中国病理生理杂志 2005 21 (4): 685-689 ISSN: 1000-4718 CN: 44-1187/R

论著

PP60c-Src在血管紧张素II诱导的大鼠血管平滑肌细胞信息转导中的作用(英)

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收稿日期 2003-8-13 修回日期 2003-10-20 网络版发布日期 2009-9-15 接受日期 2003-10-20

摘要 目的：本研究通过观察c-Src在AngII对大鼠血管平滑肌细胞(VSMC)丝裂原激活的蛋白激酶(MAPK)的激活和c-Fos蛋白表达中的影响，以进一步阐明AngII促VSMC增殖的细胞内信息转导机制。方法： 原代和传代培养SD大鼠主动脉VSMC，以脂质体包裹反义c-Src寡脱氧核苷酸(oligodeoxynucleotides, ODNs)转染培养的VSMC以抑制c-Src蛋白表达和激酶活性。以未转染的VSMC为对照，观察10-7mol/L AngII刺激对转染的VSMC的MAPK活性和c-Fos蛋白表达的影响。蛋白免疫沉淀和酶自身磷酸化率法测定c-Src激酶活性；髓鞘碱性蛋白(MBP)底物磷酸化率测定MAPK激酶活性；Western blotting免疫印迹法测定c-Src和c-Fos蛋白表达情况。结果： 转染不同浓度反义c-Src ODNs的VSMC，c-Src蛋白含量呈浓度依赖性降低，c-Src激酶活性也显著抑制，以Ang II刺激经转染反义c-Src ODNs的VSMC，c-Src激酶活性增幅仅为对照组的8.7%；MAPK活性仅为对照的1.6%；c-Fos蛋白表达的增幅为对照组的30.0%。结论： AngII可诱导VSMC c-Src激活和细胞内信息转导，且AngII引起的MAPK和c-fos的激活依赖于c-Src的激活，提示c-Src是AngII促血管平滑肌细胞增殖的重要信息分子。

关键词 肌,平滑,血管； 血管紧张素II； c-Src； c-Fos； 有丝分裂素激活蛋白激酶类

分类号 R363

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Effect of PP60c-Src on Ang II-induced signal transduction in rat vascular smooth muscle cells

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Abstract

<FONT face=Verdana>AIM: The aim of the present study was to clarify the mechanism of intracellular signal transduction in Ang II-induced proliferation of vascular smooth muscular cells (VSMC) by observing the effect of c-Src on Ang II-mediated mitogen-activated protein kinase (MAPK) activation and c-Fos protein expression in cultured VSMC of rats. METHODS: Cultured aortic VSMCs from SD rats were transfected with anti-sense c-Src oligodeoxynucleotides (ODNs) wrapt with lipofectin to inhibit c-Src activity and protein production. Untransfected VSMCs were used as control. We observed the role of Ang II stimulation in MAPK activation and c-Fos protein expression. c-Src kinase activity was measured by protein immunoprecipitation and kinase autophosphorylation. The phosphorylation rate of the substrate myelin basic protein (MBP) was employed to assess MAPK activity. Western immunoblot was used to detect protein expression of c-Src and c-Fos. RESULTS: c-Src protein expression in VSMC transfected with different concentrations of anti-sense ODNs significantly decreased in a negative dose-effect manner. c-Src kinase activity was also markedly inhibited . Following the stimulation of Ang II on transfected VSMCs with anti-sense ODNs, the increase rate of c-Src activity was 8.7% of that in control, the activity of MAPK was 1.6% compared with control and c-Fos protein expression was as 30.0% as that of control. CONCLUSION: Ang II induces c-Src activation. MAPK activation and c-Fos protein expression by Ang II is dependent on c-Src activation. These findings indicate that c-Src is an important signal factor in Ang II -induced VSMC proliferation.</FONT>

Key words Muscle smooth vascular Angiotensin II c-Src c-Fos Mitogen-activated protein kinases

DOI: 1000-4718

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