

## How does emodin protect rat liver from fibrogenesis?

October 23 2009

In the last decade, advances in the understanding of genes promoting hepatic stellate cell (HSC) activation are impressive. However, there are few breakthroughs in therapeutic intervention of hepatic fibrogenesis. Efficient and well-tolerated antifibrotic drugs are lacking and current treatment of hepatic fibrosis is limited to withdrawal of the noxious agent. Research identifying innocuous antifibrotic agents is of high priority and urgently needed. Emodin is efficacious in the management of hepatic fibrosis. However, the mechanisms underlying its effects remain to be elucidated.

A research team from China established rat models of experimental hepatic fibrosis by injection with CCl4; the treated rats received emodin via oral administration at a dosage of 20 mg/kg twice a week at the same time. Rats injected with olive oil served as a normal group. Histopathological changes were observed by hematoxylin and eosin staining. The activities of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) in serum and hepatic hydroxyproline content were assayed by biochemical analyses. The mRNA and protein relevant to hepatic stellate cell (HSC) activation in the liver were assessed using real-time reverse transcription-polymerase chain reaction, immunohistochemistry, western blotting and enzyme-linked immunosorbent assay. Their study will be published on October 14, 2009 in the *World Journal of Gastroenterology*.

The results showed that the degree of hepatic <u>fibrosis</u> increased markedly in the CCl4 group compared to the normal group, and



decreased markedly in the emodin group compared to the CCl4 group according to METAVIR scale compared with those in the normal control group. The activities of serum ALT and AST were significantly higher in rats injected with CCl4. The activities of serum ALT and AST were significantly reduced by administration of emodin. Compared with the normal controls, hepatic hydroxyproline content was significantly higher in rats injected with CCl4. Hepatic hydroxyproline content was significantly reduced in the rats treated with emodin at 20 mg/kg. Emodin significantly protected the liver from injury by reducing serum AST and ALT activities and reducing hepatic hydroxyproline content. The mRNA levels of transforming growth factor-b1 (TGF-b1), Smad4 and a-SMA in liver tissues were significantly down-regulated in SD rats that received emodin treatment. Furthermore, significant downregulation of serum TGF-b1 protein levels and protein expression of Smad4 and a-SMA in liver tissues was also observed in the rats. Emodin inhibited HSC activation by reducing the abundance of TGF-b1 and Smad4.

The researchers drew a conclusion that emodin is active as an antifibrogenic drug to reduce the biological effects of TGF-b1 in ongoing fibrogenesis. Emodin, the main active monomer isolated from Giant Knotweed Rhizome, may be an attractive therapeutic agent for the treatment of fibrotic liver diseases.

<u>More information</u>: Dong MX, Jia Y, Zhang YB, Li CC, Geng YT, Zhou L, Li XY, Liu JC, Niu YC. Emodin protects rat liver from CCl4-induced fibrogenesis via inhibition of hepatic stellate cells activation. World J Gastroenterol 2009; 15(38): 4753-4762, www.wjgnet.com/1007-9327/15/4753.asp

Source: World Journal of Gastroenterology (<u>news</u> : <u>web</u>)



Citation: How does emodin protect rat liver from fibrogenesis? (2009, October 23) retrieved 6 May 2024 from <u>https://medicalxpress.com/news/2009-10-emodin-rat-liver-fibrogenesis.html</u>

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