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论文

紫草酸镁B抑制缺血/再灌注心肌细胞c-Jun N-末端激酶3 mRNA的表达

杨丽敏;肖耀龙;欧阳家惠

1. 华中科技大学 同济医学院 病理学系, 湖北 武汉 430030； 2. 香港大学 药理学系, 香港

摘要：

目的探讨c-Jun N-末端激酶3(c-Jun N-terminal kinase 3, JNK3)在缺血/再灌注心肌细胞损伤中的作用,及紫草酸镁B对缺血/再灌注心脏具有保护作用的机制。方法复制大鼠Langendorff心肌缺血/再灌注损伤模型, 用原位杂交技术检测心肌细胞JNK3 mRNA的表达，并观察紫草酸镁B对JNK3表达的影响。结果图像分析显示，心肌缺血30 min/再灌注30 min时，JNK3表达明显高于非灌注组和对照组。0.1,1和10 μmol·L^{-1}紫草酸镁B可以抑制缺血/再灌注时JNK3 mRNA的表达。结论紫草酸镁B可通过抑制JNK3的表达以降低JNK的功能，从而减少心肌细胞凋亡的发生，对缺血/再灌注心脏产生保护作用。

关键词： c-Jun N-末端激酶3 心肌细胞 缺血/再灌注 紫草酸镁B

Inhibition of magnesium lithospermate B on the c-Jun N-terminal kinase 3mRNA expression in cardiomyocytes encountered ischemia/reperfusion injury

YANG Li-min; XIAO Yao-long; OU-YANG Jia-hui

Abstract:

AimTo study the function of c-Jun N-terminal kinase 3 (JNK3) in the process of ischemic/reperfused heart injury and the mechanism underlying the protective action of magnesium lithospermate B (MTB), a bioactive compound isolated from Danshen. MethodsBy in situ hybridization, JNK3 mRNA was detected in the ventricular preparations of the Langendorff ischemic/reperfused rat heart. The inhibitory effect of MTB on the expression of JNK3 mRNA was also investigated. ResultsThe purple and blue hybridization signals were located in the cytoplasm of the cardiomyocytes, which were weaker in the non-perfused hearts and stronger in the hearts encountered 30 min of ischemia and 30 min of reperfusion. Image analysis showed that the expression of JNK3 mRNA in the cardiomyocytes increased after 30 min of ischemia and 30 min of reperfusion, which showed significant difference compared with that in the cardiomyocytes of the non-perfused heart and the control heart ($P<0.05$). Treatment with of 0.1， 1 and 10 μmol·L^{-1} MTB abolished the elevation of JNK3 mRNA expression in the ischemic/reperfused heart ($P<0.05$). ConclusionJNK3 may be another component in the signal transduction pathway of ischemia/reperfusion induced cardiomyocyte apoptosis. MTB may protect the heart from ischemia/reperfusion injury by reducing apoptosis through inhibition of the JNK3 activity.

Keywords: cardiomyocyte ischemia/reperfusion magnesium lithospermate B c-Jun N-terminal kinase 3

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通讯作者: 杨丽敏

作者简介:

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