



中文标题 检索 药刊检索

木犀草素对缺血再灌注损伤神经元的保护作用及机制研究

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中文摘要:目的:探讨黄酮类化合物木犀草素对缺血/再灌注损伤神经元的作用以及可能的作用机制。方法:原代培养的海马神经元经2 h的氧糖剥夺和24 h的再灌注处理,分别检测细胞活性、乳酸脱氢酶漏出率和细胞凋亡率,采用Pulsine(1)四动豚阻断法对SD大鼠施行10 min全脑缺血和24 h再灌注,检测钠泵活性。结果:经氧糖剥夺/再灌注后,海马神经元的活性比未经氧糖剥夺/再灌注组显著降低,乳酸脱氢酶漏出率和细胞凋亡率均显著地增高。在氧糖剥夺期间给予的木犀草素,在10~100 μmol·L⁻¹能呈剂量依赖地逆转这些改变。进一步实验发现木犀草素能够显著地增强大鼠全脑缺血/再灌注后的钠泵活性。在神经元氧糖剥夺/再灌注模型上,利用钠泵抑制剂哇巴因能阻断木犀草素的神经元保护作用。结论:木犀草素能减轻缺血/再灌注所致的神经元损伤,其保护机制可能与增强了神经元细胞膜上的钠泵活性有关。

中文关键词:木犀草素 缺血 神经元 钠泵

Protective effects of luteolin on neurons against oxygen-glucose deprivation/reperfusion injury via improving Na⁺/K⁺-ATPase activity

Abstract:Objective: Luteolin, a flavone, has considerable neuroprotective effects by its anti-oxidative mechanism. However, it is still unclear whether luteolin can protect neurons against oxygen-glucose deprivation/reperfusion (OGD/R) induced injury. Method: After 2 hours oxygen-glucose deprivation and 24 hours reperfusion treatment in primary cultured hippocampal neurons, the neuron viability, survival rate and apoptosis rate were evaluated by MTT assay, lactate dehydrogenase (LDH) leakage assay and hoechst staining, respectively. The activity of Na⁺/K⁺-ATPase was examined in cultured neurons or in the hippocampus of SD rats treated by 10 minutes global cerebral ischemia and followed 24 hours reperfusion. Result: Treatment by OGD/R markedly reduced neuronal viability, increased LDH leakage rate and increased apoptosis rate. Application of luteolin (10-100 μmol·L⁻¹) during OGD inhibited OGD/R induced neuron injury and apoptosis in a dose-dependent manner. Compared to the control group or OGD/R-treated neurons, the activity of Na⁺/K⁺-ATPase was significantly suppressed in global ischemia/reperfusion group or OGD/R-treated neurons. Application of luteolin during ischemia or OGD preserved the Na⁺/K⁺-ATPase activity. Furthermore, inhibition of Na⁺/K⁺-ATPase with ouabain attenuated the protective effect afforded by luteolin. Conclusion: The data provide the evidence that luteolin has neuroprotective effect against OGD/R induced injury and the protective effect may be associated with its ability to improve Na⁺/K⁺-ATPase activity after OGD/R.

keywords:luteolin ischemia neuron Na⁺/K⁺-ATPase

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