论著

SOCS3基因对缺氧大鼠肺动脉平滑肌细胞原癌基因c-myc mRNA表 达及细胞增殖的影响

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目的:探讨SOCS3基因对缺氧大鼠肺动脉平滑肌细胞(PASMCs)原癌基因c-myc mRNA表达及细胞增殖的影 响。 方法: 以脂质体为载体将pEFS0CS3和pSV2neo共转染至体外培养的PASMCs,G418筛选阳性克隆,应用免疫细 胞化学法测定转染前后细胞中SOCS3蛋白表达,缺氧处理转染组和对照组PASMCs,采用半定量RT-PCR检测转染前后 常氧组、缺氧培养2 h、6 h、12 h、16 h、24 h细胞c-myc mRNA表达水平变化; [3H]-TdR掺入法检测转染前后 上述各时点细胞增殖情况。 结果: 免疫细胞化学法证实转染后细胞中有SOCS3稳定表达; 半定量RT-PCR结果显 示,SOCS3基因转染组细胞c-myc mRNA表达水平在缺氧各时点均显著低于同时点对照组细胞(P<0.01);SOCS3基 因转染组细胞在常氧和缺氧各时点[3H]-TdR掺入量显著低于对照组细胞(P<0.01)。 结论: S0CS3蛋白可能通▶文章反馈 过下调c-myc基因表达从而抑制缺氧诱导的PASMCs增殖。

缺氧; 肺动脉; 肌,平滑,血管; 信号转导; 原癌基因 关键词

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Effects of SOCS3 gene on the expression of c-myc mRNA and proliferation of rat pulmonary arterial smooth muscle cells under hypoxia conditions

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Abstract

AIM: To explore the effect of SOCS3 gene on the expression of c-myc mRNA and proliferation of rat pulmonary arterial smooth muscle cells (PASMCs) under hypoxia conditions. METHODS: PASMCs was cotransfected with pEFSOCS3 and pSV2neo by lipofectamine, and positive cell clones were obtained after being screened with G418. Expressions of SOCS3 protein in PASMCs before and after transfection were detected by immunocytochemistry, respectively. Before and after transfection, PASMCs were exposed to normoxic and hypoxia conditions at various time points, respectively, and the expressions of c-myc mRNA were assessed by semi-quantitive RT-PCR. [3H] -TdR incorporation method was used to detect the cell proliferation. RESULTS: The expression of SOCS3 protein was confirmed by immunocytochemistry in PASMCs transfected with SOCS3 gene. c-myc mRNA level in the SOCS3 gene-transfected cells exposed to hypoxia were remarkablely lower than that in the control cells, respectively (P<0.01). Compared with the control groups at the same time points, [3H] -TdR incorporation in SOCS3 gene-transfected cells was significantly low. CONCLUSION: SOCS3 protein may inhibit the proliferation of PASMCs by downregulating the c-myc gene expression under hypoxia conditions.

Key words Anoxia Pulmonary artery Muscle smooth vascular Signal transduction Protooncogenes

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