Hsa_circ_0072309

Circular RNAs have been reported to play key roles in the progression of various cancers, including gliomas.

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hsa_circ_0072309 was remarkably downregulated in Glioblastoma. Hsa_circ_0072309 inhibits proliferation and invasion of glioblastoma and affects the cytoskeletal of Glioblastoma cells. Moreover, Yuan found that the function of hsa_circ_0,072,309 in Glioblastoma was associated with HSP27, which was reported to be an important regulator of cell proliferation, invasion, and cytoskeletal. The study provides a novel view of hsa_circ_0072309 in Glioblastoma cell proliferation and invasion, indicating that hsa_circ_0072309 may act as a potential therapeutic target for Glioblastoma comprehensive treatment [1)].

Yuan et al. designed a study to investigate the role of hsa_circ_0072309 in autophagy and temozolomide sensitivity in glioblastoma (Glioblastoma).

The effect of hsa_circ_0072309 on autophagy and TMZ sensitivity were examined by GFP-RFP-LC3, transmission electron microscopy(TEM), flow cytometry, Western blot, and immunofluorescence. The mechanism of hsa_circ_0072309 regulating the p53 signaling pathway was analyzed using Western blot, IP, and rescue experiments.

Low hsa_circ_0072309 expression predicts poor prognosis for glioma patients. The regulation of hsa_circ_0072309 on autophagy and TMZ sensitivity depends on the status of p53. Hsa_circ_0072309 promoted autophagy by p53 signaling pathway and enhanced sensitivity of glioblastoma to temozolomide (TMZ) in p53 wild-type Glioblastoma, but not in p53 mutant Glioblastoma. Hsa_circ_0072309 inhibits p53 ubiquitination and increases the stability of p53 protein in the context of p53 wild-type. MiR-100 mediates hsa_circ_0072309 regulating p53. P53 inhibitor or autophagy inhibitor could reverse the effect of hsa_circ_0072309 on TMZ sensitivity in p53 wild-type Glioblastoma.

This study revealed a function of hsa_circ_0072309 promoting autophagy by p53 signaling pathway and enhancing TMZ sensitivity. These findings demonstrated that hsa_circ_0072309 may be a potential and promising target in designing the treatment strategy for Glioblastoma. [2)].

The content of circ-0072309 in serum of patients with IS (n = 70) was measured by qRT-PCR, and the ROC curve was analyzed. LIFR humanized mice were used to measure the content of circ-0072309 in ischemic hemisphere by qRT-PCR and the protein expression of cleaved-caspase-3, cleaved-caspase-8 were detected by Western blot. After that, the expression of miR-100 in serum of patients with IS and in ischemic hemisphere of MCAO mice were detected, and then, we analyzed the correlation between the expression of circ-0072309 and miR-100. The binding sites between circ-0072309 and miR-100 were predicted by online database. We detected whether cric-0072309 bind to miR-100 by Dual-Luciferase report in bEnd2. In addition, bEnd2 was treated with oxygen-glucose deprivation (OGD) to simulate injury of cerebral vascular after cerebral ischemia. After treated with miR-100 mimic or miR-100-inhibitor, we detected the cell survival and rate of cell apoptosis, and the content of cleaved-

caspase-3 and caspase-8 protein. The target mRNA of miR-100 was predicted by bioinformatics analysis and analyzed by Dual-Luciferase. After treating bEnd2 with circ-0072309 and miR-100 mimic, we analyzed the cell survival and apoptosis to identify the potential regulatory mechanism.

Results: The results of qRT-PCR showed that the expression of circ-0072309 was significantly decreased while the content of miR-100 was significantly increased in the serum of IS patients and in the ischemic hemisphere of MCAO mice. There was a negative correlation between the expression of circ-0072309 and miR-100. The results of Dual-Luciferase showed that circ-0072309 could directly bind to miR-100. After treating bEnd2 with OGD, miR-100-mimic caused a decrease rate of cell survival and an increased rate of apoptosis. Dual-Luciferase showed that miR-100 regulated cell survival and apoptosis by directly binding to mTOR. By comparing treated bEnd2 with circ-0072309, co-transfected bEnd2 with circ-0072309 and miR-100 reduced cell survival and increased apoptosis.

Conclusions: According to these results, this study revealed that the circ_0072309-miR-100-mTOR regulatory axis could alleviate IS, and it may be a potential target for the treatment of IS. [3].

Findings revealed for the first time that the hsa_circ_0072309-miR-492 axis plays an essential role in breast cancer progression [4].

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