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论文

17-β-雌二醇对心肌缺氧/复氧NF-κB及ICAM-1和VCAM-1表达的影响

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摘要:

利用乳鼠原代培养心肌细胞建立缺氧/复氧模型, 采用流式细胞术、Western blot、RT-PCR及Elisa等方法, 研究了17-β-雌二醇对缺氧/复氧诱导的ICAM-1和VCAM-1表达的影响, 分析雌激素在心肌缺氧/复氧损伤中的抗炎作用. 结果表明, 心肌缺氧/复氧使NF-κB p65活化, 雌激素对NF-κB活化有明显的抑制作用. 雌激素对NF-κB途径和对非NF-κB途径ICAM-1和VCAM-1的mRNA和蛋白表达都有抑制作用. 以上结果说明雌激素在心肌缺氧/复氧过程中的抗炎作用是通过多途径实现的.

关键词: 17-β-雌二醇; 心肌细胞; 缺氧/复氧; 核转录因子-κB; 黏附分子

Effect of 17-β-Estradiol on Hypoxia-reoxygenation-induced NF-κB Activity, ICAM-1 and VCAM-1 Expression in Cardiac Myocytes

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Abstract:

Transcription factor(NF)-κB plays an important role during hypoxia-reoxygenation in cardiac myocytes. In previous studies, it has been proved that NF-κB can bind to the specific sequences located in the promoters of ICAM-1 and VCAM-1, ediate the expression of tow adhesion molecules, and cause the cell inflammatory response and apoptosis. In order to analyze the anti-inflammation function of 17-β-estradiol during hypoxia-reoxygenation, we established the hypoxia-reoxygenation model in cardiac myocytes which were isolated from neonatal rat. And then, we detected the effect of 17-β-estradiol on activation of NF-κB p65 and studied the estrogen on how to adjust the level of ICAM-1 and VCAM-1 by Flow Cytometry, Western Blot, RT-PCR and Elisa technology. The results show that the NF-κB pathway is activated during hypoxia-reoxygenation, and 17-β-estradiol inhibits this activation significantly. 17-β-Estradiol also inhibits the expression of ICAM-1 and VCAM-1 by both NF-κB-dependent and NF-κB-independent pathway. Taken together, all results suggested that anti-inflammatory effect of 17-β-estradiol during myocardial hypoxia-reoxygenation is achieved through multichannels.

Keywords: 17-β-Estradiol; Cardiac myocytes; Hypoxia-reoxygenation; NF-κB; Adhesion molecule

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