polysaccharide, PB, contains galactose, arabinose, and rhamnose in a ratio of 3.4:1.7:1. Partial acid hydrolysis of PB yields α-(1→4)-D-galacturonan [21].

14.3 Pharmacology

Andrographolide and the related compounds, deoxyandrographolide, deoxydidehydroandrographolide, and neoandrographolide, were investigated for their pharmacological properties in experimental animals including mice, rats, and rabbits. They all showed varying degrees of antipyretic and antiinflammatory activity in animals with fever induced by 2,4-dinitrophenol or endotoxin, with edema caused by egg white, or with inflammation caused by croton oil. The antipyretic and antiinflammatory effects of andrographolide and related compounds, however, were lower than those of corticosteroids and of conventionally used nonsteroidal drugs. The pharmacological effect was highest with deoxydidehydroandrographolide, followed by deoxyandrographolide, neoandrographolide, and andrographolide. The minimal lethal dose of these compounds by oral administration was greater than 20 g/kg. The antiinflammatory effect of all four compounds disappeared in adrenalectomized animals, indicating a possible involvement of the pituitary and adrenal systems. Administration of the four compounds did not significantly affect inflammatory hyperplasia and migration of leukocytes into the inflammatory focus. The antiinflammatory mechanism was speculated to be different from that of conventional drugs [22].

Neither the extracts of leaves and stems of *A. paniculata* by oral administration nor andrographolide by s.c. or oral administration had an effect on blood sugar levels of normal or diabetic rats [23].

Pretreatment of dogs with leaves of *A. paniculata* at a dose of 500 mg/kg or with andrographolide at a dose of 5 mg/kg prevented the increase in serum levels of glutamic-oxalacetic transaminase (GOT) and GPT and the decrease in the liver levels of these enzymes when induced by oral administration of CCl_4. Simultaneous treatment did not show any effect. An inhibitory effect on formation of trichloromethyl free radical from CCl_4 is a likely explanation [24]. Pretreatment also caused a decrease of CCl_4-induced hepatic microsomal lipid peroxidation but only with a single dose and not with long-term administration of leaf extract or andrographolide. The protective action on CCl_4-induced hepatotoxicity of leaf extract was more significant than that of andrographolide [25]. NADPH-induced hepatic microsomal lipid peroxidation could not be significantly changed by oral administration of leaf extract or andrographolide [25].

After i.v. or intragastric administration to mice [^{3}H]andrographolide is rapidly absorbed and eliminated consistent with an open two compartment kinetic model. The $t_{\frac{1}{2}\alpha}$ and $t_{\frac{1}{2}\beta}$ values of i.v. administered [^{3}H]andrographolide were 0.06 and 4.7 h, respectively, whereas the corresponding values following intragastric application were 0.15 and 1.07 h, respectively. Intragastric [^{3}H]andrographolide was rapidly absorbed. It rapidly distributed to other organs, especially gallbladder, kidney, ovary, and lung. Radioactivity was low in spleen, heart, and brain. Approximately 90% of the intragastrically administered [^{3}H]andrographolide was excreted in urine and feces at 24 h and 94% after 48 h. The calculated biological availability of intra-