associated with ATP hydrolytic activity in mitochondria. Since the activity of F_1F_OATP synthase is modulated by cardiolipin, we supposed that the cardiolipin might as well regulate the iron uptake in mitochondria. To study this postulation, proteoliposome reconstituted with iron overloaded enzyme was prepared. In the absence of cardiolipin but with asolectin only, iron uptake was stimulated more by ATP (1 mM) than ADP, whereas in the presence of cardiolipin, iron uptake was augmented higher by ADP (0.1 mM) than ATP. However, this ADP stimulation was diminished with augmenting the concentration of inorganic phosphate. Interestingly proteoliposome with iron overloaded enzyme exhibited parallel uptake of calcium and iron by ADP in the absence of cardiolipin. However, in the presence of cardiolipin, uptake pattern was changed as import of iron accompanies export of calcium and vice versa. This result suggests that cardiolipin may support iron uptake for iron overloaded enzyme by modulating molecular structure of enzyme.

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3P.8 Proton/ion co-transport by protein M2 of influenza virus A

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The M2 proton channel of influenza virus A was reconstituted into liposomes using our improved detergent-removal methodology (see Sindra Peterson Årsköld's poster). This yielded tight unilamellar vesicles of reproducibly high quality and unidirectional orientation of M2, with the N-terminal out. Using the novel, membrane-impermeable pH sensor Glu3 (Leiding *et al.*, 2009, *Anal. Biochem.* 388: 296-305), we monitored intravesicular pH and thus proton flux through M2. The observed flux was amantadine-sensitive, verifying that it was indeed channel-mediated. We present the proton flux recorded through M2 under a number of conditions: under a pH gradient in the absence and presence of valinomycin, under K^+(in)/Na^+(out) concentration gradients at varying pH, and under K^+(out)/Na^+(in) concentration gradients. We also show that while acidic reconstitution conditions inhibit M2 irreversibly, lowering the pH after reconstitution at pH 7 has a fundamentally different effect. Our results point towards M2 being a co-transporter rather than a pure proton channel: it can transport Na^+ along with H^+, it can transport Na^+ and K^+ in exchange for H^+, and it can transport H^+ in both directions. While not disputing the strong preference M2 shows for protons, our results explain how M2 acidifies the viral interior without building up an electrical counterpotential, and unifies a number of apparently contradictory results in the literature.

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3P.9 Studying the mechanisms of RNA translocation into mitochondria

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A growing field of evidence confirms that a large number of human diseases, such as mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS) or myoclonic epilepsy with ragged red fibers (MERRF), arise from mutations in tRNA genes coded by the mitochondrial DNA [1]. An understanding of the fine mechanisms of tRNA import from cytosol could help us to improve delivery of "therapeutic" RNA molecules into mitochondria to cure the negative effects of these mutations at the molecular level. It has been demonstrated that the import of $tRNA^{Lys}$ into mitochondria of the yeast *Saccharomyces cervisiae* needs ATP as energy source and the electrochemical membrane potential $\Delta\psi$ [2] as a driving force for its transmembrane movement. It has also been shown that this $tRNA^{Lys}$ has to be aminoacylated in the cytosol by the lysyl-tRNA-synthetase (KRS) and that its import requires two cytosolic targeting factors—the enolase-2, an enzyme of the glycolytic pathway, and the cytosolic precursor of the mitochondrial lysyl-tRNA-synthetase (preMSK) [3,4]. Little is still known about the molecular mechanisms involved in the translocation of the $tRNA^{Lys}$ across mitochondrial membranes, and proteins implicated in its import are not identified yet. Since import of $tRNA^{Lys}$ requires the preMSK, their co-import through the pre-protein import machinery may be suggested. However, analysis of yeast strains, carrying deletions of non-essential genes coding for proteins of the pre-protein import machinery, revealed that none of these mutations had an effect on $tRNA^{Lys}$ import. With the help of Northwestern analysis, combined with tandem mass spectrometry (MALDI TOF), several proteins, among which mitochondrial porin, TOM40, adenine nucleotide translocator, HSP60 and subunit 2 of bc1 complex were identified as potential binders of $tRNA^{Lys}$ upon its import into mitochondria, and their possible impact on import was studied in comparison for wild type and Δ*POR1* yeast strain.

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3P.10 Visualization of cargo dynamics in COPII vesicle formation on artificial planar lipid membrane

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Intracellular vesicular transport in eukaryotic cell is composed of three steps; selected cargo proteins are loaded into 50–100 nm diameter membrane vesicles, cargo loaded vesicle bud from the surface of intracellular organelles and fuse with the membrane of the recipient organelles. Thereby, the membrane components of transport vesicles and cargo proteins are transported. These transport vesicles are coated with a protein complex called "coat protein", their binding is controlled by low molecular weight GTPase [1]. Coat protein complex II (COPII) vesicles are coated with the COPII coat proteins, namely, Sec23/24p and Sec13/31p and a low-molecular-weight GTPase Sar1p. The formation of the COPII vesicles starts when Sar1p, which is a low molecular weight GTPase, is converted from a GDP form (inactive form) to a GTP form (active form) by the guanine nucleotide exchange factor (GEF) Sec12p, which is present on the endoplasmic reticulum. COPII vesicle formation can be reconstituted *in vitro* by using these 5 protein complexes [2,3]. So, we try to