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FEBS openbio

Volume 9 Supplement 1 July 2019

POSTERS

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Abstracts submitted to the 44th FEBS Congress, taking place in Krakow, Poland from 6th to 11th July 2019, and accepted by the Congress Organizing Committee are published in this Supplement of *FEBS Open Bio*. Late-breaking abstracts are not included in this issue.

About these abstracts

Abstracts submitted to the Congress are **not peer-reviewed**. In addition, abstracts are published as submitted and are **not copyedited** prior to publication.

We are unable to make **corrections of any kind** to the abstracts once they are published.

Indexing

Abstracts published in *FEBS Open Bio* Supplement for the 44th FEBS Congress will be included individually in the Conference Proceedings Citation Index published by Web of Science.

How to cite these abstracts

AuthorOne, A., AuthorTwo, B. (2019). Abstract title. FEBS Open Bio, 9: Abstract number*. doi:10.1002/2211-5463.12675

P-07-006
The role of heat shock protein 72 in regulation of skeletal muscle–adipose tissue crosstalk

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Recent evidence has identified skeletal muscle as a secretory organ and released cytokines have been classified as myokines. As production of many proteins in skeletal muscle is dependent upon contraction, physical activity leads to an altered myokine response. Some studies show that physical training increases expression of brown adipocyte marker uncoupling protein 1 (UCP1) in subcutaneous adipose tissue, what can be mediated by released myokines. Moreover, heat shock protein 72 (HSP72) is elevated during physical training and have been identified as important regulator of muscles metabolism. Therefore, the aim of the study was to investigate the effect of HSP72 on skeletal muscle secretome and browning of white adipose tissue. Herein it was shown that overexpression of HSP72 both *in vitro* in C2C12 myotubes, and *in vivo* in skeletal muscle upregulates 5'AMP-activated protein kinase, energy homeostasis regulator in cell, that is naturally activated by exercises. Muscle cells with overexpression of HSP72 are characterized by increased β-oxidation rate and mitochondrial biogenesis, decreased lipid content, as well as increased activity of enzymes involved in oxidative metabolism. Overexpression of HSP72 in C2C12 myotubes increases gene expression of interleukin 6, which is known myokine that increases content of UCP1 mRNA in white adipose tissue. Furthermore, conditioned media from C2C12 cells overexpressing HSP72 increase browning of white adipocytes. Overall, so far the study showed that HSP72 regulates skeletal muscle metabolism by increasing oxidative capacity of the cells and also suggests that overexpression of HSP72 in skeletal muscle potentially can affect myokine profile and browning of adipose tissue. Acknowledgements: This research was supported by the Foundation for Polish Science, grant TEAM/2010-5/2 and National Science Centre (NCN), grant NCN UMO-2011/03/B/NZ3/00693 and UMO-2017/24/T/NZ4/00275.

P-07-007
Transcriptional regulation of plant mitochondrial biogenesis in abiotic stress

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For the proper biogenesis of plant mitochondria, coordinated regulation of mitochondrial and nuclear gene expression is expected. We studied such coordination at the transcriptional level in cauliflower (*Brassica oleracea* var. *botrytis*) mitochondria under acclimation and de-acclimation to cold, heat and drought. For that, whole transcriptome high-throughput sequencing on Illumina NovaSeq 6000 platform was employed. Key transcript alterations were further validated by reverse transcription-quantitative PCR (RT-qPCR). Some mitochondrial mRNAs (ex. *nad9*, *coxII*) were regulated inversely comparing to the protein level, suggesting more efficient use those transcripts for translation in cold acclimation and de-acclimation. Furthermore, we detected the lack of coordination of accumulation of messengers for subunits of the same protein complexes (ex. complex I and ATP synthase). However, changes in abundance of transcripts for various alternative oxidase (*AOX*) isoforms compensated unfavorable proteomic alterations in stress de-acclimation. *AOX* messengers were differentially accumulated depending on stress conditions and the extent of stress pre-acclimation. Variations of *AOX1a* transcripts suggested only partial utilization of mRNA pool in translation due to the altered availability of transcripts for translation, mRNA/ribosome interactions and miRNA action. Stress responding transcripts included inter alia mRNAs for some matrix enzymes and transcription factors (ex. from ETHYLENE RESPONSE FACTOR family). We detected downregulation of transcripts coding for enzymes of proline catabolism in cold and heat de-acclimation. De-acclimation was not always accompanied by return of transcript abundancies to the control level. In conclusion, some alterations suggest serious perturbations in mitochondrial biogenesis lasting after stress de-acclimation. Presented results extend our knowledge on new candidates participating in abiotic stress response of plant mitochondria at RNA level.

P-07-008
Venturicidin-type macrolides as FoF1-ATPase inhibitors: structure-activity relationships

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Venturicidins (also known as aabomycins) are quite rare glycosylated 20-membered macrolide antibiotics isolated from *Streptomyces* species in the 1960s. It was described as perspective for agricultural use against plant pathogenic fungi. Antifungal activity of venturicidins is linked with inhibition action on FoF1-ATP synthase localized in mitochondrial membrane. Three compounds of this type, known irumamycin and X-14952B, as well as new isoirumamycin, were isolated from culture *Streptomyces* sp. INA-Ac-5812. Structure elucidation and identification were done by using extensive spectroscopic analysis: 1D and 2D NMR, HRESI-MS, UV and IR. Stereo configuration of irumamycin and X-14952B hemiketal (C3-C7) and tail (C23-C24) fragments was deduced from NMR data (ROESY and HSQMBC). Structure of isoirumamycin differs from known congeners in the size of macrocyclic core (18-membered) according to key HMBC and ROESY correlations. Cytotoxicity of all isolated compounds was in the 5–15 μM range for various tumor cell lines and human postnatal fibroblasts. Despite this, the antifungal activity was strongly dependent on structural variations. Maximum inhibition activity was detected for irumamycin; activity decreased in the following order: irumamycin > X-14952B > isoirumamycin. Obtained data can contribute to the rational design of FoF1-ATPase inhibitors based on macrocyclic compounds.

P-07-009
MCT-mediated lactic acid import regulates mitochondrial oxidative phosphorylation via the PDK/PDH axis

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Aerobic glycolysis and mitochondrial dysfunction are key metabolic features of cancer cells, but their interplay during cancer development remains unclear. We previously reported that human hepatoma cells with mitochondrial defects exhibit lactate dehydrogenase subunit B (LDHB) downexpression. Here we