miR 145

TNF-α, miR-145-5p, and Nurr1 expressions were examined by western blot and quantitative real-time polymerase chain reaction. Western blotting and dual-luciferase reporter gene assay were conducted to examine the regulating impact that miR-145-5p had on Nurr1 and TNF-α. MiR-145-5p was remarkably upregulated in the SCI rat model's spinal cord tissues and BV2 cells treated with LPS, and Nurr1 expression was dramatically lowered. Furthermore, miR-145-5p inhibition markedly repressed inflammatory and oxidative stress responses. Moreover, it was proved that Nurr1 was a direct miR-145-5p target. The inhibition of miR-145-5p helped promote Nurr1 expression to block TNF-α signaling. MiR-145-5p inhibition mitigates inflammation and oxidative stress via targeting Nurr1 to regulate TNF-α signaling, which ameliorates SCI [1)]

Knockdown of circular RNAs CEP128 suppresses cell proliferation and improves the cytotoxicity efficacy of temozolomide in glioma cells by regulating miR 145-5p, suggesting that circular RNA CEP128 might be a promising target for overcoming the resistance of glioma cells to temozolomide [2)].

MicroRNA and aneurysm

The molecular mechanisms behind intracranial aneurysm formation and rupture remain poorly understood.

The MicroRNA and mRNA interactions and expression levels in cerebral aneurysm tissue from human subjects were profiled.

A prospective case-control study was performed on human subjects to characterize the differential expression of mRNA and MicroRNA in unruptured cerebral aneurysms in comparison with control tissue (healthy superficial temporal arteries [STA]). Ion Torrent was used for deep RNA sequencing. Affymetrix MicroRNA microarrays were used to analyze MicroRNA expression, whereas NanoString nCounter technology was used for validation of the identified targets.

Overall, 7 unruptured intracranial aneurysm and 10 STA specimens were collected. Several differentially expressed genes were identified in aneurysm tissue, with MMP-13 (fold change 7.21) and various collagen genes (COL1A1, COL5A1, COL5A2) being among the most upregulated. In addition, multiple MicroRNAs were significantly differentially expressed, with miR 21 (fold change 16.97) being the most upregulated, and miR 143-5p (fold change -11.14) being the most downregulated. From these, miR-21, miR-143, and miR 145 had several significantly anticorrelated target genes in the cohort that are associated with smooth muscle cell function, extracellular matrix remodeling, inflammation signaling, and lipid accumulation. All these processes are crucial to the pathophysiology of cerebral aneurysms.

This analysis identified differentially expressed genes and MicroRNAs in unruptured human cerebral aneurysms, suggesting the possibility of a role for MicroRNAs in aneurysm formation. Further investigation for their importance as therapeutic targets is needed [3)].

1)

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