653~657.人血管能抑素抑制小鼠Lewis肺癌移植瘤生长、转移及血管新生[J].陆卫忠,肖飞,黄桂君,李玉英,李瑾,陈维中,钱桂生.中国肿瘤生物治疗杂志,2011,18(6)

人血管能抑素抑制小鼠Lewis肺癌移植瘤生长、转移及血管新生

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基金项目：

DOI：

摘要：

目的：探讨人血管能抑素（canstatin）对小鼠Lewis肺癌移植瘤生长、转移和血管新生的影响。 方法: 将pCMV-Script/canstatin及空载体pCMV-Script通过电穿孔的方法转染A549细胞，G418筛选获得阳性克隆。RT-PCR检测转染后细胞中canstatin mRNA的表达，Western blotting检测转染后细胞中canstatin蛋白的表达。建立Lewis肺癌小鼠移植瘤模型，观察pCMV-Script/canstatin组A549细胞培养上清对小鼠Lewis肺癌移植瘤的治疗作用，免疫组化检测各治疗组荷瘤小鼠移植瘤的微血管密度。 结果： pCMV-Script/canstatin转染A549细胞在G418筛选后成功形成克隆，转染的A549细胞能有效表达canstatin mRNA和蛋白。pCMV-Script/canstatin治疗组小鼠肿瘤体积明显小于pCMV-Script组和NS组\[（1.47±0.21）cm3 vs (2 43±0.15) cm3、(2.53±0.18) cm3，P<0.01）；pCMV-Script/canstatin组、pCMV-Script组和NS组的肺转移结节数分别为(3.00±1.00)、(7.80±1.48)、(7.60±2.41)个，pCMV-Script/canstatin组肿瘤转移受到显著的抑制（P<0.01）；pCMV-Script/canstatin组小鼠的肿瘤组织微血管数明显少于pCMV-Script组和NS组\[（84.40±8.83） vs (188.68±11.15)、(190.24±12 91)个，P<0.01\]。 结论： pCMV-Script/canstatin能在A549细胞中表达并分泌至细胞外，canstatin可明显抑制Lewis肺癌移植瘤的生长、转移和血管新生。

关键词：人血管能抑素 转染 血管生成 Lewis肺癌

Canstatin inhibits growth, metastasis and angiogenesis of transplanted Lewis lung cancer in mice

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Fund Project:

Abstract:

Objective : To investigate the effect of canstatin on growth, metastasis and angiogenesis of transplanted Lewis lung cancer in mice. Methods: The recombinant pCMV-Script/canstatin vector or the empty vector was transfected into A549 cells by electroporation, and the positive clones were screened with G418. The expressions of canstatin mRNA and protein in transfected A549 cells were examined by RT-PCR and Western blotting, respectively. Furthermore, transplanted Lewis lung cancer mouse model was established, and therapeutic effect of supernatant of pCMV-Script/canstatin transfected A549 cells on transplanted Lewis lung cancer was observed. Microvessel density of transplanted tumors in different therapy groups was observed by immunohistochemistry. Results: Positive clones of A549 cells transfected with pCMV-Script/canstatin were successfully obtained by G418 screening, and could effectively express canstatin mRNA and protein. The tumor size of the pCMV-Script/canstatin group (1.47±0.21 cm3) was significantly smaller than that in the pCMV-Scrip group (2.43±0.15 cm3) and NS group (2.53±0.18 cm3) (P<0.01). The number of pulmonary metastatic nodes was 3.00±1.00, 7.80±1.48 and 7.60±2.41 respectively for pCMV-Script/canstatin, pCMV-Script and NS groups, so pCMV-Script/canstatin significantly inhibited metastasis of tumors (P<0.01). The amount of microvessel count (MVC) in pCMV-Script/canstatin group was markedly decreased compared to that of pCMV-Script and NS groups (P<0.01). Conclusion: The pCMV-Script/canstatin mediates expression of canstatin in A549 cells. Canstatin in A549 supernantant has a strong inhibitory effect on growth, metastasis and angiogenesis of Lewis lung carcinoma.

Keywords:canstatin transfection angiogenesis Lewis lung carcinoma

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