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论文

前胡丙素对Ang II致离体血管平滑肌细胞肥厚及胞内钙、NO含量和信号转导的影响

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摘要:

目的前胡丙素(Pra-C)对血管紧张素II(Ang II)致离体培养大鼠血管平滑肌细胞(SMCs)肥厚模型胞内游离钙浓度、NO含量和信号转导的影响。方法以Ang II刺激SMCs形成肥厚模型,用倒置显微镜测定SMCs面积;用Fura-2/AM测定单细胞内 $[Ca^{2+}]$ i,Griess法测定NO含量;在PMA和ST(PKC激动剂及抑制剂)、PTX(Gi蛋白敏感毒素)作用下观察Pra-C对KCI和NE所致胞内 $[Ca^{2+}]$ i浓度变化的影响。结果Pra-C组SMCs细胞面积较肥厚组减小39.01%,并接近正常细胞水平;NO含量明显增加;胞内 $[Ca^{2+}]$ i对KCI和NE激动的反应明显低于肥厚组。PMA使肥厚SMCs $[Ca^{2+}]$ i升高,而ST及PTX则使之降低,Pra-C均使之恢复正常。结论Pra-C抑制Ang II致体外培养细胞SMCs肥厚,改善肥厚细胞因PKC和Gi蛋白的信号转导改变所致的 $[Ca^{2+}]$ i改变。

关键词: 前胡丙素 血管平滑肌 细胞内游离钙 一氧化氮

EFFECTS OF PRAERUPTORIN C ON CELL HYPERTROPHY, INTRACELLULAR $[Ca^{2+}]i$, NITRIC OXIDE AND SIGNAL TRANSDUCTION IN ISOLATED HYPERTROPHIED RAT SMOOTH MUSCLE CELLS INDUCED BY ANGIOTENSIN II

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Abstract:

AIMTo investigate the effects of praeruptorin C (Pra-C) on smooth muscle cell (SMC) hypertrophy, intracellular calcium ([Ca²⁺] i), nitric oxide (NO) content and influence on cellular signal transduction in isolated cultured rat smooth muscle cell (SMC). METHODSHypertrophied smooth muscle cells (HSMCs) were induced by angiotensin II (Ang II), cell area was measured under inverted microscope. Nitric oxide (NO) concentration was measured using Griess method. [Ca²⁺] i was measured using Fura-2/AM. The responses to [Ca²⁺] i elevation stimulated by KCI (60 mmol·L⁻¹ or norepinephrine (10 µmmol·L⁻¹) were observed by incubation with phorbol 12-myristate 13-acetate (PMA), staurosporine (ST), the agonist and inhibitor of protein kinase C (PKC), and pertussis toxin (PTX), the sensitive toxin of Gi. RESULTSThe cell area of SMCs were decreased by 39.01% (P<0.001) and NO content of SMCs were significantly increased in Pra-C + Ang II group. In presence of 60 mmol·L⁻¹ KCl or 10 µmol·L⁻¹ NE, [Ca²⁺] i of SMCs in Pra-C + Ang II group was significantly decreased than that of Ang II group (P<0.001) and closed to the normal group. Incubation of SMCs with PMA, ST and PTX, $\lceil Ca^{2+} \rceil$ i of SMCs in Ang II group was increased by PMA and decreased by ST and PTX, but that of Pra-C + Ang II group was similar to the normal group. CONCLUSIONThese findings suggest that Pra-C can reduce vascular hypertrophy in isolated rat HSMCs, and this is associated with improvment of SMCs $\lceil Ca^{2+} \rceil$ i level, NO content and cellular signal transdution of PKC and Gi.

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