seems to be the protein that holds Tim22p and Tim18p. On other hand, the channel activity of TIM22 is greatest while the complex is intact. When disassembled, the detection frequency parallels the precise levels of Tim22p and Tim18p but is independent of Tim54p. In addition, overexpression of either Tim22p or Tim18p does not correlate with an increase in channel's frequency, despite it was described that reconstituted Tim22p alone forms pores. Taken together, these results imply that Tim18p is the putative receptor for internal signal peptides that trigger the activity of the TIM22 channel, formed by Tim22p.

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16P.3 Mitochondrial potassium ion channels from embryonic hippocampal neurons

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Recently, it has been shown that potassium transport, *via* ion channels, through the mitochondrial inner membrane can trigger neuroprotection. Until now five different mitochondrial potassium channels have been reported: ATP-regulated (mitoK_{ATP}), large-conductance calcium activated (mitoBK_{Ca}), intermediate conductance calcium-activated ($K_{Ca}3.1$), voltage-dependent (mitoKv1.3) and two-pore domain potassium channel (TASK-3). Our data provides evidence for the presence of mitoBK_{Ca} channels in the inner mitochondrial membrane of the rat hippocampus. The channel conductance calculated based on current-voltage relations was equal to 289 pS. The activity of the channel decreased at the low calcium concentration. The effect was reversed after application of NS1619, an activator of the BK type channels. Additionally, channel activity was blocked by paxilline (inhibitor of the BK type channels). Moreover we demonstrated that the probability of opening this channel is increased after the application of the arachidonic acid. We also identified a novel channel by patch-clamp, which has current–voltage characteristics similar to the rectifying channels. The channel conductance was equal to 60 pS. Patch-clamp studies showed that this channel is not sensitive to the known activators and inhibitors of the mitochondrial potassium channels. This channel was sensitive to the changes in the intracellular pH, and also regulated by the arachidonic acid.

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16P.4 Potassium and chloride channel activities from potato *Solanum tuberosum* tuber mitochondria

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Single channel activities were measured after reconstitution of the inner membranes from potato tuber mitochondria into planar lipid bilayers. After incorporation, in gradient 50/450 mM KCl (*cis*/*trans*), we found the ATP-regulated potassium channel with mean conductance of 164 pS, which the channel activity was inhibited by 1 mM Mg/ATP. Another detected one was the large-conductance potassium channel, which revealed the characteristic features for mammalian BK_{Ca} channels, i.e. the channel conductance was equal to 312 pS and was blocked by 200–400 nM iberiotoxin (IbTx) but did not exert the sensitivity on calcium. Furthermore, we observed also the chloride channel activity that was inhibited by 200 μM DIDS. The mean channel conductance of 117 pS and kinetic behavior were similar to that of those characterized for mitochondrial chloride channels, for example 108 pS. Finally, the high-conductance channel slightly selective for cations with an average conductance of 908 pS was observed. After addition of DIDS the channel activity was completely and irreversibly inhibited. Analysis of the electrophysiological specificity of this channel indicates its dissimilarity.

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16P.5 The kinetic and molecular mechanisms of the Zn^{2+}-activated mitochondrial swelling

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It is known that Zn^{2+} activates the mitochondrial (Mit) permeability transition that accompanied with opening the permeability transition pore and the Mit swelling. The latter was frequently leading to the cell damage and death. Nevertheless the characteristics of the Zn^{2+}-induced Mit swelling remain elusive. The aim of this research was to study the kinetic and molecular mechanisms of the Mit swelling stimulated by Zn^{2+}. The experiments were performed on the isolated Mit obtained from the rat liver by differential centrifugation. Mit swelling was measured spectrophotometrically and analyzed by: (i) maximal rate; (ii) lag-time; (iii) peak amplitude. We showed, that Zn^{2+} (0.1–7.0 μM) activated Mit swelling. The increase of extra-Mit concentration of Zn^{2+} caused decrease in the lag-time, but did not induce significant changes in the amplitude of the Zn^{2+}-induced Mit swelling. The dependence of the rate of Mit swelling on extra-Mit concentration of Zn^{2+} was bell-shaped (parabolic) with the peak magnitude equal to 3.0 μM of extra-Mit Zn^{2+}. V_{max} and $K_{0.5}$ of the ascending branch of the parabola were 0.283 $A_{540}/min \times mg$ of protein and 0.219 μM accordingly. We also found, that cyclosporin A (10 μM, 1 min) completely inhibited the Zn^{2+} (0.1, 3.0 μM)-induced Mit swelling. In the presence of dithiothreitol (1 mM, 1 min) the maximal rate, lag-time and the peak amplitude of the Zn^{2+} (0.1, 3.0 μM)-induced Mit swelling were 65 ± 12 ($p \leq 0.01$) and $31 \pm 5\%$ ($p \leq 0.001$), 233 ± 54 ($p \leq 0.01$) and $371 \pm 95\%$ ($p \leq 0.05$), and 94 ± 8 ($p > 0.05$) and $93 \pm 8\%$ ($p > 0.05$) correspondingly. In the presence of 2-ethoxy-1-ethoxycarbonyl-1,2-dihydroquinoline (1 mM, 1 min) the maximal rate, lag-time and the peak amplitude of the Zn^{2+} (0.1, 3.0 μM)-induced Mit swelling were 126 ± 16 ($p \leq 0.01$) and $72 \pm 13\%$ ($p \leq 0.05$), 34 ± 8 ($p \leq 0.05$) and $39 \pm 11\%$ ($p \leq 0.05$), and 132 ± 10 ($p \leq 0.01$) and $108 \pm 8\%$ ($p \leq 0.05$), correspondingly. In the presence of CGP-37157 (1 μM, 1 min) the maximal rate, lag-time and the peak amplitude of the Zn^{2+} (0.1, 3.0 μM)-induced Mit swelling were 121 ± 5 ($p > 0.05$) and $74 \pm 6\%$ ($p \leq 0.01$), 84 ± 21 ($p > 0.05$) and $76 \pm 15\%$ ($p \leq 0.05$), and 84 ± 12 ($p \leq 0.05$) and $87 \pm 7\%$ ($p \leq 0.01$), correspondingly. Thus, our results demonstrated that Zn^{2+} stimulates Mit swelling. This process is mediated by opening the permeability transition pore and regulated by the Mit SH-, COOH-groups and the Na^+/Ca^{2+}-exchanger.

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