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论著

CFTR氯通道在硫化氢诱导的心肌保护及细胞增殖中的作用

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摘要 目的： 探讨囊性纤维化跨膜传导调节因子（CFTR）氯通道在硫化氢（H2S）诱导的心肌保护及细胞增殖中的作用。方法：应用氯化钴（CoCl2）在大鼠H9c2心肌细胞建立化学性缺氧损伤心肌细胞实验模型；CCK-8试剂盒检测心肌细胞存活率；Hoechst 33342核染色法检测心肌细胞凋亡。结果：在400-2 000 μmol/L浓度范围内，CoCl2呈剂量依赖性地抑制H9c2心肌细胞的存活率，600 μmol/L CoCl2 能诱导H9c2心肌细胞产生明显的凋亡；在100-800 μmol/L浓度范围内，硫氢化钠（NaHS）呈剂量依赖性地促进H9c2心肌细胞增殖；NaHS能保护H9c2心肌细胞对抗CoCl2引起的细胞损伤作用，使细胞存活率升高，凋亡率降低；100 μmol/L CFTR氯通道拮抗剂5-硝基-2-（3-苯丙胺）-苯甲酸（NPPB)能明显地阻断NaHS对CoCl2的细胞毒性的抑制作用，但不能阻断NaHS抗心肌细胞凋亡作用及促进心肌细胞增殖作用。结论：CFTR氯通道可能参与H2S的抗CoCl2引起的心肌细胞毒性作用。

关键词 氯通道； 硫化氢； 心肌保护； 细胞增殖 氯化钴
分类号 R363

Roles of CFTR Cl- channels in hydrogen sulfide-induced cardioprotection and cell proliferation in H9c2 cells

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Abstract

<FONT face=Verdana>AIM: To explore the roles of cystic fibrosis transmembrane conductance regulator (CFTR) Cl- channels in hydrogen sulfide (H2S)-induced cardioprotection and cell proliferation in H9c2 cells. METHODS: Cobalt chloride (CoCl2) was used to set up the chemical hypoxia-induced injury model in H9c2 cells. Myocardial cell viability was detected by the CCK-8 assay kit. Apoptotic changes in H9c2 cells were observed by using Hoechst 33342 staining and photofluorography. RESULTS: At the concentrations from 400 to 2 000 μmol/L, CoCl2 dose-dependently inhibited cell viability in H9c2 cells. CoCl2 at concentration of 600 μmol/L significantly induced H9c2 cell apoptosis. Sodium hydrosulfide (NaHS) at concentrations from 100 to 400 μmol/L dose-dependently enhanced proliferation in H9c2 cells. NaHS protected H9c2 cells against CoCl2-induced injury, including an increase in cell viability and a decrease in percentage of apoptosis. 5-nitro-2-(3-phenylpropylamino)-benzoic acid (NPPB, 100 μmol/L), an inhibitor of CFTR Cl- channels alone did not damaged H9c2 cells, but considerably blocked the inhibitory effect of NaHS on CoCl2 cytotoxicity. However, NPPB did not antagonize the NaHS-induced antiapoptotic effect and cell proliferation in H9c2 cells. CONCLUSION: CFTR Cl- channels may be involved in the inhibitory effect of H2S on CoCl2-induced cytotoxicity in H9c2 cells. </FONT>

Key words Chloride channels Hydrogen sulfide Cardioprotection Cell proliferation Cobalt chloride

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