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重组人p53腺病毒联合奥沙利铂对胃癌细胞SGC-7901的生长抑制作用

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Effects of Recombinant Adenovirus-p53 Combined with Oxaliplatin on Growth Inhibition of Human Gastric Cancer Cell Line SGC-7901

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摘要 目的观察外源p53基因在胃癌细胞SGC-7901内的表达及其对胃癌SGC-7901细胞生长的影响、对胃癌细胞化疗敏感度的作用及机制。方法用重组人p53腺病毒注射液（rAd-p53）及奥沙利铂（OXA）单独及联合作用于胃癌细胞株SGC-7901不同时间后，CCT-8法检测其对体外培养SGC-7901细胞的抑制率，免疫组织化学SP法检测p53蛋白的表达情况，流式细胞仪（FCM）分析其细胞凋亡蛋白Caspase-3的表达情况。 结果rAd-p53及OXA单独作用SGC-7901细胞时，随药物浓度及作用时间的增加，细胞的生长抑制率逐渐增高；两者联合作用48h，其在较低浓度时即可显著抑制细胞生长（$P<0$ 05）；OXA（6.25 μg/ml）与rAd-p53(5×107、5×108、5×109vp/ml)联合作用48h后，胃癌细胞caspase-3蛋白的含量较对照组升高，但p53蛋白无明显升高。结论OXA和 rAd-p53单药可抑制胃癌细胞的生长，两者联合对胃癌细胞的抑制作用明显增强； rAd-p53有增强OXA化疗敏感度的作用，其机制与通过线粒体途径激活下游的 caspase-3诱导细胞凋亡有关。

关键词： rAd-p53 OXA 胃癌细胞 生长抑制 Caspase-3

Abstract： ObjectiveTo observe the expression of exogenous p53 gene in gastric cancer cells and its effects on the growth of tumor cells; and to investigate the effects of adenovirusmediated p53 gene on chemosensitivity of human gastric cell and the value of gene therapy combined with chemotherapy. MethodsToinvestigate the effects of recombinant adenovirus-p53 (rAd-p53) and oxaliplatin(OXA) alone and combined on the gastric cancer cell line SGC-7901 for different times,CCK-8 assay was used to examine the suppressive rate of cell growth dealt; p53 protein expression was detected by immunohistochemistry assay; and protein caspase-3 expression in cell was induced by different drugs alone and in combination by flow cytometry. ResultWhen rAd-p53 and OXA was alone used to treat the gastric cancer cell line SGC-7901, with an increase in drug concentration and treated time, the cell growth inhibition rate gradually increased; when rAd-p53 and OXA was combined to treat the gastric

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cancer cell line SGC-7901 for 48 h, the cell growth inhibition rate gradually increased at the lowest concentration. When rAd-p53 (5×107, 5×108, 5×109vp/ml) and OXA (6.25μg/ml) was combined to treat the gastric cancer cell line SGC-7901 for 48h, compared with the control group, the content of caspase-3 protein in gastric cancer cell gradually increased, but p53 protein expression didn't increase. ConclusionThe proliferation of SGC-7901 could be inhibited by rAd-p53 OXA alone, but when rAd-p53 was combined with OXA, the proliferation of cells was higher than that used alone. The combination of rAd-p53 and OXA induced cell apoptosis through mitochondrial pathway to activate downstream of caspase-3.

Key words: Recombinant adenovirus-p53 Oxaliplatin Gastric cancer cell Growth inhibition caspase-3

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