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The effect of arsenic trioxide on human hepatoma cell line BEL-7402 cultured *in vitro*

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Abstract

AIM: To study the effect of a wide range of concentration of arsenic trioxide on human hepatoma cell line BEL-7402 and its mechanism.

METHODS: The BEL-7402 cells were treated with arsenic trioxide (a final concentration of 0.5, 1 and 2 μmol/L, respectively) in various durations or for 4 successive days. The cell growth and prolife ration were observed by cell counting and cell-growth curve. Morphologic change s were studied under electron microscopy. Flow cytometry was used to assay cell DNA distribution and the protein expression of *Bcl*-2 and Bax was detected by immunocytochemical method.

RESULTS: The cell growth was significantly inhibited by the different concentrations of arsenic trioxide as revealed by cell counting and cell growth curve. Arsenic trioxide treatment at 0.5, 1 and 2 μmol/L, resulted in a sub-G1 cell peak. The decreased G0/G1 phase cell and the increased percentage of S phase cell were observed by flow cytometer, suggesting that the inhibiting effect of arsenic trioxide on BEL-7402 cell lay in G_0/G_1 phase cell. Apoptotis related morphology, such as intact cell membrane, nucleic condensation, apoptotic body formation, can be seen under the electron microscopy. High protein expression level of *Bcl*-2 and Bax was detected in 1 and 2 μmol/L arsenic trioxide treated cells, but that of Bax was more significant. Arsenic trioxide treatment at 0.5 μmol/L resulted in higher expression level of *Bcl*-2 and lower expression level of Bax compared with control ($P_1 \leq 0.01$, $P_2 < 0.01$).

CONCLUSION: Arsenic trioxide not only inhibited the proliferation but also induced apoptosis of human hepatoma cell line BEL-7402. The induced apoptosis effect of 1 and 2 μmol/L arsenic trioxide was relative to the expression level of *Bcl*-2 and Bax.

Key words: Arsenic trioxide; Human hepatoma cell line; Apoptosis; Gene expression; *In vitro*; Genes suppressor, tumor

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