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P#49

Persistent Activation of p38 MAPK in CCl_4-Induced Rat Liver Cirrhosis

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The hepatic stellate cell (HSC) is the primary cell-type in the liver responsible for excess collagen synthesis during hepatic fibrosis. Previous our studies, however, has demonstrated that the number of HSC decreased on stage of liver cirrhosis rather than stage of liver fibrosis. We therefore supposed that there is another pathway of collagen synthesis on the stage of liver cirrhosis and investigated relationship between MAPK signaling cascade, one of intracellular signaling pathway which is stimulated in activated HSCs, and CCl_4-induced rat liver cirrhosis. The maximal activation of phosphorylated c-Jun-NH_2-terminal (p-JNK) and phosphorylated extracellular regulated kinase (p-ERK) was detected weeks 12, stage of liver fibrosis and mild cirrhosis, after treatment and decreased weeks 14, stage of severe cirrhotic condition Phosphorylated p38 MAPK (p-p38), however, was detected since weeks 8 and persistently increased until weeks 14. p-JNK was colocalized with α -SMA and p-ERK was colocalized with ED1. p-p38 was localized in fibrous septa and seemed like that colocalized with p-ERK from week 8 to 12, however, p-p38 was increased at weeks 14 conversely. p38 MAPK was known to inhibits the proliferation rate of HSCs and regulate α1(I) collagen gene expression and increase α1(I) collagen mRNA stability and believed p38 MAPK to be may inhibit proliferation through an antagonistic effect on the cell cycle proliferation associated protein cyclin D1. These results show that p38 may be critical role to inhibit the proliferation rate of producer cells of extracellular matrix on liver cirrhosis and provide the first *in vivo* demonstration of liver cell-type specific and time-course activation of MAP kinase cascade during the process of liver fibrosis and cirrhosis.

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P#50

Immunohistochemical Characterization of Canine Haemangiopericytoma Occurs on The Forelimb.

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