论著

Manumycin通过诱导细胞凋亡抑制人胰腺导管癌细胞Panc-1活性

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目的:观察manumycin对人胰腺导管癌细胞Panc-1的抑制效应,并探讨其诱导细胞凋亡是否经 p38MAPK介导。 方法: 用MTT法检测manumycin对Panc-1细胞的抑癌作用。用caspase-3活性检测试剂 盒定量检测manumycin诱导细胞凋亡的水平及评估特异性的p38MAPK抑制剂SB203580对它的影响。 结果: 经manumycin(6 µmol/L、18 µmol/L、54 µmol/L)处理Panc-1细胞24 h,对Panc-1细胞生长具有明显的抑 ▶ 复制索引 制作用, 其抑制率分别为8.9%、21.9%和67.0%, 其中后二者的细胞活性与对照组相比有显著差异 (P<0.01), 呈量效关系。用药24 h的IC50为34.7 μmol/L。同时,此药物可明显增加caspase-3的活性,且 这一效应可部分地被p38抑制剂SB203580阻断。 结论: Manumycin可通过诱导Panc-1细胞凋亡而产生抑 癌作用,p38MAPK是manumycin诱导细胞凋亡的通路之一。

Manumycin; 胰腺导管癌; 细胞凋亡; p38MAP激酶

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Manumycin inhibits activity of pancreatic duct cancer cell line Panc-1 via inducing apoptosis

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Abstract

AIM: In this study, we investigated the anticancer effect and mechanisms of manumycin on pancreatic cancer cell line-Panc-1 and the role of p38MAPK pathway in apoptosis. METHODS: The test of anticancer effect was performed by MTT assay. Apoptosis was induced in the cells by manumycin and then treated with SB203580, a specific p38MAPK inhibitor. A quantitative caspase-3 activity assay kit was used in this experiment. RESULTS: Manumycin (6 µmol/L, 18 µmol/L, 54 µmol/L) significantly inhibited cell growth of pancreatic cancer cell line Panc-1. The inhibition rates 24 h after treatment with 6 μmol/L, 18 μmol/L and 54 µmol/L manumycin were 8.9%, 21.9% and 67.0%, respectively. Compared with the control group, the survival levels of the last two groups were of significant statistical difference (P<0.01). The anticancer effects also showed dosage-effect relationship, the value of IC50 24 h after treatment was 34.7 µmol/L. In addition, this reagent simultaneously activated caspase-3 protein, which was partly blocked by p38MAPK specific inhibitor, SB203580. CONCLUSION: Manumycin exerted anticancer effect on Panc-1 cell line via inducing cell apoptosis, which was partly regulated by p38MAPK.

Key words Manumycin Pancreatic neoplasms Apoptosis p38 MAP kinase

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