

论著

## 凋亡素诱导HepG2细胞凋亡过程中Mcl-1 mRNA和蛋白水平的变化及意义

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**摘要** 目的: 探讨凋亡素诱导肝癌细胞HepG2凋亡过程中Mcl-1 mRNA和蛋白水平的变化及其意义。方法: 经脂质体介导将pCDNA3.0-VP3转入HepG2细胞内,48 h后采用Western blotting检测凋亡素、Mcl-1和细胞色素C,实时定量RT-PCR检测细胞内的Mcl-1 mRNA。结果: VP3基因成功地转入HepG2细胞内并稳定表达凋亡素。与空白对照相比,表达凋亡素的细胞出现Mcl-1 mRNA含量减少( $0.09\% \pm 0.00\%$  vs  $0.41\% \pm 0.14\%$ ,  $P < 0.05$ ),细胞内Mcl-1水平下降( $0.43\% \pm 0.01\%$  vs  $0.90\% \pm 0.04\%$ ,  $P < 0.01$ ),线粒体释放细胞色素C增加( $0.98\% \pm 0.02\%$  vs  $0.62\% \pm 0.03\%$ ,  $P < 0.01$ )。结论: 凋亡素诱导HepG2细胞凋亡过程中存在细胞内Mcl-1 mRNA和蛋白水平的下降,以及线粒体细胞色素C的释放增加。凋亡素诱导的细胞凋亡可能与其下调细胞内Mcl-1 mRNA与蛋白水平有关。

**关键词** [HepG2细胞](#) [Myeloid cell leukemin-1](#) [凋亡素](#) [细胞凋亡](#)

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## Variation and significance of Mcl-1 mRNA and protein concentration in the apoptosis of HepG2 cells induced by apoptin

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### Abstract

<FONT face=Verdana>AIM: To investigate the variation and significance of mRNA and protein concentration of myeloid cell leukemin-1 (Mcl-1) in apoptotic HepG2 cells induced by apoptin. METHODS: The apoptin expression vector pCDNA3.0-VP3 was transfected into HepG2 cells via liposome. Mcl-1 mRNA was analyzed by real-time quantitative reverse transcriptase-polymerase chain reaction. The protein of apoptin, Mcl-1 and cytochrome C were detected by Western blotting. RESULTS: The VP3 gene was transfected into HepG2 cells successfully and expressed steadily. Compared to blank control, Mcl-1 mRNA and protein levels of VP3 positive cells were decreased (mRNA:  $0.09\% \pm 0.00\%$  vs  $0.41\% \pm 0.14\%$ ,  $P < 0.05$ ; protein:  $0.43\% \pm 0.01\%$  vs  $0.90\% \pm 0.04\%$ ,  $P < 0.01$ ). Released cytochrome C from mitochondrion was increased ( $0.98\% \pm 0.02\%$  vs  $0.62\% \pm 0.03\%$ ,  $P < 0.01$ ). CONCLUSION: In the course of the apoptosis of HepG2 cells induced by apoptin, the amount of Mcl-1 mRNA and protein is decreased, and released cytochrome C from mitochondrion is increased. The apoptosis induced by apoptin may be correlated with the down-regulation of Mcl-1 mRNA and protein.</FONT>

**Key words** [HepG2 cells](#) [Myeloid cell leukemin-1](#) [Apoptin](#) [Apoptosis](#)

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