Iron is an essential element for living organisms. However, as it is a transition metal, it can participate in Fenton reaction resulting in generation of free radicals and oxidative damage to tissues. Antioxidants may prevent possible iron toxicity by chelating free iron or scavenging free radicals. Falvonoids are naturally occurring substances that are capable of formation of complexes with metals, including iron. They have been show to possess antioxidant activity, which depends on molecular complexity of numerous types of flavonoids, e.g. quercetin and silibinin. Bisphosphonates are synthetic drugs used to treat various metabolic diseases of bones. Their principál effect is an inhibition of osteclast activity leading to a decreased bone resorption. Bisphosphonates have been however shown to exert some antioxidant activity in *in vitro* experiments, too.

The aim of this PhD thesis was to investigate the role of iron in toxicity of other metals (cadmium) and the effect of flavonoids (quercetin and silibinin) and bisphosphonates (clodronate, etidronate and risedronate) on iron-induced oxidative damage *in vivo.* Experiments were performed in male mice (CD-1, Charles River, 25-35 body weight). Iron was administered intraperitoneally or in the diet. Cadmium was administered subcutaneously. Flavonoids and bisphosphonates were administered orally. The level of lipoperoxidation, the content of glutathione in the liver and activities of catalase and glutathione peroxidase in the liver, and superoxide dismutase in erythrocytes were measured. Tissue content of cadmium, iron and trace elements was also evaluated. Iron administration induced oxidative damage to tissues (increased lipoperoxidation and, in some cases, decreased glutathione level, decreased aktivity of superoxide dismutase and glutathione peroxidase). Diets with different content of iron influenced oxidative effects of cadmium (a pronounced lipoperoxidation by cadmium in animals fed diet with high iron content). Both anti- and pro-oxidative effect of quercetin in dependence on the dose was shown. High doses led to oxidative damage (lipoperoxidation induction and decreased glutathione levels), whereas lower dose administered for a long period (7 weeks) had antioxidative effect (lower lipoperoxidation and increased activity of glutathione peroxidase). The influence of quercetin on iron-induced oxidative damage was low, if any. A positive effect on iron-decreased glutathione level was observed. A decrease in iron accumulation in the liver after quercetin administration may support chelating properties of quercetin. Predominatly pro-oxidative activity of silibinin characterised by induced lipoperoxidation and decreased glutathione level was observed. Moreover, ironinduced lipoperoxidation was further increased by silibinin. Silibinin did not affect ""on distribution in mice. A certain antioxidative effect of silibinin was emonstated, however (a positive effect on iron-decreased glutathione level). Bisphosphonates were shown to have antioxidant activity against iron-induced oxidative damage (decreased iron-induced lipoperoxidation and higher glutathione level). All bisphosphonates had significant effect on distribution of iron and other trace elements.