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On the Structure and Function of the ATF Active K^+ Channel

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Abstract: Various ion channels and ion transport systems located in the inner membrane of the mitochondria play an important role in the processes of signal transmission, cell function and death in the cell. Due to the importance of the role of these ion-transport systems in cell function, the study of their control mechanisms is relevant. Mitochondrial ion transport systems are involved in intracellular Ca^{2+} - signaling, ion homeostasis, and a number of physiological processes

Key words: ion channels, ion homeostasis, ion-transport systems, Ca^{2+} - signaling

Introduction

The MitoK^+ / ATF -channel is also one of the ion transport systems that plays an important role in mitochondrial functional activity. The role, pharmacology and regulation of this channel in cell activity are widely studied. It is known that in the mitochondria K^+ uniport transport system, K^+ / H^+ - changers play an important role in the management of the normal size of mitochondria [1]. MitoK^+/ATF -channel has been shown to play a protective role in pathological conditions such as ischemia [2]. In the mitochondria, ion transport plays an important role in normal and pathological conditions [Kachaeva., 2007].

Mitochondria is also characterized by the presence of outer and inner membranes as well as various cation and anion channels located in them [Bernardi., 2015;]. Among these channels, the Ca^{2+} - dependent mega-channel, the ATF-dependent K^+ channel, and various ion channels are particularly important. The following is an analysis of the structural and functional processes of these canals based on the data presented in the literature.

The structure and function of the ATF-dependent K^+ - channel (mitoK^+/ ATF) have been studied in detail by many authors. Pathological conditions such as stroke, heart attack, ischemia are directly related to the disruption of oxygen homeostasis at the cellular level and the occurrence of hypoxia. In addition, the conformation and control of the K^+ channel (mitoK^+/ATF-channel) associated with mitochondria ATF in the normal evening of mitochondria physiology is important. This channel belongs to the family of ATF-dependent K^+ channels.

The conformation and function of the mitoK^+/ATF-channel are currently being studied by a number of authors[1,2,3]. The main function of the Mito$K$+/ ATF-channel is to ensure the entry of K^+ ions into the mitochondria matrix. It is assumed that the transport of ions from the K^+ membrane by transport diffusion in the attenuated state of entry of K^+ mitoK^+/ATF-channel.

The MitoK^+/ATF-channel is located inside the mitochondria membrane. According to its structural unit, it is close to a group of cytoplasmic channels. This channel consists of subunits and managers. Channel subunits with a molecular weight of 43–46 kDa form the basis of the mitoK^+/ATF-channel and are not controlled by specific modulators. In addition, the channel contains subunits with a molecular weight of 60–174 kDa, which are sensitive to specific modulators and are controlled by their action. The regulatory function of the subunits is performed by a protein moiety combined with sulfamochevina glibinklamide. At present, the activity of mitoK + / ATF-channel using the Petch-clamp method is sufficiently studied. Experimental data confirm the heterogeneity of MitoK^+/ATF-channel protein components.

There is currently a K^+/ H^+ - exchange uniport system that transports K^+ ions in mitochondria. This uniport ion transport system is inhibited by ATF at 55 kDa.

The MitoK^+/ATF-channel is involved in the normal course of mitochondria physiology, stabilizing mitochondria volume. At present, a number of modulators that control the activity of mitoK^+ / ATF-channel and the mechanisms of their effect on channel function are sufficiently studied. For example, modulators such as diazoxide, nicorandil serve to increase the activity of mitoK^+/ ATF-channel. Modulators such as cromacalim, pinacidil, and apricalim simultaneously increase the activity of mitoK^+ / ATF and tsitoK^+ / ATF channels. MitoK^+/

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$_{ATF}$ channel inhibitors (ATF, glibinklamide) are used to regulate the channel. The mitochondrial membrane serves to provide metabolic exchanges between the cell cytoplasm and through the function of ion transport systems located in the membrane, the exchange of ions required for the oxidative phosphorylation process inside the mitochondria is carried out. Through the mitochondrial membrane, various anions are transported together with N^+ by the simport method or by antiport to ON^- anions. The inner membrane of the mitochondria contains ion exchange systems that carry cations. In summary, the uniport transport system of Ca^{2+} ions is located in the mitochondrial membrane. The mito$K_{+/ ATF}$ - channel located in the mitochondrial membrane has been shown to act as a protector in tissue cells.

References

1. Alavian K.N., Beutner G., Lazrove E., Sacchetti S., Park H.A., Licznerski P., Li H., Nabili P., Hockensmith K., Graham M., Porter G.A. An uncoupling channel within the c-subunit ring of the F1FO ATP synthase is the mitochondrial permeability transition pore// – Proc. Natl. Acad. Sci. – USA. – 2014) – P. 10580-10585.
2. Bernardi P., Di Lisa F. The mitochondrial permeability transition pore: molecular nature and role as a target in cardioprotection // J Mol Cell Cardiol. – 2015. – V.78. – P. 100-106.
3. Juhaszova M., Wang S., Zorov D.B., Nuss H.B., Gleichmann M., Mattson M.P., Sollott S.J. The Identity and Regulation of the Mitochondrial Permeability Transition Pore. Where the Known Meets the Unknown //– Ann. N.Y. Acad. Sci. – 2008. –V. 1123. – P. 197–212.