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PI3K/Akt抑制剂渥曼青霉素对白血病细胞增殖和凋亡的影响 点此下载全文

王晓南 吴 青 张连生 吴一品 舒砚文

武汉科技大学医学院 基础医学实验中心 湖北 武汉 430065;华中科技大学 同济医学院 附属协和医院 血液病研究所 湖北
医学实验中心 湖北 武汉 430065;华中科技大学 同济医学院 附属协和医院 血液病研究所 湖北 武汉 430022;华中科技
究所 湖北 武汉 430022

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摘要：

目的：研究PI3K/Akt抑制剂渥曼青霉素对白血病细胞增殖和凋亡的影响，并探讨其可能的作用机制。方法：以不同浓度的
K562,采用MTT法检测细胞增殖活性，单细胞凝胶电泳技术检测细胞DNA损伤形成的“彗星”样拖尾现象，Annexin V FITC
ting、RT PCR检测渥曼青霉素作用K562细胞后总Akt和磷酸化Akt以及NF κB基因及蛋白表达水平的变化。结果：渥曼青霉素
增殖,其24 h的IC 50 是25 nmol/L。渥曼青霉素诱导K562细胞发生凋亡，其作用呈明显剂量依赖性增强。渥曼青霉素
星”拖尾现象，其尾长与拖尾率显著高于对照组(P <0.01)。渥曼青霉素能同时在蛋白和基因水平，以剂量依赖性方式抑
蛋白没有明显影响。结论：渥曼青霉素以时间和剂量依赖方式明显抑制K562细胞的增殖及诱导其凋亡，其机制可能与其下调
表达有关。

关键词：渥曼青霉素 白血病细胞 转录核因子κB 磷酸化Akt 凋亡

Effect of PI3K/Akt inhibitor wortmannin on proliferation and apoptosis of leukemia K562 cells Do

WANG Xiao-nan WU Qing ZHANG Lian-sheng WU Yi-pin SHU Yan wen

Laboratory of Basic Medicine, Wuhan University of Science and Technology, Wuhan 430065, Hubei, China
Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 4300
Basic Medicine, Wuhan University of Science and Technology, Wuhan 430065, Hubei, China;Institute of H
Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430022, Hub
Affiliated Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wu

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Abstract:

Objective:To study the effect of wortmannin (WM), a PI3K/Akt inhibitor, on the proliferation and apo
possible mechanism. Methods:Human leukemia cell line K562 was treated with different concentrations o
cells was examined by MTT assay. DNA damage in K562 cells was examined by single cell gel electropho
cells was detected by Annexin V FITC/PI double staining. The expressions of total Akt, phosphorate A
and protein were detected by RT PCR and Western blotting, respectively. Results:WM inhibited the prolif
time dependent manner, with the IC 50 value of 24 h being 25 nmol/L. WM also induced apoptosis
manner. DNA damage in K562 cells was demonstrated by appearance of comet tail after treatment with
the tail length being significantly higher than those in the control group (P <0.01). WM dose depende
but not the total Akt, mRNA and protein expressions. Conclusion:WM can inhibit proliferation and induce
and time dependent manner, probably through down regulation of phosphorate PI3K/Akt signal pathwa

Keywords:wortmannin NF κB phosphorate Akt apoptosis K562 cell