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Mechanism investigation of growth inhibition and apoptosis caused by Bruceine D in K562 cells

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Objective: To observe the changes of mitochondrial membrane potential (MMP), cytochrome c (Cyto-*c*), Caspase-3, 9 activity and cleavage of PARP, and determine the apoptosis signaling pathway in Bruceine D -treatment K562 cells.

Method: MTT assay was used to evaluate the cell growth inhibition of Bruceine D *in vitro*; Flow cytometry were performed to analyze MMP; Western Blot analysis was applied to detect Cyto-*c*, Caspases-3,-9 and PARP in K562 cells.

Results: IC_{50} value of Bruceine D against K562 cells was 6.37 ± 0.39 μM. The percentages of MMP after the treatment of 3.0,6.0,12.0 μM Bruceine D for 24 h were 79.84 ± 4.46%, 59.74 ± 7.48%, 40.66 ± 4.37% ($P<0.05$) respectively. The release of Cyto-c, activity of Caspase-3, 9 and cleavage of PARP increased compared with the control groups in the Bruceine D induced K562 cell. Moreover, Bruceine D could decrease Phosphorylation level of AKT and ERK.

Conclusions: The collapse of MMP, the increased Cyto-*c*, the up-regulation of Caspase-3, -9 activity and the augmented cleavage of PARP emerged after K562 cells treated by Bruceine D. The apoptosis of K562 cells induced by Bruceine D might be related to the mitochondrial pathway of apoptosis. Reduction of AKT and ERK Phosphorylation level might be mechanism of growth inhibition of K562 cells mediated by Bruceine D.

Biography

Jian-ye Zhang received BS of Pharmaceutical Sciences and MS of Pharmacognosy from Peking University, PhD of Oncology in Sun Yat-sen University. He has been a visiting scholar at School of Chinese Medicine, Hong Kong Baptist University for two years. He is currently Committee Member of the Society of Anti-Cancer Drugs, Chinese Anti-Cancer Association and Committee Member of Division of Tumor Pharmacology, Chinese Pharmacological Society.

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