

effects on mice. By i.p. administration it increased the weight and cell number of mouse spleen, elevated the response of mouse spleen against sheep red blood cells, and stimulated phagocytic activity of peritoneal macrophages [1]. The number of activated macrophages in the spleen of the treated animals was also increased. If the polysaccharide fraction was given i.v. or intragastrically, even at higher doses, the phagocytic function of peritoneal macrophages did not change significantly [13]. Astragalin II decreased the alkaline RNase activity in liver and spleen of mice and had a smaller effect on acid RNase but no effect on serum RNase. The polysaccharide fraction also increased hepatic RNase inhibitor activity [14].

The natural killer cytotoxicity of lymphocyte effector cells was markedly enhanced when treated with partially purified human interferon- α or with extract of *Astragalus*. They stimulated each other: the natural killer cytotoxicity increased five- to sixfold after treatment of effector cells with both agents [15].

Saponin astramembrannin I, at a dose of 10 mg/kg applied i.v., induced accumulation of cAMP in rabbit plasma. The increase in cAMP started after 30 min and reached a maximum in 0.5–4 h after a single injection. Saponin affected DNA biosynthesis in partially hepatectomized mice and increased incorporation of [3 H]thymidine into regenerating mouse liver [16].

Antiinflammatory effects of astramembrannin I were demonstrated in rats. It inhibited the increase in vascular permeability induced by serotonin or histamine when given i.v. at a dose of 5 mg/kg or orally at a dose of 50 mg/kg. Oral administration of astramembrannin I caused a dose dependent reduction in carrageenan-induced edema of the hind paw of rats. Hypotensive activity of astramembrannin I was observed after i.v. administration of 15 or 10 mg/kg to anesthetized cats or rats [17].

A clinical effect of *A. membranaceus* in the treatment of chronic hepatitis was also reported. Elevated levels of serum GPT returned to normal in 1–2 months, and symptoms were relieved. Patients had a good appetite and a sense of well-being after treatment, without showing significant side effects. Experiments on animals with toxic liver damage induced by CCl $_4$ indicated that the root of *A. membranaceus* might protect the liver, prevent decrease of hepatic glycogen contents, and raise the levels of total serum protein and albumin [18].

Phagocytosis of the reticuloendothelial cells of patients with chronic hepatitis was also stimulated, and cellular immunity was enhanced [18].

A decoction of the seed of *A. complanatus* given orally to mice increased the lymphocyte transformation rate and thus specific cellular immunity; however, the treatment had no effect on the spleen index [19].

References

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