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Abstracts

S1 Respiratory Chain and Photosystems

Lectures

1L.1 Electron transfer routes in cyanobacterial thylakoid membranes — Impact on biohydrogen production

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Optimization of electron transfer from water to biohydrogen production in a model organism *Synechocystis* sp. PCC 6083 involves several steps. PSII function can be improved by introducing a proper PSII reaction center D1 protein (encoded by the *psbA* gene family). To this end, an expression of a specific *psbA* gene that encodes a D1′ protein was detected under anaerobic conditions. We have also demonstrated a novel and crucial function for Flavodiiron (FDP) proteins Flv2 and Flv4 in photoprotection of PSII. The rate of accumulation of *flv2* and *flv4* transcripts upon shift of cells from high to low CO_2 is strongly dependent on light intensity. Characterization of FDP inactivation mutants revealed a specific decline in PSII centers and impaired translation of the D1 protein in Δflv2 and Δflv4 when grown at air level CO_2 whereas at high CO_2 the FPDs were dispensable. Δ*flv2* and Δ*flv4* were also more susceptible to high light induced inhibition of PSII than WT or Δ*flv1* and Δ*flv3*. Of the four flavodiiron proteins (Flv1–4) in Synechocystis 6803, a physiological function of Flv1 and Flv3 is in the Mehler reaction. Up to 30% of electrons derived from water by PSII may be directed to molecular oxygen *via* Flv1 and Flv3, and thus this route might seriously compete for electrons with the hydrogenase. Besides FDPs, the multiple NDH-1 complexes in cyanobacterial thylakoid membranes have a crucial role in electron transfer reactions, particularly in cyclic electron transfer around PSI, in respiratory electron transfer and in carbon concentrating mechanisms. Moreover, interplay between the FDPs and NDH-1 complexes is demonstrated to occur in electron transfer reactions.

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1L.2 Structural and functional insights into mitochondrial complex I

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The molecular mechanism how in complex I (NADH:ubiquinone oxidoreductase) proton translocation is linked to electron transfer is still unknown. Using the strictly aerobic yeast *Yarrowia lipolytica* as a powerful genetic system to study mitochondrial complex I, we have explored the ubiquinone reducing catalytic core of complex I by extensive site-directed mutagenesis. This functionally central region of complex I is located at the interface between the 49 kDa and the PSST subunit of the peripheral arm, where iron–sulfur cluster N2 serves as the immediate reductant of ubiquinone. We have located the likely entry pathway for ubiquinone leading to a conserved tyrosine located next to cluster N2 and investigated the role of the isoprenoid side chain for the interaction of ubiquinone with complex I. We have also mapped domains interacting with representatives of the different classes of hydrophobic complex I inhibitors. New evidence on the location of the ubiquinone binding pocket within complex I and the path leading to the site where the hydrophobic substrate is reduced will be discussed. We propose that long range conformational changes drive proton pumping of complex I through a two-state stabilization change mechanism involving distinct binding modes of charged ubiquinone intermediates.

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1L.3 High resolution structure of the electron input side of the respiratory complex I

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The respiratory complex I couples the electron transfer from NADH to ubiquinone with the translocation of protons across the membrane. The three-dimensional structure of the peripheral arm of *Thermus thermophilus* complex I was recently determined at a resolution of 3.3 Å, revealing the putative electron transfer pathway. The mode of interaction of nicotinamide nucleotides with the complex is important to understand because it was proposed that nucleotide binding results in conformational rearrangements essential for energy conservation by coupling redox processes to active proton pumping. In order to study the interaction and binding of nucleotides and inhibitors to the NADH binding site of complex I, we have heterologously overproduced and crystallized the electron input module of the enzyme from the hyperthermophilic bacterium *Aquifex aeolicus*, consisting of subunits NuoE and NuoF, in *E. coli*. The preparation contains the NADH binding site and the FMN as well as the binuclear Fe/S cluster N1a and the tetranuclear cluster N3. The structures of a native form, a form with bound nucleotides, and forms with several inhibitors were solved at resolutions up to 1.9 Å, revealing the molecular details of the binding and interaction modes of the coenzyme. Cluster N1a was proposed to be implicated in an intricate mechanism to minimize the generation of reactive oxygen species: After one electron is passed from the reduced FMN cofactor to the tetranuclear cluster N3 and further down the

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