

Mitochondria are cellular organelles that play a central role in the production of ATP, which is then consumed for all types of cellular functions, starting from metabolism to cellular movement. Mitochondria are constantly changing shape through fusion, fission and swelling, to best meet the energy requirements of the cell. The mechanisms of this process are still not fully understood, even though it is known that altered mitochondrial dynamics is linked to various pathologies. The inner mitochondrial membrane is a complex structure composed of mitochondrial cristae. Mitochondrial cristae change during metabolism, and their opening is critical for the progress of apoptosis. The above processes, i.e., swelling, fusion and fission, must affect the mechanical tension in the mitochondrial membranes. However, data on the mechanical sensitivity of mitochondria are almost non-existent. Mechanical stress in the membrane is perceived directly by mechanosensors located in the lipid membranes. The special class of these proteins are mechanosensitive channels that react to changes in membrane tension by opening of their pores and allowing the flow of ions, what in turn regulates electrical potential and compartment volume. Recently, we have discovered that one of mitochondrial channels is mechanosensitive. In our project we would like to investigate the effects of stretching of mitochondrial membrane in various experimental systems: in isolated membrane patches, in intact isolated mitochondria and in mitochondria in intact cells to check, how the channel responds to mechanical stress under these conditions. We hypothesize that mechanosensitive channel could react to mechanical stimuli under all these circumstances. We would like also to prove our premise that the mechanosensitive channel plays an important role in fusion and fission of mitochondria. To this end, we would like check mitochondrial dynamics - how mitochondria fuse and divide - inside the cells, which do or do not express the mechanosensitive channel. Our project as one of few carried out up to date will directly address the mechanosensitivity of mitochondria and possible regulation of mitochondria functions *via* mechanical forces. This is very intriguing since dysfunction of the mitochondria underlies etiology of many severe disorders and recognition of their mechanosensitivity could lead to new types of treatment.