论著

PI3K-ERK信号在胰岛素诱导的血管平滑肌细胞增殖中的作用

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摘要 目的: 探讨胰岛素促血管平滑肌细胞增殖的分子机制。方法: 原代培养的SD大鼠血管平滑肌细胞为研究对象, [3H]-TdR掺入实验观察胰岛素刺激后血管平滑肌细胞的增殖, 免疫印迹分析PI3K-ERK信号在平滑肌细胞增殖中的作用。结果: 胰岛素明显促进血管平滑肌细胞增殖, 该作用呈现剂量依赖性关系, PI3K抑制剂LY294002、MAPK抑制剂PD98059可抑制胰岛素的促增殖作用, [3H]-TdR掺入分别下降48.8%、43.6%,抑制PI3K可下调胰岛素刺激的磷酸化ERK1/2蛋白表达和ERK活性,分别下降112%、127%。结论: PI3K-ERK1/2信号通路参与了胰岛素促平滑肌细胞增殖过程。

关键词 <u>磷脂酰肌醇3激酶;细胞外信号调节激酶类;胰岛素;血管平滑肌细胞;细胞增殖</u> 分类号 0253

Inducing vascular smooth muscle cell proliferation by insulin involves in phosphatidylinositol 3-kinase and ERK1/2

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Abstract

AIM: To investigate the effect of insulin on vascular smooth muscle cells (VSMCs) proliferation and to evaluate the intracellular signaling pathways involved.METHODS: VSMCs separated from Sprague-Dawley rats were used in this study. The proliferation of VSMCs induced by insulin was assayed by [3H]-thymidin incorporation. The protein expression and activity of p-ERK1/2 were determined by immunblot and $[\gamma\text{-}32P]$ ATP incorporation.RESULTS: Insulin induced cell proliferation in a concentration-dependent manner. The proliferative effect of insulin on VSMCs was inhibited partly by LY294002 (48.8%), an inhibitor of PI-3 kinase, and the ERK1/2 inhibitor PD98059 (43.6%), respectively. Moreover, phosphorylation of ERK1/2 and activity of ERK1/2 induced by insulin were also inhibited partly by LY294002.CONCLUSION: PI-3 kinase and ERK1/2 are involved in insulin induced VSMCs proliferation.

Key words Phosphoinositide 3-kinase Extracellular signal-regulated kinases Insulin Vascular smooth muscle cells Cell proliferation

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