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论文

二乙酰二脱水卫矛醇诱导白血病HL-60细胞凋亡及其机理

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摘要:

目的研究二乙酰二脱水卫矛醇(DADAG)诱导人白血病HL-60细胞凋亡及其机理。方法MTT法观察DADAG的体外抗增殖作用;透射电镜、DNA梯形条带和流式细胞仪检测HL-60细胞凋亡;Western blotting法和caspase-3检测试剂盒分析DADAG诱导HL-60细胞凋亡与Bcl-2家族成员和caspase-3的关系。结果DADAG明显抑制HL-60细胞增殖和诱导细胞发生凋亡。8 μg·mL^{-1} DADAG处理HL-60细胞不同时间后,Bcl-X_L 蛋白水平呈时间依赖性地下降,而Bad蛋白水平上调。DADAG处理HL-60细胞24 h后,caspase-3酶活性达峰值。Caspase-3抑制剂z-DEVD.fmk可部分逆转DADAG诱导HL-60细胞凋亡的作用,而caspases广谱抑制剂z-VAD.fmk可完全逆转此作用。结论DADAG诱导HL-60细胞凋亡依赖caspase-3途径的激活,而caspase-3的激活可能与Bcl-2家族成员密切相关。

关键词: 二乙酰二脱水卫矛醇 细胞凋亡 HL-60细胞

APOPTOSIS INDUCED BY DIACETYLDIANHYDROGALACTITOL AND ITS MECHANISM IN HL-60 LEUKEMIA CELLS

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Abstract:

AIMTo investigate the apoptosis induced by diacetyldianhydrogalactitol (DADAG) and its mechanism in human HL-60 leukemia cells. METHODSInhibition of proliferation was measured by MTT assay. DADAG-induced apoptosis in HL-60 cells was observed by electron microscopy, flow cytometry and DNA fragmentation assay. The levels of Bcl-2 family proteins were detected by Western blotting. Caspase-3 activity was determined by ApoAlert CPP32 colorimetric assay kit. RESULTS DADAG exhibited potent antiproliferative activity and induced apoptosis in HL-60 cellls. After treatment with DADAG 8 μg·mL^{-1} for various times, the Bcl-X_L protein level decreased in a time-dependent manner, while the Bad protein level was up-regulated. The caspase-3 activity increased markedly after treatment with DADAG for 24 h. The apoptotic signals were suppressed by z-VAD.fmk (a general inhibitor of caspases), whereas z-DEVD.fmk, a selective inhibitor of caspase-3, only induced partial reversion of the apoptotic effects. CONCLUSIONDADAG-induced apoptosis in HL-60 cells required caspase-3 activation and caspase-3 activation was related with Bcl-2 family members.

Keywords: apoptosis HL-60 cells diacetyldianhydrogalactitol

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