basal promoter activity and b) p53 can induce the caspase-8 promoter via an yet unknown mechanism but independently of SP1 binding.

**IS THERE ANY LIVER ANTI-FIBROTIC EFFECT OF K-17.22? AN EXPERIMENTAL STUDY WITH IMMUNOHISTOCHEMICAL ANALYSIS IN A RAT MODEL.**

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The aim of this study was to test in a liver fibrosis model a novel natural compound, which has been shown to significantly decrease GPT level in HCV-patients. 150 SD rats were allocated into 2 groups. A) fibrosis model (02ml/kg CCL4 i.p. injection twice/week); B) as A but added with K-17.22 50mg/kg (Kyotsu, Japan). At sacrifice liver samples were used for histology, hydroxyproline determination, immuno-histochemical analysis of activated stellate cells, Northern blot analysis of Tissue Inhibitor Metallo Proteinase-1 (TIMP-1) and a2-procollagen mRNA. Blood biochemistry, Type IV collagen levels was not affected, hyaluronic acid level increased over 2-fold in A group (p<0.001). However, this was completely prevented by K-17.22 (p<0.001 vs A) Accordingly, increased GOT, GPT, ALP and Bilirubin level (p<0.001 vs healthy control) significantly improved in B group (p<0.05 vs A). Group A showed an increased hydroxyproline content (190± 205 vs 343±61, p<0.001) which was significantly reduced by the supplementation with K-17.22 (p<0.05 vs A). Histology of liver in A group showed the typical ongoing fibrosing features while an overt reduction of the above morphological abnormalities appeared in group B. K-17.22 significantly decreased the number of activated stellate cells and the expression and densitometric assessment of both TIMP-1 and a2-procollagen mRNA (p<0.05). These data suggest that the present novel phytotherapeutic compound K-17.22 exerts a potent antifibrotic effect and further studies are awaited to corroborate its clinical potential.

**METADOXINE PARTIALLY PREVENTS FIBROSIS AND COMPLETELY PRESERVES GLYCOCEN STORES IN PROLIFERATIVE BILIARY OBSTRUCTION IN THE RAT: A COMPARISON WITH COLCHICINE.**

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**Category 3: Molecular and cell biology (gene expression, signalling, fibrosis)** 79

**ANALYSIS IN A RAT MODEL.**

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**DIFFERENTIAL GENE EXPRESSION PROFILE IN BUDD-CHIARI SYNDROME AND CIRRHOSIS.**

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Budd-Chiari syndrome (BCS) induces necrosis, regeneration, and fibrosis without chronic inflammation. To gain further insights into the pathogenesis of liver injury, we investigated whether the expression of a set of genes known to be involved in chronic inflammatory liver diseases differs in BCS livers.

**Materials and Methods** Using real-time RT PCR, mRNA expression of 35 genes involved in extracellular matrix regulation, growth factors and angiogenesis was quantified in liver explants from patients with BCS (5