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基础医学

血管紧张素Ⅱ通过NF-κB信号传导途径促进人脐静脉内皮细胞内皮脂肪酶表达

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摘要:

目的 探讨血管紧张素Ⅱ促进内皮脂肪酶(EL)表达的信号传导通路。方法 体外培养人脐静脉内皮细胞(HUVECs)分为3组: ①血管紧张素Ⅱ(Ang II)刺激组: 在培养液中加入Ang II, 使之终浓度为 $10\mu\text{mol/L}$; ②PDTC预处理组: 核因子(NF-κB)抑制剂吡咯烷二硫氨基甲酸(PDTC)(10mmol/L)预处理HUVECs 1h后加入Ang II($10\mu\text{mol/L}$)刺激; ③对照组: 不加任何刺激因子。上述各组细胞分别孵育2、4、8、12、24h后终止实验, 收集细胞, 采用western blot法检测不同时间各组EL、NF-κB p65(NF-κB p65)的表达。结果 ①Ang II可以上调HUVECs的EL、NF-κB p65的表达, 两者的升高趋势一致。HUVECs经Ang II刺激后, 在4、8、12h其EL的表达量较对照组明显增加($P < 0.05$); 2、4、8、12h时NF-κB p65的表达量较对照组明显增加($P < 0.05$)。②PDTC可以抑制EL的表达。HUVECs经PDTC处理后, 其EL蛋白表达量在作用2、4、8、12、24h与Ang II刺激组比较明显降低($P < 0.05$)。结论 Ang II可能通过NF-κB p65信号传导通路促进内皮细胞中内皮脂肪酶EL的表达。

关键词: 人脐静脉内皮细胞; 内皮脂肪酶; 血管紧张素Ⅱ; 核因子亚单位p65

Angiotensin II upregulates the expression of endothelial lipase in human umbilical vein endothelial cells through the NF-κB signal pathway

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Abstract:

Objective To study the signal pathway of endothelial lipase (EL) expression regulated by angiotensin II (Ang II). Methods The human umbilical vein endothelial cells (HUVECs) were cultured in vitro and divided into 3 groups. ①The Ang II group ($10\mu\text{mol/L}$ Ang II was added); ②The PDTC group (pretreated by 10mmol/L PDTC); ③The control group (no stimulating factors was added). After incubation of 2, 4, 8, 12, 24h respectively, the HUVECs were collected and then EL and NF-κB p65 in HUVECs were detected by Western blot. Results Ang II increased the expression of EL and NF-κB p65 in HUVECs, and two proteins rose in the same trend. EL expressions at 4, 8, 12h were significantly higher than those of the control group ($P < 0.05$) and NF-κB p65 expressions at 2, 4, 8, 12h were significantly higher than those of the control group ($P < 0.05$) after the stimulating of Ang II. PDTC down regulated the expression of EL. After treated with PDTC, the expressions of EL decreased significantly at 2, 4, 8, 12, 24h than those of the Ang II group at the same time ($P < 0.05$). Conclusion Ang II may increase the expression of EL through the signal pathway of NF-κB p65.

Keywords: Human umbilical vein endothelial cells; Endothelial lipase; Angiotension II; NF-κB p65

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