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Cytoskeleton and regulation of mitochondrial function: The role of beta-tubulin II and plectin

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The control of mitochondrial function is a cardinal issue in the field of cardiac bioenergetics, and the analysis of mitochondrial regulations is central to basic research and in the diagnosis of many diseases. Interaction between cytoskeletal proteins and mitochondria can actively participate in mitochondrial regulation. Potential candidates for the key roles in this regulation are the cytoskeletal proteins plectin and tubulin. Analysis of cardiac cells has revealed regular arrangement of β-tubulin II, fully co-localized with mitochondria. β-tubulin IV demonstrated a characteristic staining of branched network, β-tubulin III was matched with Z-lines and β-tubulin I was diffusely spotted and fragmentary polymerized. In contrast, HL-1 cells were characterized by the complete absence of β-tubulin II. Comparative analysis of cardiomyocytes and HL-1 cells with cardiac phenotype revealed a dramatic difference in the mechanisms of mitochondrial regulation. In the heart, colocalization of β-tubulin isotype II with mitochondria suggests that it can participate in the coupling of ATP-ADP translocase (ANT), mitochondrial creatine kinase and VDAC. This mitochondrial supercomplex is responsible for the efficient intracellular energy transfer via the phosphocreatine pathway. We found also that in skeletal muscle of plectin knockout mice, mitochondrial content was reduced; mitochondria were aggregated in sarcoplasmic and subsarcolemmal regions, and were no longer associated with Z-disks. Our results show that the depletion of distinct plectin isoforms (P1b and P1d) affects mitochondrial network organization and function in different ways. Existing data suggest that cytoskeletal proteins may control the VDAC, contributing to the regulation of mitochondrial and cellular physiology.

Biography

Andrey V Kuznetsov has worked on the problems of delicate mechanisms of energy metabolism in the heart and their changes under pathophysiological conditions. He has studied mitochondrial function and regulation in normal cells and in various pathological states, in particular in the field of mitochondrial cardiac system, mitochondrial interactions with other intracellular structures and systems, performing analysis of mitochondria *in situ* and *in vivo*. Also, he studied the roles of mitochondrial damage in ischemia reperfusion injury, apoptosis, calcium homeostasis, oxidative stress, mitochondrial ROS production, cardiac and muscle diseases with imaging of mitochondria and mitochondrial function, mitochondrial dynamics (fission, fusion, motility) in various living cells. He has extensive knowledge in cardiac bioenergetics and mitochondrial physiology and experience in optical imaging (fluorescent, two-photon) of single cell and single mitochondria, imaging of mitochondrial Ca2+, ROS, membrane potential, mitochondrial dynamics for basic science and in clinically oriented studies.

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