do not code for proteins but instead play important regulatory roles in the cell. MEG3 produces a long non-coding RNA that has been implicated in the regulation of cell growth, differentiation, and apoptosis (programmed cell death).

Research suggests that MEG3 may function as a tumor suppressor, inhibiting the growth of cancer cells. Reduced expression of MEG3 has been observed in various types of cancers, and its overexpression has been shown to inhibit tumor cell proliferation. Additionally, MEG3 has been implicated in the regulation of other cellular processes, including angiogenesis and the cell cycle.

The precise mechanisms by which MEG3 exerts its regulatory effects are still an active area of research, and ongoing studies aim to elucidate its role in different biological contexts. Understanding the functions of genes like MEG3 is crucial for gaining insights into normal cellular processes as well as diseases, including cancer.

Maternally Expressed Gene 3 (MEG3) in glioma

Maternally Expressed Gene 3 (MEG3) is an imprinted gene located at 14q32 that encodes a noncoding RNA (ncRNA) associated with tumorigenesis of several cancers, including glioma.

- Prognostic significance of miR-378 in cancers: a meta-analysis
- Arenobufagin Induces Ferroptosis in Glioblastoma Cells via Modulating the MiR-149-5p/AEBP1 Axis
- LncRNA Meg3 Aggravates Renal Fibrosis Caused by Unilateral Ureteral Obstruction in Rats by Activating the Hedgehog Pathway
- scRNA-seq and proteomics reveal the distinction of M2-like macrophages between primary and recurrent malignant glioma and its critical role in the recurrence
- MicroRNA-592 Inhibits the Growth of Ovarian Cancer Cells by Targeting ERBB3
- Transfer RNA-derived small RNAs (tsRNAs) sequencing revealed a differential expression landscape of tsRNAs between glioblastoma and low-grade glioma
- The regulatory and modulatory roles of TRP family channels in malignant tumors and relevant therapeutic strategies
- Targeting cancer stem cells in medulloblastoma by inhibiting AMBRA1 dual function in autophagy and STAT3 signalling

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The purpose of a study was to evaluate the effect of MEG3 on glioma cells and the use of potential chemotherapeutics in glioma by modulating MEG3 expression. Cell viability, migration and chemosensitivity were assayed. Cell death was evaluated in MEG3 overexpressing and MEG3

suppressed cells. MEG3 expression was compared in patient-derived glioma cells concerning IDH1 mutation and WHO grades. Silencing of MEG3 inhibited cell proliferation and reduced cell migration while overexpression of MEG3 promoted proliferation in glioma cells. MEG3 inhibition improved the chemosensitivity of glioma cells to 5-fluorouracil (5FU) but not to navitoclax. On the other hand, there is no significant effect of MEG3 expression on temozolamide (TMZ) treatment which is a standard chemotherapeutic agent in glioma. Suppression of the MEG3 gene in patient-derived oligodendroglioma cells also showed the same effect whereas glioblastoma cell proliferation and chemosensitivity were not affected by MEG3 inhibition. Further, as a possible cell death mechanism of action apoptosis was investigated. Although MEG3 is a widely known tumour suppressor gene and its loss is associated with several cancer types, here we reported that MEG3 inhibition can be used for improving the efficiency of known chemotherapeutic drug sensitivity. They propose that the level of MEG3 should be evaluated in the treatment of different glioma subtypes that are resistant to effective drugs to increase the potential effective drug applications [1)].

In a study, MEG3 was downregulated in glioma tissue. In addition, downregulation of MEG3 was observed in human glioma cell lines compared with normal astrocyte cells. Furthermore, overexpressed MEG3 inhibited the proliferation, migration and invasion of glioma cells. Additionally, microRNA-96-5p (miR-96-5p) was a promising target of MEG3, and the promoting effects of miR-96-5p on cell growth and metastasis could be reversed by upregulated MEG3. Metastasis suppressor 1 (MTSS1) was predicted as the putative target of miR-96-5p, and its expression was restored by MEG3. In summary, the present data provided novel insight into the roles of MEG3 in glioma, and MEG3/miR-96-5p/MTSS1 signaling could be a promising therapeutic target for the treatment of patients with glioma [2)].

In a study Wang et al. assayed the expression of MEG3 in glioma tissue samples by real-time polymerase chain reaction assay, and defined the biological functions and target genes by CCK-8 assay, flow cytometry, and RNA immunoprecipitation. We first demonstrated that MEG3 expression was markedly decreased in glioma tissues compared with adjacent normal tissues. Moreover, ectopic expression of MEG3 inhibited cell proliferation and promoted cell apoptosis in U251 and U87 MG human glioma cell lines. We further verified that MEG3 was associated with p53 and that this association was required for p53 activation. These data suggest an important role of MEG3 in the molecular etiology of glioma and implicate the potential application of MEG3 in glioma therapy [3)].

The underlying mechanism of MEG3 in glioma remains elusive. In a study, MEG3 was found downregulated in glioma tissues compared with normal brain tissues. Downregulated expression of MEG3 was also detected in two human glioma cell lines (U-251, M059J) compared with normal astrocyte cells. MEG3 was then overexpressed by ligating to a lentiviral vector. Overexpressed MEG3 inhibited the proliferation of U-251 cells, and restrained the expression of proliferation marker proteins Ki67 and proliferating cell nuclear antigen (PCNA). However, cell apoptosis rate of U-251 cells and the expression of apoptosis marker proteins (caspase-3 and caspase-9) were elevated by MEG3. Furthermore, miR-93 was predicted a direct target of lncRNA-MEG3 by bioinformatics analysis. Overexpressed MEG3 counteracted the role of miR-93 in facilitating proliferation and inhibiting apoptosis in U-251 cells. Moreover, MEG3 restained the activation of phosphatidylinositol 3 kinase/protein kinase B (PI3K/AKT) pathway by reducing cytomembrane translocation of AKT. Finally, the in vivo experiment revealed that MEG3 strongly reduced tumor growth, tumor volume and the

expression of Ki67 and PCNA. lncRNA-MEG3 also inhibited the level of miR-93 and the expression of PI3K/AKT pathway related proteins in vivo. Taken together, the research indicated a MEG3-miR-93-PI3K-AKT pathway in regulating the growth of glioma, providing a promising therapy for glioma. [4].

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