bition of oxidized form of SDH by malonate (malonate was added before quinone) activates respiration rate. Double inhibitor titration method showed that in presence of DTD inhibition of SDH is accompanied by shift of the limiting stage of Q-cycle from i-center to o-center. According to suggested model the electron fluxes from SDH and DTD compete with each other in i-center of Q-cycle resulting in super reduction of i-center. Thus partial inhibition of the one of this fluxes yields oxidation of i-center and leads to increasing of respiration rate.

doi:10.1016/j.bbabio.2008.05.386

S13.44 Role of THE NapGH menaquinol dehydrogenase complex in *Wolinella succinogenes* nitrate respiration

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Many membrane-integral quinone-reactive enzyme complexes that are part of both eukaryotic and prokaryotic respiratory electron transport chains contain one or more haem *b* molecules. In recent years, however, a variety of novel proteins devoid of haem *b* emerged that are proposed to fulfil a similar function in anaerobic respiratory systems of various bacteria, e.g. members of the *c*-type cytochrome family NapC/NrfH and iron–sulfur proteins such as NapH. The *napH* gene is frequently present in gene clusters encoding components of the bacterial periplasmic nitrate reductase system. It is predicted to contain four transmembrane segments and to form a quinol oxidising complex with another iron–sulfur protein, NapG. We show here that NapH and NapG of the nitrate-respiring ε-proteobacterium *Wolinella succinogenes* indeed form a membrane-bound complex that mediates electron transfer from menaquinol to nitrate. The NapG subunit is located at the periplasmic side of the membrane where it acts as an electron transfer adapter protein that specifically donates electrons to the nitrate reductase NapA. A NapH homologue, NosH, is also able to form a functional complex with NapG. Deletion of either *napH* or *napG* almost abolished growth by nitrate respiration. The possible function of the essential cytoplasmic poly-cysteine clusters of NapH in the bioenergetics of nitrate respiration and/or in redox-driven enzyme maturation is discussed.

doi:10.1016/j.bbabio.2008.05.387

S13.45 *Chlamydomonas reinhardtii* mitoproteome adaptation in response to inactivation of the energy-dissipating alternative oxidase 1 by RNA interference

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The mitochondrial alternative oxidase (AOX) is an ubiquinol-oxygen oxidoreductase which catalyses ubiquinol oxidation by molecular oxygen. Thus AOX competes for electrons with the cytochrome pathway, generating an electron partitioning and decreases the oxidative phosphorylation yield. AOX from the unicellular green alga *Chlamydomonas reinhardtii* is encoded by two genes, the AOX1 gene being much more transcribed than AOX2. In addition, the expression of the AOX1 gene is down-regulated by ammonium and stimulated by nitrate. In this work, we performed a comparative proteomics approach (2D-DIGE) to study the effects of the inactivation of AOX1 by RNA interference on the mitochondrial proteome of *Chlamydomonas reinhardtii* cultivated on nitrate. Our results indicate that 88 protein spots are statistically up or down-regulated in our experimental conditions. Interestingly, observed up and down-regulations were related to proteins involved in protection against ROS and RNS. Moreover, other important enzymes of the main mitochondrial metabolic pathways (Krebs cycle, amino-acid metabolism and several subunits of the mitochondrial respiratory chain complexes) were also regulated indicating the important impact of the alternative oxidase expression in oxidative stress defence as well as in metabolic turnover.

doi:10.1016/j.bbabio.2008.05.388

(S14) Mitochondria and ageing symposium lecture abstracts

S14/1 Cardiolipin as an oxidative target in cardiac mitochondria in the aged rat

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The aged heart sustains greater injury during ischemia (ISC) and reperfusion (REP) compared to the adult heart. In the Fischer 344 (F344) rat, aging decreases oxidative phosphorylation and complex III activity increasing the production of reactive oxygen species in interfibrillar mitochondria (IFM) located among the myofibrils. In the isolated, perfused 24 month old elderly F344 rat heart 25 min. of stop-flow ISC causes additional damage to complex III, further decreasing the rate of OXPHOS. We did not observe further progressive mitochondrial damage during REP. We next asked if ISC or REP increased oxidative damage within mitochondria of the aged heart. Cardiolipin (CL) is a phospholipid unique to mitochondria consisting predominantly of four linoleic acid residues (C18:2). Following ISC and REP in the aged heart, there is a new CL species containing three oxygen atoms added to one linoleic residue. ISC alone was sufficient to generate this new oxidized molecular species of CL. Based upon oxidative damage to CL, complex III activity, and oxidative phosphorylation, mitochondrial damage thus occurs in the aged heart mainly during ISC, rather than during REP. Mitochondrial damage during ischemia sets the stage for mitochondrial-driven cardiomyocyte injury during reperfusion in the aged heart.

Supported by: NIH POI AG15885.

doi:10.1016/j.bbabio.2008.05.389

S14/2 Mitochondrial volume regulation by a redox switch on the adenine nucleotide translocase

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Mitochondrial volume regulation plays an important role in the control of oxidative phosphorylation and protection against cell injury;