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中国肿瘤临床 2012, Vol. 39 Issue (21): 1602-1607 DOI: doi:10.3969/j.issn.1000-8179.2012.21.010

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前列腺癌PC-3 多西紫杉醇耐药细胞株的蛋白组学分析*

马伟明, 萧 畔, 俎树祿, 马天加, 周春文, 张怀强

山东大学第二医院泌尿外科 (济南市250033)

Evaluation of Docetaxel-sensitive and Docetaxel-resistant Proteome in PC- 3 Cells

Weiming MA, Pan XIAO, Shulu ZU, Tianjia MA, Chunwen ZHOU, Huaiqiang ZHANG

Department of Urinary Surgery, The Second Hospital of Shandong University, Ji'nan 250033, China

摘要

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摘要 目的: 比较前列腺癌PC-3 细胞株对多西紫杉醇 (docetaxel) 耐药前后的蛋白质差异性表达, 了解前列腺癌PC-3 细胞株耐药性产生机制。**方法:** 利用逐渐加量的方式培养前列腺癌PC-3 多西紫杉醇耐药细胞株, 利用双向荧光差异凝胶电泳 (DIGE) 定量筛选PC-3 细胞敏感株与耐药株的差异蛋白, 并用基质辅助激光解吸电离飞行时间质谱技术 (MALDI-TOF/TOF-MS) 对差异位点蛋白进行成分鉴定。**结果:** 利用DIGE结合MALDI-TOF/TOF-MS质谱技术分析, PC-3 细胞耐药株较敏感株成功分离出49种差异表达蛋白质, 29种表达上调, 20种表达下调。其中ATP synthase、Galectin- 1 等参与肿瘤血管的生成, Calreticulin、CathepsinD Coflin- 1 蛋白参与肿瘤的转移; 78kDaglucoase-regulatedprotein (GRP 78)、Microtubule-associatedprotein- 6 等参与肿瘤的耐药性调节。**结论:** 人前列腺癌PC-3 细胞株多西紫杉醇耐药前后存在蛋白质的差异性表达, 为进一步发现前列腺癌转移及耐药性的分子机制以及晚期激素非依赖性前列腺癌的靶向药物治疗提供实验依据。

关键词: 多西紫杉醇 PC-3 细胞 耐药性 蛋白质组学

Abstract: Objective: This study aims to evaluate docetaxel-sensitive and docetaxel-resistant proteome in PC- 3 prostatic cancer cells, as well as the molecular mechanisms of the docetaxel-resistant PC- 3 cells. Methods: Docetaxel-resistant PC-3 cells were cultured by a dose escalation of docetaxel. The global profiling of the protein expression was investigated through the docetaxel-sensitivity and drug resistance of PC-3 cells using 2-dimensional polyacrylamide gel electrophoresis, matrix-assisted laser desorption, or ionization time-of-flight mass spectrometry. Results: Compared with docetaxel-sensitive PC-3 cells, 49 more differential proteins were found in the docetaxel-resistant PC- 3 cells after performing the DIGE and MALDI-TOF-TOF examinations. The expression of 29 proteins was up-regulated, whereas that of 20 proteins was down-regulated. Among these proteins, ATP synthase and galectin-1 contributed to the formation of tumor vessels, whereas Calreticulin, Cathepsin D, and Coflin contributed to tumor metastasis. Moreover, the 78kDa glu-cose-regulated protein (GRP 78) and microtubule-associated protein- 6 were involved in the drug-resistance regulation of the tumor. Conclusion: A proteomic differential expression was observed between docetaxel-sensitive and docetaxel-resistant PC-3 cells. This finding will be helpful in understanding further the molecular mechanisms of prostate cancer invasion and drug resistance to provide new experimental evidence for the drug therapy of advanced androgen-independent prostate cancer.

Key words: Docataxel PC-3 cells Drug resistance Proteomics

收稿日期: 2012-07-15; 出版日期: 2012-11-15

基金资助:

山东大学第二医院泌尿外科 (济南市250033)

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· Evaluation of Docetaxel-sensitive and Docetaxel-resistant Proteome in PC- 3 Cells[J]. Chinese Journal of Clinical Oncology, 2012, 39(21): 1602-1607.

链接本文:

http://118.145.16.228:8081/Jweb_zgzllc/CN/doi:10.3969/j.issn.1000-8179.2012.21.010 或 http://118.145.16.228:8081/Jweb_zgzllc/CN/Y2012/V39/I21/1602

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地址: 天津市河西区体院北环湖西路肿瘤医院内 300060

电话/传真: (022)23527053 E-mail: cjco@cjco.cn cjcotj@sina.com 津ICP备1200315号