Abstract

The mitochondrial permeability transition pore (mPTP) is highly evolutionarily conserved channel found in the inner membrane of mitochondria. This pore is non-selectively permeable for molecules below 1,5 kD. Consequences of the pore opening due to an increase of Ca^{2+} or reactive oxygen species (ROS) and following depolarization of the membrane involve a disruption of the proton gradient, decrease in the production of ATP and prevalently a cell death.

Death of a cell as a result of the mPTP opening is a physiopathological mechanism which follows ischemic diseases and neurodegenerative disorders such as Alzheimers and Huntingtons disease.

Study of a structure and function of mPTP is essential for the research of mechanisms and progression of diseases, and it is crucial for the development of responding drugs and an overall decrease in the morbidity of the patients.

This work compiles the course of the research into structure and function of the channel under physiological and pathological conditions and briefly puts down some of the experimental methods.