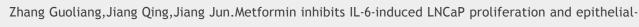
«上一篇/Previous Article|本期目录/Table of Contents|下一篇/Next Article»

mesenchymal transition[J]. J Third Mil Med Univ, 2013, 35(13):1337-1340.

[1]章国亮,姜庆,江军.二甲双胍抑制IL-6诱导的LNCaP增殖及上皮-间质转化[J].第三军医大学学报,2013,35(13):1337-1340.





击复



二甲双胍抑制IL-6诱导的LNCaP增殖及上皮到:

本期目录/Table of Contents

下一篇/Next Article

上一篇/Previous Article

《第三军医大学学报》[ISSN:1000-5404/CN:51-1095/R] 卷: 35 期数: 2013年第13 期 页码: 1337-1340 栏目: 论著 出版日期: 2013-07-15

工具/TOOLS

引用本文的文章/References

下载 PDF/Download PDF(904KB)

立即打印本文/Print Now

查看/发表评论/Comments

导出

Title: Metformin inhibits IL-6-induced LNCaP proliferation and epithelial-mesenchymal transition

章国亮,姜庆,江军

重庆医科大学附属第二医院泌尿外科: 第三军医大学大坪医院野战外科

研究所泌尿外科

Zhang Guoliang; Jiang Qing; Jiang Jun

Department of Urology, Second Affiliated Hospital, Chongqing Medical University, Chongqing, 400010; Department of Urology, Institute of Surgery Research, Daping Hospital, Third Military

Medical University, Chongqing, 400042, China

统计/STATISTICS

摘要浏览/Viewed 210

全文下载/Downloads 96

评论/Comments

RSS XML

关键词: 前列腺癌; 二甲双胍; 上皮-间质转化; LNCaP

Keywords: prostate cancer; metformin; epithelial-mesenchymal transition;

LNCaP

分类号: R392.3; R737.25; R977.15

文献标志码: A

作者:

Author(s):

摘要: 目的 初步探讨二甲双胍抑制前列腺癌发展的作用。 方法 使

用慢病毒感染构建IL-6过表达细胞株 LNCaP- IL-6 (IL-6过表达病毒转

染)和LNCaP-ctr(空病毒转染),运用激光共聚焦扫描确定转染效

果,ELISA检测LNCaP、LNCaP-IL-6和LNCaP-ctr细胞IL-6分泌水平,倒置显微镜观察细胞形态。实验分为LNCaP-ctr组、LNCaP-IL-6组、LNCaP-

ctr+二甲双胍组和LNCaP-IL-6+二甲双胍组,MTT技术检测相对细胞数

量,流式细胞技术检测二甲双胍对细胞周期的影响。实验分为LNCaP-

IL-6和LNCaP-IL-6+二甲双胍组,运用细胞迁移实验检测两组细胞迁移速

度,Western blot技术检测两组细胞TWIST和E-Cadherin表达水

平。 结果 激光共聚焦扫描和ELISA检测证明IL-6过表达细胞株 LNCaP-IL-6构建成功, LNCaP-IL-6细胞IL-6分泌水平约为LNCaP-ctr的5

000倍。MTT实验显示第5天LNCaP-IL-6相对细胞数量约为LNCaP-ctr的

1.4倍,处理组的LNCaP-IL-6相对细胞数量约为LNCaP-ctr的1.6倍;流式细胞仪检测显示二甲双胍阻滞LNCaP的细胞周期为S期。细胞形态学观察和Western blot检测证实IL-6诱导LNCaP发生上皮-间质转化,细胞迁移实验显示二甲双胍可抑制LNCaP-IL-6细胞的迁移,Western blot进一步证实二甲双胍降低LNCaP-IL-6 TWIST表达及升高E-Cadherin 表达。 结论 二甲双胍能够抑制IL-6诱导的LNCaP细胞的上皮-间质转化。

Abstract:

Objective To investigate the inhibitory effect of metformin on prostate cancer development. Methods Lentivirus transfection was used to construct a LNCaP cell line with stable over-expression of interleukin 6 (IL-6)(LNCaP-IL-6), and LNCaP cells transfected with normal lentivirus were used as control (LNCaPctr). Laser scanning confocal microscopy and ELISA were used to confirm IL-6 over-expression in the LNCaP cell line. The morphology of the cells was observed under an inverted microscope. The following experiments were carried out in the four groups: LNCaP-ctr group, LNCaP-ctr with metformin group, LNCaP-IL-6 group and LNCaP-IL-6 with metformin group. MTT assay was used to detect cell relative number, flow cytometry was used to analyze cell cycle, and wound healing assay was used to detect the invasive ability of LNCaP-IL-6. Western blotting was used to detect E-Cadherin and TWIST expression levels in LNCaP-ctr cells and LNCaP-IL-6 cells. Results Laser scanning confocal microscopy and ELISA confirmed that the LNCaP-IL-6 cells were successfully constructed, and the secretion level of IL-6 in LNCaP-IL-6 cells was 5 000 times more than that in LNCaP-ctr cells. MTT assay indicated that the relative number of LNCaP-IL-6 cells was 1.4 times as much as that of LNCaP-ctr cells, and was 1.6 times as much as that in the LNCaP-IL-6 with metformin group after cultured for 5 d. Flow cytometry results indicated that LNCaP cell cycle was arrested at S phase. Morphology observation and Western blot analysis showed epithelia-mesenchymal transition in LNCaP-IL-6 cells. Wound healing assay proved that metformin inhibited LNCaP-IL-6 migration. Western blot analysis showed metformin could decrease TWIST expression and increase E-Cadherin expression. Conclusion Metformin reverses IL-6induced EMT in LNCaP cells.