impairment of over expression of the longest tau isoform (hTau40) and mutant tau (TauP301L) on mitochondrial function in SH-SY5Y cells. Additionally, we tested the influence of inhibitors of the mitochondrial respiratory chain complexes, as in human P301L FTDP17 brains the level of complex V is reduced. We found that over expression of human wtTau or TauP301L leads to mitochondrial dysfunction in our cell model. Already under basal conditions the ATP level and the metabolic activity are significant decreased in TauP301L cells compared to hTau40 cells. Additional stress with the complex inhibitors results in a dose-dependent loss of metabolic activity, reduced ATP levels and depolarized MMP in all three cell types.

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S13.40 Characterization of the redox centres in arsenite oxidase

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Arsenic is a toxic element, present in water predominantly as As^{V} and As^{III}. Microorganisms strongly affect its speciation in the environment, for example by converting As^{III} to As^{V} via their arsenite oxidase, thereby detoxifying their growth medium. The crystal structure of the *Alcaligenes faecalis* enzyme revealed a linear arrangement of its three redox centres, suggesting a linear electron transfer from Molybdenum to a [3Fe–4S] and on to a Rieske-type [2Fe–2S] centre. An electrochemical study determined an $n=2$ redox transition for the Mo atom and E_m values for the three centres rendering the proposed electron transfer thermodynamically unfavourable at pH 6. We have recently addressed this question using i) another enzyme, that from NT-26, ii) another experimental approach, *i.e.* EPR and iii) a pH-screening between 6 and 9.5. As already established for mesophilic cytochrome *bc*-Rieskes, the E_m of the Rieske centre remains constant (at 220±10 mV) up to pH 8 and decreases above pH8 with a slope of −80 mV/pH. In this pH range, the redox state of the [3Fe–4S] centre was unstable even at cryogenic temperatures. Titrating the [3Fe–4S] centre was only possible in the presence of sulphite (shown to be an inhibitor of arsenite oxidase) yielding $E_{m6}=+260$ mV, *i.e.* close to that reported for *Alcaligenes*. We interpret these observations as reflecting a redox re-equilibration between the [3Fe–4S]- and the Mo-centres. The results are discussed to propose an electron flow model through the enzyme.

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S13.41 Bamboo mitochondrial energy metabolic pathways in *Bambusa oldhamii* and *Phyllostachys edulis* during rapid shooting stage

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The energy-conversing and energy-dissipating systems were studied in young bamboo shoot mitochondria isolated from summer bamboo *Bambusa oldhamii* and from winter bamboo *Phyllostachys edulis*. The mitochondrial respiration rates of NADH, succinate or malate oxidation were measured at 15, 28, and 42 °C. Temperature raised from 15 °C to 28 °C, the increased respiration rate of *P. edulis* were higher than that in *B. oldhamii*, whereas the temperature raised from 28 °C to 42 °C, the increased respiration rate of *P. edulis* were lower than those of *B. oldhamii*. The calculated Q_{10} values of *B. oldhamii* at intervals of 15~28 °C and 28–42 °C were about 1.9–2.4 and different from those of *P. edulis*. Moreover, the membrane thermostability of *B. oldhamii* mitochondria was suggested to be lower than of *P. edulis* as the critical temperature of *B. oldhamii* was about 20 °C and that of *P. edulis* mitochondria about 25 °C. Furthermore, alternative oxidase (AOX), plant uncoupling mitochondrial protein (PUMP), and plant mitochondrial potassium channel ($PmitoK_{ATP}$) were investigated. In the presence of SHAM, an AOX inhibitor, more than 50% of the respiration rate was inhibited in *B. oldhamii* whereas only a small portion of 6.9% respiration in *P. edulis* was inhibited. In the presence of PUMP activator linoleic acid, mitochondrial membrane potential was collapsed about 85% in *P. edulis* and 30% in *B. oldhamii*. It showed that the activity of $PmitoK_{ATP}$ in *P. edulis* mitochondria was probably 2 folds of that in *B. oldhamii* as a rapid swelling occurred in *P. edulis* with addition of KCl whereas a mild swelling occurred in *B. oldhamii*. The results may support that *P. edulis* adapting to chilling environment was correlated to higher energy-dissipating capacity than *B. oldhamii* favoring to moderate environment.

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S13.42 Alternative oxidase 1a in *Arabidopsis thaliana* is required for normal stress response

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The aim of this study was to determine the function of the alternative oxidase in *Arabidopsis*. Treatment of *alternative oxidase 1a* mutant plants (*aox1a*) with moderate light and drought resulted in changes is respiration, photosynthesis, reactive oxygen species and metabolites that were absent or much less pronounced in Col-0 plants. These changes were accompanied by drastic changes in the transcriptome during the stress treatment, affecting genes encoding proteins involved in a wide variety of processes in various locations in the cell. Functional analysis of the *AOX1a* promoter revealed that it contain *cis*-acting regulatory elements previously identified to be involved in stress responses in a variety of genes, in particular stress responses mediated by abscisic acid. These results indicate that AOX1a is required for a normal stress response in *Arabidopsis* and its regulation interacts with mainstream stress signalling pathways.

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S13.43 Inhibitors of succinate dehydrogenase (SDH) and complex III promote respiration of liver mitochondria under conditions of functioning DT-Diaphorase (DTD)

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Mitochondrial complex III interacts with three dehydrogenises. Two of them complex I and DTD oxidize NADH, SDH oxidizes succinate. Both NADH and succinate are synthesized in Krebs cycle. The competition of DTD and SDH in course of their interaction with bc_1-complex was investigated. All measurements were carried out with malate in the capacity of respiration substrate. Complex I was inhibited by rotenone. Duroquinone or CoQ_0 was taken as a second substrate of DTD. On the one hand we found out that low concentrations of Q-cycle o-center inhibitor myxothiazol under conditions of functioning DTD initiate small stimulation of respiration. And on the other hand inhi-

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.

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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.

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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.

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(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.

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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;