gland tumors, as also reported for human breast cancer. It was also possible to show that the immunoexpression of HER-2/neu (c-erbB-2) can be a factor in mammary carcinogenesis. This fact opens the possibility of using anti-HER-2/neu(c-erbB-2) antibodies in the diagnosis and treatment of canine mammary gland tumors such as in human case.

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P#48

Inhibition of Renal Fibrosis by ENA-A Resources

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Adriamycin (ADR), which is widely used in the treatment of various neoplastic conditions, exerts diverse side effects in several organs. Nephrotoxicity of adriamycin has been recently documented in a variety of animal species. The present study was designed to investigate the effect of ENA-A Resources, active alkaline mineral water on the ADR-induced fibrosis. This renal fibrosis rat model induced by adriamycin (ADR) had been reported, but it was not often in mice in intraperitoneal (I .P) approach. The study was carried out with male BALB/C mice. Test animals were divided into four groups of fifteen mice each as follows: Group I; control group (saline, I.P). Group II; ADR group (ADR, 5mg/kg body wt, I.P, twice a week). Group III; 5%ENA+ADR group (ADR, 5mg/kg body wt, I P, twice a week and 5% ENA-A Resources solution by drinking water). Group IV; 10%ENA+ADR group (ADR, 5mg/kg body wt, I.P, twice a week and 10% ENA-A Resources solution by drinking water). Intraperitoneal (I.P) injections of adriamycin (ADR) resulted in significant decrease of body weight in all the ADR-treated groups and death of the mice. Ten days after ADR treatment, there was a few histological changes observed. Glomeruli and tubular epithelial cell damaged, inflammatory cells infiltrated, interstitial fibroblasts proliferated, extracellular matrix and collagen deposited, and these histopathological changes became more and more worse went with the course of the experiment. The ENA-A Resources inhibited against ADR-induced renal fibrosis and delayed the life-span compared with ADR-injected mice. These data suggest that administration of ENA-A Resources is a promising approach in the treatment of nephrosis caused by ADR.

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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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Immunohistochemical Characterization of Canine Haemangiopericytoma Occurs on The Forelimb.

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