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PRINT: ISSN 0972-3757 ONLINE: ISSN 2456-6330

Int J Hum Genet, 24(1): 63-72 (2024)

DOI: 10.31901/24566322.2024/24.01.809

Participation of MiR-145-5p in Neuronal Ischemia/Reperfusion Injury by Targeting Fibroblast Growth Factor-5

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KEYWORDS Fibroblast Growth Factor-5. Ischemia/Reperfusion Injury. miR-145-5p. Neuron. Oxidative Stress

ABSTRACT The researchers made efforts in elucidating the mechanism for miR-145-5p participating in neuronal ischemia/reperfusion injury (I/R) via targeting fibroblast growth factor-5 (FGF5). OGD/R was performed to construct the neuronal injury model. After OGD/R, the oxidative stress injury of nerve cells was enhanced significantly, the levels of lactic dehydrogenase, reactive oxygen species and malondialdehyde rose, while the superoxide dismutase activity decreased. Both miR-145-5p and cell apoptosis rate increased. MiR-145-5p had a targeting relation to FGF5. After miR-145-5p inhibitor transfection of nerve cells, inhibition of oxidative stress together with apoptosis was observed. FGF5 siRNA could reverse the impact on oxidative stress, FGF5, and apoptosis exerted by miR-145-5p-inhibitor in the OGD/R model. Being a latent target of I/R treatment, miR-145-5p induces the oxidative stress injury as well as apoptosis of HT22 cells by targeting and binding FGF5.