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基础医学

藤黄酸下调Bcr-Abl蛋白对慢粒细胞株K562增殖和凋亡的影响

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摘要：

目的 研究藤黄酸对人慢性粒细胞性白血病细胞株K562抑制增殖及诱导凋亡的作用，并探讨其可能作用机制。方法 用藤黄酸处理K562细胞，采用MTS法和台盼兰计数法测定细胞活力及增殖；碘化丙啶(PI)单染荧光显微镜观察细胞形态改变；采用AnnexinV-FTIC/PI流式细胞术检测细胞凋亡；利用Western blotting检测凋亡相关蛋白及细胞增殖信号通路的变化情况。 结果 藤黄酸对K562细胞的增殖抑制作用呈时间和浓度依赖性(P<0.05)。藤黄酸处理后的细胞，经PI染色，荧光显微镜下观察可见死亡细胞形态发生改变，细胞核红染。流式细胞术检测显示加药处理组细胞以凋亡方式为主，凋亡率比对照组明显升高(P<0.05)。Western blotting检测发现凋亡相关蛋白激活，Bcr-Abl及下游的信号通路受到不同程度的抑制(P<0.05)。结论 藤黄酸对K562细胞具有凋亡诱导和增殖抑制作用，作用机制与caspase系统激活和Bcr-Abl增殖相关通路受抑有关。

关键词： 藤黄酸；K562细胞；凋亡；Bcr-Abl

Effects of Garcinia acid by down regulation of Bcr-Abl protein on the proliferation and apoptosis of chronic myelogenous leukemia cell line K562

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Abstract:

Objective To study the influence of Gambogic acid (GA) on the proliferation and cell apoptosis of chronic myeloid leukemia cells, and to investigate the possible mechanism. Methods After treatment with various concentrations of GA, cell viability was determined by MTS assay and trypan-blue counting method. The morphologic changes of K562 cells induced by GA were observed under fluorescence microscope with Propidium Iodide (PI) staining. Cell apoptosis was analyzed by Flow Cytometry staining with AnnexinV-FTIC/PI. The changes of apoptosis and proliferation-related proteins were tested by Western blotting. Results GA inhibited the proliferation of K562 cells in a time- and dose- dependent manner(P<0.05). After treatment with various concentrations of GA, morphological changes were observed under fluorescence microscope, and nucleus of death cells were stained red with PI. Flow Cytometry showed that the death type of the treatment group was mainly apoptosis, and the apoptosis rate significantly increased compared with the control group (P<0.05). The caspase protein was activated, and Bcr-Abl protein and downstream signal pathway were inhibited(P<0.05). Conclusion GA could induce apoptosis and inhibit the proliferation of K562 cells. This effect may be mediated by activated caspase protein, reduced expression of Bcr-Abl protein and its downstream signal pathway.

Keywords: Gambogic acid; K562 cells; Apoptosis; Bcr-Abl

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