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1-磷酸鞘氨醇后适应对H9c2心肌细胞缺氧/复氧损伤的保护作用

Protective Effects of Sphingosine-1-phosphate Postconditioning on Hypoxia / Reoxygenation Injury in Rat H9c2 Cardiomyocytes

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英文关键词：S1P hypoxia /reoxygenation cardiomyocytes apoptotic intracellular calcium HSP70

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中文摘要:

目的 研究1-磷酸鞘氨醇(S1P)后适应对H9c2心肌细胞缺氧复氧损伤的保护作用,并探讨其作用机制。方法 将培养的H9c2心肌细胞随机分为5组,即①正常(Con)组;②缺氧/复氧(H/R)组;③S1P低浓度(L)组;④S1P中浓度(M)组;⑤S1P高浓度(H)组。测定各组H9c2心肌细胞的存活率; 收集细胞培养液测定超氧化物歧化酶(SOD)活性和丙二醛(MDA)含量;流式细胞仪检测心肌细胞凋亡百分率;Fura 2-AM标记细胞内游离钙离子,检测荧光强度以反映细胞内游离钙离子浓度的变化;Western Blot法测定保护性蛋白热休克蛋白70 (HSP70)的表达情况。结果 对于H/R 损伤的H9c2心肌细胞,S1P能够提高细胞的存活率,降低细胞内MDA含量及细胞内钙离子浓度,提高细胞内SOD活力,增强抗凋亡蛋白HSP70的表达,且呈一定的浓度依赖性。结论 S1P可以减轻心肌细胞氧化应激损伤,改善心肌细胞活力并减少凋亡。S1P对心肌细胞的保护作用可能是通过减少Ca2 超负荷,增加抗凋亡蛋白HSP70表达来实现的。

英文摘要:

ABSTRACT: OBJECTIVE To investigate protective effects and mechanism of sphingosine 1-phosphate (S1P) postconditioning on H9c2 cardiomyocytes exposed to hypoxia/reoxygenation injury. METHODS H9c2 cardiomyocytes were randomly divided into five groups: ①Control group; ②Hypoxia /reoxygenation (H/R) group; ③S1P low concentration (L)group; ④S1P midlle concentration (M) group; ⑤S1P high concentration (H) group. The survival rates of cardiomyocytes were detected. The activity of

superoxide dismutase (SOD) and the content of malondialdehyde (MDA) in the culture medium was measured. The apoptotic percentage was measured with flow cytometry. The free intracellular calcium ions were labeled by Fura 2-AM and the fluorescent intensity produced by Fura 2-AM was measured, which reflected the changes of the concentration of the free intracellular calcium ions. Western blot was performed to examine expression of heat shock protein 70 (HSP70) in cardiomyocytes respectively. RESULTS S1P could protect H9c2 cardiomyocytes against hypoxia/eoxygenation injury , increase cell survival rate, decrease the content of MDA and free intracellular calcium, increase the activity of SOD, increase the expression of HSP70, with a certain concentration dependent manner. CONCLUSION S1P could reduce hypoxia/reoxygenation-induced oxidative stress in cardiac myocytes, improve myocardial viability and reduce the apoptotic rate. The mechanism may depend on reduce intracellular calcium overload , increase the expression of HSP70.

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