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Curcumin Reduces the Expression of Main Fibrogenic Genes and Phosphorylation of Smad3C Signaling Pathway in TGFB-Activated Human HSCs. A New Remedy for Liver Fibrosis

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Abstract : The hepatic disease causes approximately 2 million deaths/year worldwide. Liver fibrosis is the last stage of numerous chronic liver diseases, and until now there is no definite cure or drug for it. Activation of hepatic stellate cells (HSCs) is the main reason for fibrosis. Transforming growth factor (TGF-β), as a main profibrogenic cytokine, if increased in these cells, leads to liver fibrosis through smad3 signaling pathways and increasing the expressions of Collagen type I and III, and actin-alpha smooth muscle (αSMA) genes. Curcumin (CUR) is a polyphenolic compound and an active ingredient derived from the rhizome of the turmeric plant that exerts effective antioxidant, anti-inflammatory, and antimicrobial activity. It has been shown that daily consumption of curcumin may have a protective effect on the liver against oxidative stress associated with alcohol consumption. In this study, we investigate the role of Curcumin in decreasing HSC activation and treating liver fibrosis. First, the human HSCs were treated with 2 ng/ml of (TGF-β) for 24 hours to become activated, then with Silibinin for 24 hours. Total RNAs were extracted, reversely transcribed into cDNA, Quantitative Real-time PCR, and western blot were performed. The mRNA expression levels of Collagen type I and III, αSMA genes, and the level of smad3 phosphorylation in TGF-β activated human HSCs treated with Curcumin were significantly reduced compared to human HSCs untreated with Curcumin. Curcumin is effective in reducing the expression of fibrogenic genes in the activated human HSCs treated with TGFB through downregulation of the TGF-β/smad3 signaling pathway. Therefore, Curcumin possesses significant antifibrotic properties in hepatic fibrosis

Keywords : hepatic fibrosis, human HSCs, curcumin, fibrogenic genes
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