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CHFR基因CpG岛甲基化对食管癌Eca109细胞增殖和凋亡的影响 点此下载全文

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摘要：

目的：研究CHFR基因启动子甲基化状态与食管癌Eca109细胞增殖和凋亡的关系。方法：分别用不同浓度（2、5、10 μmol/L）5-氮杂-2′-脱氧胞苷（5-Aza-2′-deoxycytidine，5-Aza-CdR）处理Eca109细胞，未经药物处理的细胞为对照组。甲基化特异性PCR（methylation-specific PCR，MSP）检测CHRF基因CpG岛的甲基化状态，RT-PCR检测CHFR mRNA的表达情况，MTT法及流式细胞术检测不同浓度5-Aza-CdR 处理对Eca109细胞增殖及凋亡的影响。结果：对照组Eca109细胞CHFR CpG岛处于高甲基化状态，5-Aza-CdR处理后CHFR CpG岛可发生不同程度的剂量依赖性的去甲基化。对照组Eca109细胞无CHFR mRNA表达，5-Aza-CdR处理组（2、5、10 μmol/L）CHFR mRNA相对表达量显著增加（0.174±0.010、0.221±0.013、0 356±0.014）。不同浓度5-Aza-CdR 处理后，Eca109细胞增殖受到抑制\[作用72 h时的抑制率分别为（30.87±0.74）%、（44.60±0 79）%、（56.67±0.35）%\]，细胞凋亡显著增加\[(7.46±1.46)%、(16.27±1.61)%、(25.29±2.25)% vs (1.83±0.41)%，P<0.01\]。结论：食管癌Eca109细胞经5-Aza-CdR 处理后，CHFR基因CpG岛发生部分去甲基化，出现CHFR mRNA表达，并抑制Eca109细胞增殖和促进细胞凋亡。

关键词：CHFR基因 甲基化 食管癌 Eca109细胞 增殖 凋亡

Effect of CHFR CpG island methylation status on proliferation and apoptosis of esophageal cancer Eca109 cells Download Fulltext

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Abstract:

Objective:To study the relationship of CHFR CpG island methylation status on proliferation and apoptosis of esophageal cancer Eca109 cells. Methods: Eca109 cells were treated with different concentrations (2, 5, and 10 μmol/L) of 5-Aza-2′-deoxycytidine (5-Aza-CdR), and untreated Eca109 cells were used as control. Methylation-specific PCR (MSP) analysis was used to evaluate the CpG island methylation status of CHFR gene, and RT-PCR was used to detect the CHFR mRNA expression in Eca109 cells. MTT and flow cytometery were used to determine the proliferation and apoptosis of Eca109 cells treated with different concentrations of 5-Aza-CdR. Results: CHFR CpG island was hypermethylated in the untreated Eca109 cells, and methylated CHFR CpG was demethylated to different degrees in 5-Aza-CdR treatment groups. No expression of CHFR mRNA was found in untreated Eca109 cells, but the relative expression of CHFR mRNA was remarkably increased in 5-Aza-CdR (2, 5, and 10 μmol/L) treatment groups (0.174±0.010, 0.221±0.013, and 0.356±0 014). Different concentrations of 5-Aza-CdR inhibited the proliferation (\[30.87±0.74\]%,\[44.60±0.79\]%, and \[56.67±0 35\]%), and promoted apoptosis of Eca109 cells (\[7.46±1.46\]%, \[16.27±1.61\]%, \[25.29±2 25\]% vs \[1.83±0.41\]%, P<0 01). Conclusion: 5-Aza-CdR can partly demethylate CHFR CpG island in esophageal cancer Eca109 cells, inducing CHFR mRNA expression, inhibiting proliferation and promoting apoptosis of Eca109 cells.

Keywords:CHFR methylation esophageal cancer Eca109 cell proliferation apoptosis

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