Mitochondria are the main organelle responsible for cellular energy metabolism and redox homeostasis. Energy storage molecule adenosine triphosphate (ATP) is produced in mitochondria due to ATP synthase activity (complex V). Its activity is driven by a flux of protons across a gradient generated by electron transfer between basal oxidoreductases of mitochondrial electron transport chain (mtETC) – complex I, III and IV. The regulation of reduction state of the cell is achieved mainly due to alternative pathways activity in (mtETC), which consist of alternative oxidase (AOX) and type II (rotenone-insensitive) NAD(P)H dehydrogenases. Cyanide-resistant AOX transfers electrons from reduced ubiquinone to oxygen omitting two coupling places (complex III and IV of mtETC), thus lowering energetic efficiency of respiration. The genetic basis, biochemical features and physiological role of AOX is well documented. In opposite to that, there is limited research concerning type II NAD(P)H dehydrogenases, which bypass of complex I. It was found that *Arabidopsis thaliana* (a model plant) genome encodes 7 isoforms of type II NAD(P)H dehydrogenases and its proteins are localized in the inner mitochondrial membrane from the matrix side and the intermembrane space. Type II NAD(P)H dehydrogenases are small proteins and their activity is not linked with proton gradient formation therefore is not controlled by energy status of the cell. It was postulated that they may work as the "safety valve" preventing over-reduction of cell but in light of the latest data, their function seems to be more complicated. Using reverse genetics approach together with molecular, biochemical and physiological research we would like to study the role of type II NAD(P)H dehydrogenases *in vivo*.