

论著

p53沉默对人鼻咽癌细胞株CNE2放射生物学特性的影响

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摘要

目的: 研究p53沉默对鼻咽癌(NPC)细胞株CNE2放射敏感性的影响, 探讨NPC中过表达的p53蛋白是否参与了放射诱导的细胞损伤和凋亡过程。方法: 以稳定干扰p53基因表达的鼻咽癌细胞株CNE2sip53和对照细胞株CNE2/pSUPER为对象, 采用集落存活分析法分析在不同放射剂量照射后的细胞存活分数(SF)并计算放射生物学参数D₀, α , β 和放射敏感比(SER); 采用MTT法检测放射线对细胞生长的影响; 采用流式细胞术检测放射线对细胞周期及凋亡的影响。结果: CNE2sip53细胞照射后的SF值高于CNE2/pSUPER细胞; 与CNE2/pSUPER细胞相比, CNE2sip53细胞D₀值和SF₂值均增大, 而 α 值, β 值和SER均减小; 放射线诱导CNE2sip53细胞凋亡及对细胞增殖的抑制作用弱于其对CNE2/pSUPER细胞的作用; 放射线诱导CNE2/pSUPER细胞阻滞于G₁期, 而诱导CNE2sip53细胞阻滞于G₂期。结论: 稳定沉默鼻咽癌细胞中p53基因表达后, 细胞对放射的敏感性降低, 提示NPC细胞中过表达的p53蛋白参与了放疗诱导的NPC细胞损伤和凋亡过程。

关键词 [鼻咽癌](#); [放射生物学](#); [p53](#)

分类号

Effect of p53 knockdown on the radiobiological characteristics of nasopharyngeal carcinoma cell line CNE2

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Abstract

Objective To investigate the effect of p53 knockdown on the radiobiological characteristics of a nasopharyngeal carcinoma (NPC) cell line CNE2 and to explore whether the overexpressed p53 protein in NPC is involved in the process of cell damage and apoptosis induced by radiation. Methods NPC cell line CNE2sip53 stably transfected by p53 siRNA vector and control cell line CNE2/pSUPER transfected with empty vector were used in this study. A clonogenic survival assay was performed to obtain a irradiation dose-survival curve and a survival fraction (SF), and then calculated radiobiological parameters, such as SF₂, D₀, α , β and sensitization enhancement ratios (SER). MTT assay and flow cytometry were performed to determine the effects of irradiation on the growth, cell cycle distribution and apoptosis, respectively. Results SF of CNE2sip53 was significantly higher than that of CNE2/pSUPER. When compared with CNE2/pSUPER, SF₂ and D₀ of CNE2sip53 were significantly increased, whereas α , β and SER were significantly decreased. The number of apoptotic cells induced by radiation in CNE2sip53 was significantly decreased compared with CNE2/pSUPER, and the inhibition of cell growth induced by radiation in CNE2sip53 was also significantly lower than that in CNE2/pSUPER. Irradiation could arrested CNE2/pSUPER cells at G₁ phases while arrested CNE2sip53 cells at G₂ phases. Conclusion Stably knocking down the expression of p53 could decrease the radiation sensitivity of CNE2. It suggests that the overexpressed p53 protein in NPC might be involved in the process of cell damage and apoptosis induced by radiation.

Key words [nasopharyngeal carcinoma](#); [radiobiology](#); [p53](#)

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