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Abstract

**PROAPOPTOTIC ACTIVITY OF EZHU OIL AND FURANODIENE ON
CULTURED HUMAN HEPG2 CELL**

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Ezhu, one of traditional Chinese medicines, is derived from three species of *Curcuma* including *Curcuma phaeocaulis* Val., *C. kwangsiensis* S. G. Lee et C. F. Liang and *C. wenyujin* Y. H. Chen et C. Ling according to Chinese Pharmacopoeia. In this study, we evaluate the cytotoxicity of ezhu oil (*C.wenyujin*) and a pure compound furanodiene isolated from ezhu oil to human HepG2 cells and investigate the underlying mechanism of the antiproliferative effect.

Ezhu oil has been found to be toxic to HepG2 cell and to induce cellular apoptosis. Ezhu oil causes cell cycle S/G2 phase arrest and the antiproliferative mechanism has been focused particularly on mitochondrial changes, the active caspase-3 enzyme expression, release of cytochrome c, the activation of caspases-3 and the cleavage of PARP.

Furthermore, to detect the toxicity of furanodiene on HepG2 cell, we attempted to characterize in detail the cell signaling cascades resulting in furanodiene-induced human HepG2 cell apoptosis, which caused G2/M arrest, focused particularly on mitochondrial changes, the role of p38 and ERK mitogen-activated protein kinase (MAPK) activation. We found that furanodiene-induced mitochondrial changes also included the release of cytochrome c, the activation of caspases-3 and the cleavage of PARP leading to the activation of p38 and suppression of ERK in HepG2.

This is the first study reporting the proapoptotic activities and mechanisms of ezhu oil and furanodiene in human HepG2 cells.

Key words: *Ezhu oil*, furanodiene, HepG2, apoptosis, caspase-3, mitochondria, cytochrome c, MAPK, ERK, P38.
