عنوان مقاله:

Quercetin protects PC-12 cells against hypoxia injury by down-regulation of miR-122

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خلاصه مقاله:

Objective(s): Impairment of nerve cells of brain induced by hypoxia results in energy-deprivation and dysfunction, which accompanies with neurons apoptosis. Improving function of nerve cells is important for treating cerebral anoxia. This study aimed to investigate the role of Quercetin (Quer) in hypoxia-induced injury of pheochromocytoma (PC-12) cells. Materials and Methods: PC-12 cells were cultured under anoxic condition for induction of hypoxia injury and/or treatment with Quer, transfection with pre-miR-122, anti-miR-122 or their negative controls. After Quer treatment, viability, migration, and cell apoptosis of PC-12 cells were analyzed by CCK-8 assay, transwell assay and flowcytometry analysis, respectively. Cell proliferation-associated proteins and cell apoptosis-associated proteins were analyzed by Western blot. Relative miR-122 expression in Quer-treated cells or transfection efficacy of miR-122 was analyzed by qRT-PCR. Finally, main components in AMP-activated protein kinase (AMPK) and Wnt/β-catenin signaling pathways were analyzed by Western blot.Results: Quer alleviated hypoxia-induced injury in PC-12 cells by increasing viability, promoting cell proliferation, enhancing migration and repressing apoptosis. Also, miR-122 was down-regulated in Quer-treated cells. miR-122 overexpression decreased cell viability and migration, and increased cell apoptosis in hypoxia- treated PC-12 cells, but miR-122 silencing led to the opposite results. We also found that AMPK and Wnt/β-catenin signaling pathways were activated by Quer in hypoxia-induced injury, but were inactivated in hypoxia-induced cells by overexpression of miR-122.Conclusion: Quer could repress hypoxia-induced injury in PC-12 cells by activating AMPK and Wnt/β-catenin signaling pathways via down-regulation of miR-122.

کلمات کلیدی:

AMPK pathway, Hypoxia injury, miR-122, Quercetin, Wnt/β-catenin pathway

لینک ثابت مقاله در پایگاه سیویلیکا: