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Molecular composition of the mitochondrial permeability transition pore.

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Mitochondrial damage caused by calcium overload is a critical step in stress-induced cell death during stroke and heart attack. This damage is caused by dramatic increase in permeability of the mitochondrial inner membrane – a phenomenon known as Permeability Transition (PT). PT is caused by calcium induced opening of the large pore at the mitochondrial inner membrane. Molecular composition of the PT pore is not completely understood as is subject of hot debates. Here I will discuss current understanding of the mechanisms of PT pore formation and present our resent experimental data of the electrophysiological recordings of PT pore activity in mitochondrial inner membrane from wild-type and mutant cells lacking putative components of the PT. Further, I will present recordings of the channel activity of the purified and fully synthetic components of the PT reconstituted into artificial planar lipid bilayers. Altogether our experiments suggest that PT can occur by different mechanisms that involve such mitochondrial proteins as C-subunit of the ATP synthase and Adenine Nucleotide Translocator. Existence of several pathways for PT helps to resolve long standing controversies regarding its exact molecular composition.

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