地黄寡糖对脑缺血再灌注所致痴呆大鼠学习记忆功能的影响

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摘要　　目的　研究地黄寡糖对脑缺血再灌注致痴呆大鼠学习记忆功能的影响及其可能机制。方法　采用ip硝普钠及双侧颈总动脉夹闭10 min-再灌10 min-夹闭10 min的方式制备脑损伤模型。地黄寡糖6.4，32.0或160.0 mg·kg^{-1}于造模前3 d至造模后7 d ip给药，每日1次，共10 d。于术后d 7开始进行水迷宫实验测定大鼠学习记忆能力；术后d 10取海马，HPLC异硫氰酸苯酯柱前衍生法测定海马谷氨酸（Glu）含量；Western蛋白印迹法测定海马磷酸化细胞外信号调节酶2（p-ERK2）含量。结果　模型组大鼠学习记忆能力明显下降，海马Glu含量明显升高，p-ERK2含量降低。地黄寡糖可剂量依赖性地增强缺血再灌注损伤大鼠的学习记忆能力，降低海马Glu含量，提高海马p-ERK2含量。结论　地黄寡糖可以改善脑缺血再灌注致痴呆大鼠的学习记忆能力，这种作用可能与抑制Glu过量释放、进而使ERK2信号途径正常发挥有关。

关键词　　地黄寡糖　脑缺血　再灌注损伤　学习　记忆　谷氨酸盐类　细胞外信号调节酶　海马

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Effect of *Rehmannia glutinosa* oligosaccharides on learning and memory abilities in rats with focal cerebral ischemia and reperfusion injury

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Abstract

　AIM To investigate the effect of *Rehmannia glutinosa* oligosaccharides (ROS) on learning and memory abilities in rats with focal cerebral ischemia-reperfusion injury and its possible mechanism. **METHODS** Rat model of ischemia-reperfusion injury was established by 10 min ischemia-10 min reperfusion-10 min ischemia (I-R-I) of bilateral common carotid arteries and pretreated (ip) with sodium nitroprusside 2.5 mg·kg^{-1}. The rats were given (ip) ROS (6.4, 32.0 or 160.0 mg·kg^{-1}) once daily for 10 d (from 3 d prior to 7 d after I-R-I). On d 7-9 after I-R-I, learning and memory abilities of rats were tested by Morris water maze. On d 10 after I-R-I, hippocampal glutamate (Glu) content and hippocampal phospho-extracellular signal regulated kinase 2 (p-ERK2) expression were detected by HPLC and Western blot, respectively, after finishing Morris water maze test. **RESULTS** Compared with the sham group, learning and memory abilities of model group were decreased, hippocampal Glu content was increased, and hippocampal p-ERK2 expression was decreased significantly. ROS treatment significantly improved the learning and memory abilities of rat with I-R-I injury, decreased hippocampal Glu content, and increased hippocampal p-ERK2 expression. **CONCLUSION** ROS can improve learning and memory abilities of rats with I-R-I injury, which may be related with inhibiting the elevation of hippocampal Glu content and sustain the ERK2 signaling pathways.

Key words　Rehmannia glutinosa oligosaccharides　cerebral ischemia　reperfusion injury　learning　memory　glutamates　extracellular signal　regulated kinase　hippocampus

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