[1]刘雪萍,贺斌峰,陈华萍,等.新型纳米载体Ac-αCD携带的Bcl-xl反义寡核苷酸对肺动脉平滑肌细胞增殖和凋亡的作用[J].第三军医大学学报,2013,35(09):846-849.

Liu Xueping,He Binfeng,Chen Huaping,et al.Effect of a novel nanosystem of Ac-αCD encapsulating Bcl-xl antisense oligonucleotide on proliferation and apoptosis in pulmonary arterial smooth muscle cells[J].J Third Mil Med Univ,2013,35(09):846-849.

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《第三军医大学学报》[ISSN:1000-5404/CN:51-1095/R] 卷: 35 期数: 2013年第09期 页码: 846-849 栏目: 论著 出版日期: 2013-05-15

Title: Effect of a novel nanosystem of Ac-αCD encapsulating Bcl-xl antisense oligonucleotide on proliferation and apoptosis in pulmonary arterial smooth muscle cells

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关键词: 纳米载体; Bcl-xl; 反义寡核苷酸; 肺动脉平滑肌细胞

Keywords: nanosystems; Bcl-xl; antisense oligonucleotide; pulmonary arterial smooth muscle cells

分类号: R322.121; R329.28; R394.2

文献标志码: A

摘要: 目的　研究纳米载体Ac-αCD携带的Bcl-xl反义寡核苷酸(antisense oligonucleotide, ASON)对大鼠肺动脉平滑肌细胞（rat pulmonary arterial smooth muscle cells，RPASMCs）增殖和凋亡作用。　方法　设计合成5′端标记Cy3的Bcl-xl ASON，由纳米载体Ac-αCD携带。实验分3组：纳米载体携带的Bcl-xl ASON组（ASON-NPs组）、单纯纳米载体组（NPs组）和空白对照组，分别使用纳米载体Ac-αCD携带

的Bcl-xl ASON、纳米载体Ac-αCD和培养液处理RPASMCs 48 h，激光共聚焦显微镜观察RPASMCs对纳米载体携带的Bcl-xl ASON的摄取情况；RT-PCR、Western blot检测Bcl-xl的mRNA和蛋白表达；MTT检测处理后细胞的增殖抑制率；流式细胞仪检测细胞凋亡率。　　结果　激光共聚焦显微镜下可见ASON-NPs组细胞质内大量呈颗粒状均匀分布的红色荧光物质，空白对照组和NPs组细胞细胞质内未见红色荧光物质；ASON-NPs组处理的RPASMCs的Bcl-xl mRNA和蛋白表达显著低于空白对照组和NPs组($P<0.05$)； ASODN-NPs组、NPs组、空白对照组细胞抑制率分别为：（53.61±3.02）%、（6.30±1.90）%、（1.40±0.62）%，凋亡率分别为：（53.04±2.09）%、（10.98±2.03）%、（2.19±0.11）%、ASON-NPs组和NPs组细胞抑制率、凋亡率均显著高于空白对照组 ($P<0.01$)，ASON-NPs组均显著高于NPs组 ($P<0.01$)。　　结论　　纳米载体Ac-αCD携带的Bcl-xl反义寡核苷酸能被RPASMCs有效摄取，从而抑制其增殖，促进凋亡。

Abstract: Objective　To determine the effect of an Ac-αCD nanosystem encapsulating Bcl-xl antisense oligonucleotide (ASON) on the proliferation and apoptosis in pulmonary arterial smooth muscle cells.　Methods　Bcl-xl ASON that had been hallmarked with the Cy3 in 5′-end was synthesized, and then encapsulated into the nanosystem Ac-αCD. Primarily cultured SD rat pulmonary arterial smooth muscle cells were treated by Ac-αCD-Bcl-xl ASON or Ac-αCD for 48 h, and the cells without nanosystem served as control. Confocal microscopy was employed to observe the taking of the nanosystem by the cells. Expression of Bcl-xl at mRNA and protein levels, grow inhibitory rate and apoptotic rate were detected by RT-PCR and Western blotting, MTT assay and flow cytometry, respectively.　Results　There were a great deal of brilliantly red-fluorescent granules distributed evenly in the cytoplasm in the cells treated by Ac-αCD-Bcl-xl ASON. No such red-fluorescent granule was seen in the other 2 kinds of cells. The expression of Bcl-xl at mRNA and protein levels were significantly lower in the cells treated by Ac-