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MTF-1对食管鳞癌EC109细胞株生长和迁移的影响

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Title: Effects of MTF-1 on cell proliferation and migration in human esophageal carcinoma EC109 cells

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关键词: [金属反应转录因子1](#); [食管鳞癌](#); [细胞增殖](#); [细胞迁移](#)

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摘要: 目的 研究外源性金属反应转录因子-1 (metal-responsive transcription factor-1, MTF-1) 对食管鳞癌EC109细胞的增殖、凋亡、迁移及细胞周期的影响,并探讨其作用机制。 方法 构建MTF-1真核表达载体pcDNA3.1-MTF-1,转染EC109细胞,采用RT-PCR和Western blot分别检测MTF-1 mRNA和蛋白水平的表达。采用CCK-8法检测细胞生长,流式细胞仪检测细胞凋亡和周期,Transwell实验检测细胞迁移能力。 结果 与转染pcDNA3.1的对照组相比,转染pcDNA3.1-MTF-1显著上调了EC109细胞中MTF-1表达量。过表达MTF-1 48 h后,EC109细胞生长显著加快 ($P<0.01$); 细胞凋亡率降低 ($P<0.05$); 细胞周期比例发生显著改变, G₁期细胞比例降低 ($P<0.01$), S期细胞比例升高 ($P<0.01$); 细胞迁移能力无显著改变。 结论 MTF-1通过抑制细胞凋亡、促进细胞周期G₁到S期的转换进而促进食管鳞癌EC109细胞的生长,但对EC109的迁移能力没有显著影响。

Abstract: Objective To determine the effects of exogenous metal-responsive transcription factor-1 (MTF-1) on the cell proliferation, apoptosis, cell cycle and migration in human esophageal carcinoma EC109 cells. Methods The CDS fragment of MTF-1 was cloned into the pCDNA3.1 vector to construct recombinant vector pcDNA3.1-MTF-1. And then pcDNA3.1-MTF-1 and pcDNA3.1 (control) were transferred into EC109 cells separately. The mRNA and protein expression levels of MTF-1 were detected by RT-PCR and Western blotting. CCK8

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assay was used to detect the effect of MTF-1 overexpression on EC109 cell proliferation and the effects on apoptosis and cell cycle were analyzed by flow cytometry. Transwell chamber assay was used to examine the migration potential of the EC109 cells. Results Compared with pcDNA3.1 group, pcDNA3.1-MTF-1 group expressed high-level exogenous MTF-1 and the proliferation of EC109 cells were significantly increased in 48 to 96 h ($P<0.01$). After pcDNA3.1-MTF-1 transfection for 48 h, apoptosis of EC109 cells was significantly inhibited ($P<0.05$). Meanwhile, the percentage of EC109 cells in G_1 phase was decreased ($P<0.01$) and that in S phase was increased significantly ($P<0.01$) as compared to the control group. The cell migration abilities didn't show significant difference between the 2 groups. Conclusion Exogenous MTF-1 stimulates the proliferation of EC109 cells by inhibiting apoptosis and promoting G_1/S transition, while does not affect migration of EC109 cells obviously.

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