It has been known for years that mitochondria, energy centers of cells, not only have their own DNA, but also a system of gene expression separate from the nuclear one. It encompasses many steps, one of which is the synthesis of proteins (translation) carried out by specific complexes, called ribosomes. They consist of two subunits: small and large, which contain proteins and ribosomal RNA (rRNA). During our studies of the mitochondrial translation in *Arabidopsis rps10* mutant, whose ribosomes did not form properly due to deficiency of the S10 protein (one of the proteins in the small ribosomal subunit), we observed some intriguing changes at the post-transcriptional level. These included, among others, a drastic increase in the number of transcripts from non-coding areas and problems with the correct formation of the 18S rRNA transcript. The literature data revealed that similar changes were previously observed in an *Arabidopsis* mutant deficient in a key enzyme in the metabolism of mitochondrial RNA, 3'->5' exonuclease, mtPNPase (polynucleotide phosphorylase). The main role of this enzyme is the degradation of the by-products generated during RNA maturation and defective transcripts. The aim of this project is to discover how reduced level of ribosomal S10 protein affects mtPNPase functionality and thus some aspects of RNA metabolism in *Arabidopsis* mitochondria. Our preliminary data indicate that the mtPNPase level of transcript and protein increases in *rps10*, however, some disturbances occur during the formation of PNPase complexes. The effect of the reduced amount of S10 protein in mtPNPase complex formation may be due to an altered mitoribosome-enzyme interaction or may be related to not yet known extra-ribosomal function of S10 protein. We favor the first option, but both will be tested. To understand how deficiency of S10 protein leads to impaired homeostasis of mtPNPase complexes, we will monitor the size, composition and integrity of the complexes formed by mtPNPase and/or by S10 protein by various biochemical methods, molecular and cell biology. We also plan to perform *"omic"* approach, complexome profiling, that will enable the detection of differences in the formation of complexes related to RNA metabolism between *rps10* and wild type. In parallel with the study of the complexes, we will determine the level of the known PNPase substrates in *rps10*, and we will also try to investigate if other defects of mitoribosome biogenesis, not related to S10 deficiency, affect the functionality of PNPase. An additional goal of this project is to identify the mechanism that leads to the formation of monocistronic transcripts from the bicistronic units in the *rps10* mitochondria. Two non-mutually exclusive mechanisms will be tested. PNPase plays an important role in the first one and RNA polymerase in the other. We would like to emphasize that RNA metabolism in plant mitochondria is not similar to RNA metabolism in animal and yeast mitochondria. In contrast to the study of animal and yeast mitochondria, the knowledge of the relationship between the translation apparatus and post-transcriptional processing in plant mitochondria is extremely poor, making this project innovative. Our study may reveal the function of the S10 protein outside the ribosome and/or deepen our understanding of the regulatory function of ribosomes by challenging the still common dogma that the ribosome has no influence on the other stages of gene expression than translation.