membrane damage) or are we dealing with a new proton transporter was open. In this work, TCP-C15 was shown to slowly incorporate into membrane. Therefore, by long incubation (10–20 min) we showed an authentic uncoupling effect (stimulation of mitochondrial respiration) with nanomolar concentrations of this compound. With the help of a new experimental method, it was shown that the mitotrophic compound of SkQ set, which in reductive conditions in the mitochondrion transforms into the hydroquinone form, shows uncoupling effect at nanomolar concentrations.

References

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3P.6 Identification and characterization of functional residues in a multi-subunit type Na\(^+\)/H\(^+\) antiporter Mrp complex from alkaliphilic Bacillus pseudofirmus OF4
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Mrp antiporters catalyze secondary Na\(^+\)/(Li\(^+\))/H\(^+\) antiport and/or K\(^+\)/H\(^+\) antiport that is physiologically important in diverse bacteria. Mrp is unique among antiporters in requiring all six or seven hydrophobic gene products (MrpA to MrpG) of the mpr operon for antiport activity. The MrpA, D and C proteins of the MrpA-B-C-D module have sequence similarity with Complex I subunits whereas the MrpE-F-G module is Mrp-specific [1,2]. A panel of site-direct mutants in 28 conserved or specific motif-related amino acid residues from an alkaliphile Mrp Na\(^+\)/H\(^+\) antiporter was constructed. Each mutant transporter was expressed in antiporter-deficient Escherichia coli strain KNabc and the mutations were classified into 7 groups based on assays of growth/sodium-resistance, antiport properties, Mrp subunit levels, and formation of monomeric and dimeric Mrp complexes that are the active forms [1]. The analyses identified charged residues that are important for antiport activity and that are conserved across the large Mrp subunits of the MrpA-D module, MrpA, MrpD, as well as membrane-bound subunits (NuoLMN) of complex I. These included MrpA-K223, K299 and MrpD-K219 as well as acidic residues that had been identified in Bacillus subtilis Mrp [3]. This study also extended evidence for a key role for MrpE of the MrpE-F-G module. MrpE is required for normal membrane levels of other Mrp proteins and normal complex formation. Conversely, some mutations in the MrpA-D module affected membrane levels of MrpE. Residues that are required for formation of the monomeric form or both forms of hetero-oligomeric Mrp complexes were identified for the first time. A mutation of Proline81 in MrpG produced a novel Mrp that supported sodium-resistance but lacked antiport activity. While a pair of tyrosines and a VFF motif with acidic residues that had been identified in Bacillus subtilis Mrp [3]. This study also extended evidence for a key role for MrpE of the MrpE-F-G module. MrpE is required for normal membrane levels of other Mrp proteins and normal complex formation. Conversely, some mutations in the MrpA-D module affected membrane levels of MrpE. Residues that are required for formation of the monomeric form or both forms of hetero-oligomeric Mrp complexes were identified for the first time. A mutation of Proline81 in MrpG produced a novel Mrp that supported sodium-resistance but lacked antiport activity. While a pair of tyrosines and a VFF motif with acidic residues that had been identified in Bacillus subtilis Mrp [3].

References

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3P.7 Effect of cardiolipin on the iron uptake of F\(_{1}\)F\(_{0}\)ATP synthase in heart mitochondria
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Excessive iron is known to amplify ROS by Fenton and Haber-Weiss reaction, with subsequent damage of proteins, lipids and DNA of mitochondria. As one of the components affected by iron, cardiolipin, a tetra-acyl phospholipid, is crucial for oxidative phosphorylation of mitochondria. Recently, we showed that iron uptake was