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МАТЕРИАЛЫ КОНФЕРЕНЦИИ  
И ШКОЛЫ

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**Cd<sup>2+</sup>, POTASSIUM CHANNELS AND RESPIRATORY CHAIN  
OF MITOCHONDRIA: MOLECULAR MECHANISM(S) OF INTERACTIONS**

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It is considered now that nonselective pore of inner mitochondrial membrane, which plays an important role in regulation of different types of cell death, is formed by conformational changes and assembling of several preexisted proteins of mitochondria. In particular, F<sub>1</sub>F<sub>0</sub>-ATPase, cyclophilin D, adenine nucleotide translocase, P<sub>i</sub> carrier, various components of mitochondrial electron transport chain, such as I, III and IV respiratory complexes as well as some other proteins participate in its formation and/or regulation. It is known also that respiratory complex I can play in some cases the role of Na<sup>+</sup>/H<sup>+</sup> exchanger, the complex II is involved in formation/regulation of ATP-dependent potassium channel of mitochondria (mitoK<sub>ATP</sub>), while the complex IV – in formation and/or regulation of mitochondrial Ca<sup>2+</sup>-activated large conductance potassium channel (mitoBK<sub>Ca</sub>). Previously we have shown that cytotoxic action of divalent metal ions of cadmium

(Cd<sup>2+</sup>), which is one of the most important environmental pollutants that is extremely dangerous for all living things, is modulated by effectors of the nonselective pore and the respiratory chain. The aim of the present work was comparative investigation of mechanism(s) of modulating action of various effectors of two mentioned above K<sup>+</sup> channels against the toxic action of Cd<sup>2+</sup>, namely diazoxide (mitoK<sub>ATP</sub> activator) or NS1619, NS004 and paxilline – mitoBK<sub>Ca</sub> openers/inhibitor, correspondingly. As an experimental model, isolated liver mitochondria and two cell lines (AS-30D, PC12) of rat were used. Possible involvement of mitochondrial respiratory chain in modulating action of the K<sup>+</sup> channels effectors against the Cd<sup>2+</sup>-induced toxicity and cell death is discussed.

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