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

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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 (H₂O₂)- 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 H₂O₂ (500 µM) or tert-butyl hydroperoxide (200 µM) for 24 h. Curcumin pretreatment dramatically decreased H₂O₂- and tert-butyl hydroperoxide-induced ROS production, but failed to suppress cytotoxicity of those compounds. H₂O₂ induced decreases in mitochondrial membrane potential (ΔΨm) and increases in DNA fragmentation could not be reversed by curcumin. Furthermore, curcumin enhanced expression of H₂O₂-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 H₂O₂-induced HepG2 cell injury.

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