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No protective effect of curcumin on hydrogen peroxide-induced cytotoxicity in HepG2 cells

Xiuping Chen[1,2], Zhangfeng Zhong[1,2], Zengtao Xu[1,2], Lidian Chen[3], Yitao Wang[1,2]

[1]State Key Laboratory of Quality Research in Chinese Medicine, Macau 999078, China

[2]Institute of Chinese Medical Sciences, University of Macau, Macau 999078, China

[3]Department of Pharmacy, Fujian University of Traditional Chhinese Medicine, Fuzhou, 350004, China

Correspondence: Yitao Wang, e-mail: ytwang@umac.mo; Xiuping Chen, e-mail: xpchen@umac.mo

Abstract:
Scavenging of intracellular reactive oxygen species (ROS) is one of the potential mechanisms contributing to the protective effects of many antioxidants. Curcumin, a natural product, is an effective ROS scavenger. However, the role of its ROS scavenging ability in its cytoprotective action remains to be clarified. Herein, the protective effects of curcumin on hydrogen peroxide (H_2O_2)- and *tert*-butyl hydroperoxide-induced ROS formation and HepG2 cell injury were determined. HepG2 cells were pretreated with curcumin for 30 min and then treated with H_2O_2 (500 μM) or *tert*-butyl hydroperoxide (200 μM) for 24 h. Curcumin pretreatment dramatically decreased H_2O_2- and *tert*-butyl hydroperoxide-induced ROS production, but failed to suppress cytotoxicity of those compounds. H_2O_2 induced decreases in mitochondrial membrane potential ($\Delta\Psi m$) and increases in DNA fragmentation could not be reversed by curcumin. Furthermore, curcumin enhanced expression of H_2O_2-induced pro-apoptotic protein Bax expression and inhibited expression of anti-apoptotic proteins Bcl-2 and Bcl-xL. In addition, curcumin significantly decreased p38MAPK and phospho-CDC-2 protein expression and increased phospho-p38MAPK, p42/44MAPK, and phospho-p42/44MAPK protein expression. These results suggest that short pretreatment and subsequent longer co-treatment of low concentrations of curcumin showed no obvious protective effect on H_2O_2-induced $HepG_2$ cell injury.

Key words:
curcumin, hydrogen peroxide, reactive oxygen species, cytotoxicity