SUMMARY

Sulphur (S) is an essential macronutrient for plants as a constituent of proteins (in sulphur-containing amino acid residues or iron-sulphur clusters), membrane sulpholipids, glutathione and many other compounds. Sulphur deficiency affects plant growth and development. Previous studies of S-deficient plants were focused mainly on transcriptomic and metabolomic analyses and indicated possible modifications of energy metabolism. Biochemical studies characterizing these changes were scarce. The aim of the presented thesis was to analyze the structure and functioning of the respiratory chain and the role of mitochondria in cellular reactive oxygen species (ROS) metabolism in Arabidopsis thaliana grown under long-term sulphur deficiency. It has been shown that S deficiency altered the functioning of the mitochondrial electron transport chain, leading to a lower activity of protein complexes (I and IV). The studies of additional components of the plant respiratory chain indicated an increased activity of type II external NADH dehydrogenases and an unchanged activity of alternative oxidase and type II internal NADH dehydrogenases. The plasticity of the plant respiratory chain allowed to survive S deficiency, however it has also led to a new energy and redox homeostasis expressed by a lower ATP level and a higher reduction state of pyridine nucleotides. There was no effect of S deficiency on the structure of oxidative phosphorylation system. S deficiency resulted in increased production of superoxide anion in mitochondria and higher levels of hydrogen peroxide in leaves and roots. The levels of low-molecular-mass antioxidants, glutathione and ascorbate, were lower than those in control plants, while the anthocyanin level was higher. The increased content of ROS led to the activation of enzymatic antioxidant defence systems. However, the oxidative stress was present in leaves of S-deficient A. thaliana, as indicated by increased protein carbonylation, observed mainly in chloroplasts. In mitochondria, the level of protein carbonylation was similar to that in the control plants, probably due to an increased activity of mitochondrial isoform of superoxide dismutase and further higher activity of proteases removing oxidized proteins. S-deficiency caused a lower level of non-protein thiols. However, the level of protein thiols remained unchanged. Probably, the preferential incorporation of S to the protein thiols is important for the survival of S-deficient plants. In the dissertation the role of mitochondria in the adaptation of plants to S deficiency is discussed.