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长春新碱诱导肝癌细胞自噬性凋亡过程中泛素与bcl-2 的变化

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Changes of Ubiquitin and bcl-2 During Autophagic Apoptosis of HepG2 Cells Induced by Vincristine

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摘要

目的 探讨长春新碱（VCR）诱导HepG2肝癌细胞自噬性凋亡过程中泛素表达的变化及阻断泛素一蛋白酶体通路对此凋亡和bcl-2表达的影响。方法 应用VCR诱导HepG2细胞自噬性凋亡，采用流式细胞仪检测凋亡及泛素的表达；以RT-PCR检测bcl-2的表达。结果 VCR处理后发生自噬性凋亡的HepG2细胞中泛素含量增加（P〈0．01）。加用蛋白酶体特异抑制剂乳胞素后的乳胞素＋VCR组凋亡率明显比单用VCR组高（P〈0．01），而bcl-2表达则比单用VCR组更低。结论 泛素一蛋白酶体通路参与了VCR诱导的HepG2细胞自噬性凋亡及对bcl-2蛋白的调控。对蛋白酶体功能的抑制可以促进VCR诱导的HepG2细胞凋亡。

关键词： 长春新碱 泛素-蛋白酶体通路 肝癌细胞 凋亡 bcl-2

Abstract： Objective To study the changes of ubiquitin during vincristine (VCR) mediated autophagic apoptosis of Hep G2 cells ,and determine the influences of inhibiting ubiquitin-proteasome pathway on this apoptosis and bcl-2. Methods To induce autophagic apoptosis of Hep G2 cells t reated with VCR. The apoptosis and the expression of ubiquitin were detected with flowcytometry(FCM) and the expression of bcl-2 was examined by RT-PCR technique. Results VCR could significantly increase ubiquitin level in Hep G2 cells that underwent autophagic apoptosis (P < 0. 01) . There are higher apoptosis rate (P < 0. 01) and lower expression of bcl-2 in the cells by using VCR combined with lactacystin (a proteasome inhibitor) than that in the cells treated with VCR alone. Conclusion Ubiquitin-proteasome pathway is involved in the VCR-induced autophagic apoptosis of hep G2 cells and in regulating the levels of bcl-2 , which might have a role in mediating autophagic apoptosis in Hep G2 cells. The inhibition of Ubiquitin-proteasome pathway can enhance VCR-induced apoptosis in Hep G2 cells.

Key words： Vincristine Ubiquitin-proteasome pathway Hepatoma cell Apoptosis bcl-2

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