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CA916798基因通过PI3K/AKT通路参与肺癌顺铂耐药

《第三军医大学学报》[ISSN:1000-5404/CN:51-1095/R] 卷: 35 期数: 2013年第07期 页码: 618-621 栏目: 论著 出版日期: 2013-04-15

Title: CA916798 gene is involved in cisplatin resistance in human lung cancer through PI3K/AKT pathway

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关键词: CA916798; 耐药; 顺铂; PI3K/AKT

Keywords: CA916798; drug resistance; cisplatin; PI3K/AKT

分类号: R73-361; R734.2; R979.11

文献标志码: A

摘要: 目的 在前期体外研究的基础上,建立裸鼠移植瘤模型,观察阻断PI3K/AKT通路对肺腺癌细胞A549及多药耐药肺腺癌细胞A549/CDDP内CA916798基因mRNA表达的影响。方法 建立A549、A549/CDDP裸鼠移植瘤模型,以LY294002作用A549组和A549/CDDP组后,比较各组移植瘤生长情况,HE染色观察组织结构变化,实时荧光定量PCR检测肿瘤细胞中CA916798基因mRNA的表达水平。结果 成功建立裸鼠移植瘤模型,以LY294002分别阻断A549、A549/CDDP组裸鼠移植瘤细胞的PI3K/AKT通路后,移植瘤体积明显缩小,CA916798基因的表达明显下调($P<0.05$)。结论 抑制PI3K/AKT通路能够明显抑制肺腺癌细胞A549及A549/CDDP的恶性增殖。

Abstract: Objective To determine the effect of blocking the PI3K/AKT pathway on the expression of CA916798 gene in nude mouse transplanted tumor of human lung adenocarcinoma cell line A549 and the multidrug-resistant cell line A549/CDDP. Methods A total of 20 nude mice were randomly and equally divided into 4 groups, that is, A549, A549/CDDP, A549+LY294002, and A549/CDDP+LY294002 groups. The animal model with A549 cells and A549/CDDP cells transplanted tumor was established in these corresponding nude mice. LY294002 of 25 mg/kg was treated in the mice of the 2 later groups. The size of tumor was measured, and morphology of the mass was observed after HE staining. Real-time fluorescent quantitative PCR was performed to detect CA916798 mRNA expression in the tumor. Results The model of transplanted tumor was

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established in nude mice successfully. Blocking PI3K/AKT pathway with LY294002 resulted in significantly decreased expression of CA916798 at mRNA level ($P<0.05$), and reduced tumor volume. Conclusion Inhibition of PI3K/AKT pathway significantly inhibits the malignant proliferation in lung adenocarcinoma cell A549 and multidrug-resistant cell line A549/CDDP.

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更新日期/Last Update: 2013-04-07