#### 论著

## 维生素E琥珀酸酯经由活性氧诱导胃癌细胞SGC-7901凋亡

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收稿日期 2008-12-2 修回日期 2009-2-19 网络版发布日期:

摘要 背景与目的: 研究维生素E琥珀酸酯(VES)对人胃腺癌SGC-7901细胞生长的抑制作用,探索VES诱导SGC-7901细胞凋亡情况及其可能机制。 材料与方法: 胃癌SGC-7901细胞分为VES不同浓度处理组和1 mmol/L 甘露醇+20 μg/ml VES 处理组 (细胞先用1 mmol/L的甘露醇处理2 h后,加入20 μg/ml的VES处理), SGC-7901细胞经受试物处理24 h后,采用MTT、单细胞凝胶电泳方法和流式细胞技术,检测VES诱导SGC-7901细胞的氧化损伤及凋亡的情况,用相应的生化检测试剂盒检测细胞内活性氧及谷胱甘肽的含量变化。 结果: 0、1.25、2.5、5、10、20 μg/ml的VES对胃癌细胞SGC-7901细胞的生长均有不同程度的抑制作用,其中20 μg/ml VES组抑制作用最大,其生长抑制率达76.91%。研究结果还显示随VES浓度的增加,细胞凋亡率呈现增加的趋势。 彗星实验结果显示,各实验组随着VES浓度的增加,彗星尾长与彗星细胞率呈现增加的趋势。 VES 10、20 μg/ml组与阴性对照组相比, SGC-7901细胞内谷胱甘肽含量显著下降(P<0.05),同时活性氧含量显著增加(P<0.05)。1 mmol/L 甘露醇+20 μg/ml VES 处理组与VES 20 μg/ml处理组比较: SGC-7901细胞DNA损伤程度较轻,细胞凋亡率明显低于VES 20 μg/ml处理组,其细胞内活性氧的生成减少,细胞内谷胱甘肽的消耗减少(P<0.05)。 结论: VES可显著抑制胃癌细胞SGC-7901的生长,且随VES剂量的增加,其抑制作用越强。细胞内活性氧含量增加、谷胱甘肽耗竭所导致的氧化损伤作用可能是VES诱导细胞凋亡的机制之一。

关键词 维生素E琥珀酸酯 SGC-7901细胞 活性氧 谷胱甘肽

# Vitamin E Succinate-induced Apoptosis of SGC-7901 Cells via Reactive Oxygen Species

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**Abstract** BACKGROUND AND AIM: To study the growth inhibition effect of Vitamin E succinate(VES) on SGC-7901 cells, and explore SGC-7901 VES-induced apoptosis and its possible mechanism. MATERIALS AND METHODS: After SGC-7901 cells were treated with 0,5,10,20 µg/ml VES and 1 mmol/L of mannitol +20 µg/ml VES(cells were treated with 1 mmol/L of mannitol for 2 hours, then with 20 µg/ml VES for 24 hours) for 24 h MTT, single-cell gel electrophoresis and flow cytometry were used to detect the oxidative damage and apoptosis induced. The corresponding detection kits were used to measure the reactive oxygen species and glutathione content. RESULTS: 0, 1.25, 2.5, 5, 10, 20 µg/ml of VES inhibited cell growth to different degrees. 20 µg/ml VES inhibited cell growth to the greatest degree of 76.91%. With increasing concentration of VES, apoptosis showed an upward trend. In single-cell gel electrophoresis, with increasing VES concentration, the comet tail length and the comet rate showed a rising trend. Compared with the negative control group, glutathione content of cells in 10 and 20 μg/ml VES groups were significantly decreased, while the active oxygen content were significantly increased. 1 mmol/L of mannitol could significantly reduce DNA damage and apoptosis caused by VES, while lowering the cell oxygen and glutathione contents significantly. CONCLUSION: VES could significantly inhibit SGC-7901 cell growth in a dose-depedent manner. Oxidative damage caused by increased oxygen content and glutathione depletion may be one of the mechanisms of apoptosis induced by VES.

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**Keywords** Vitamin E succinate SGC-7901 cells reactive oxygen species glutathione

DOI

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