

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 significantly 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

S. Duval, W. Nitschke, B. Schoepp-Cothenet

CNRS Institut de Biologie Structurale et Microbiologie, Marseille France

E-mail: duval@ibsm.cnrs-mrs.fr

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

Lee-Feng Chien, Hui-Ping Chen, Yuan-Chin Wu

Department of Life Sciences, College of Life Sciences, National Chung Hsing University, Taichung 40227, Taiwan

E-mail: lfchien@dragon.nchu.edu.tw

The energy-converting 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

James Whelan

ARC Centre of Excellence in Plant Energy Biology, MCS Building M316 University of Western Australia, 35 Stirling Highway, Crawley 6009, Western Australia, Australia

E-mail: seamus@cyllene.uwa.edu.au

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 in 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)

Konstantin A. Motovilov, Lev S. Yaguzhinsky

Belozersky Institute of Physico-Chemical Biology, Lomonosov Moscow State University, Moscow, Russia

E-mail: oven24@gmail.com

Mitochondrial complex III interacts with three dehydrogenases. 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 bc₁-complex was investigated. All measurements were carried out with malate in the capacity of respiration substrate. Complex I was inhibited by rotenone. Duroquinone or CoQ₀ 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-