

论著

冬凌草甲素诱导人雄激素非依赖性前列腺癌PC-3细胞凋亡及其机制

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摘要:

目的:探讨冬凌草甲素抑制人雄激素非依赖性前列腺癌细胞株PC-3细胞的增殖、诱导其凋亡的作用。方法:用不同浓度的冬凌草甲素干预PC-3细胞,通过MTT实验和细胞的药物浓度-时间生长曲线分析观察其对PC-3细胞活力的影响;用流式细胞仪分析PC-3早期凋亡细胞的百分率;Western印迹检测Bax Bcl-2和caspase-3蛋白表达的变化。结果:冬凌草甲素呈时间和浓度依赖性地抑制PC-3细胞增殖,药物抑制PC-3细胞活力的IC₅₀约为10.29 μmol/L;凋亡细胞形态学鉴定、流式细胞仪检测结果均表明冬凌草甲素能以浓度依赖性方式诱导PC-3细胞凋亡($P<0.05$);冬凌草甲素以浓度依赖性方式抑制PC-3细胞的Bcl-2蛋白表达,而上调Bax蛋白表达并活化caspase-3。结论:冬凌草甲素可能通过线粒体途径诱导PC-3细胞凋亡。

关键词: 冬凌草甲素 PC-3细胞 增殖 抑制 凋亡

Oridonin induced the apoptosis of PC-3 cells and its mechanism

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Abstract:

Objective To observe the proliferation inhibition and apoptosis promotion effect of oridonin on PC-3 cells. Methods PC-3 cells were treated with different concentrations of oridonin. MTT assay and drug concentration-time survival curve were used to test the effect of oridonin on the PC-3 cells. The percentage of earlier apoptosis cells was analyzed by flow cytometry. The protein expression of caspase-3, Bcl-2, and Bax in the PC-3 cells was detected by Western blot. Results Oridonin effectively inhibited the proliferation of PC-3 cells in both concentration- and time-dependent manner, and the IC₅₀ of PC-3 cells was 10.29 μmol/L. Hochest33258 staining and flow cytometry detected that oridonin induced the apoptosis of PC-3 cells in a concentration-dependent manner ($P<0.05$). Oridonin down-regulated Bcl-2, up-regulated Bax protein, and activated caspase-3 in a concentration-dependent manner in the PC-3 cells. Conclusion The apoptosis of PC-3 cells induced by oridonin might be associated with the mitochondrial pathway.

Keywords: oridonin PC-3 cell proliferation inhibition apoptosis

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