Institute of Physiology,
Pathophysiology and Biophysics

vetmeduni vienna

XXX Workshop

New insights in mitochondrial research

Tuesday, 28th February 2023

Seminar room, Vetmeduni Vienna

10:00 -10:05	**Taraneh Beikbaghban** *Vetmeduni Vienna*	Opening
Session I:		
10:05 – 10:35 (20+10)	**Felix Sternberg** *Vetmeduni, Vienna*	"Regional differences in the transcriptional response of the murine brain towards fasting, ketogenic interventions, and variations in fatty acid composition."
10:35 – 11:05 (20+10)	**Jila Nasirzade** *Vetmeduni, Vienna*	"Investigation of UCP2 expression in mouse bone marrow-derived macrophages under basal condition and nutritional shortage"
Short break		
11:20 – 12:05 (35+10)	**Karin Nowikovsky & Ashita Vadassery Vivekanandan** *Vetmeduni, Vienna*	"The language of LETM1 and mitochondrial K+ homeostasis in development, NAD+ metabolism and circadian rhythms"
12:05 – 12:25 (10+10)	**Maria Andreeva** *Vetmeduni, Vienna*	"Analysis of the protein expression of Knock-in UCP2-ALFA-Tag in THP1 cell line"
Lunch break		
14:00 – 14:30 (20+10)	**Taraneh Beikbaghban** *Vetmeduni, Vienna*	"Role of Un-Coupling Protein 2 (UCP2) in metabolic flexibility of human leukemic cancer cells"
Session II:		
14:30-15:00 (20+10)	**Olga Jovanović** *Vetmeduni, Vienna*	"Action of long acyl chain aldehydes on biological membrane and transporters"
15:00 – 15:30 (20+10)	**Kristina Žuna** *Vetmeduni, Vienna*	"2-oxoglutarate/malate carrier enhances the fatty acid–mediated proton transport in lipid bilayer membranes"
Short break		
15:45 – 16:15 (20+10)	**Giorgia Roticiani** *Vetmeduni Vienna*	"Mechanism of the proton transport activation in UCP1"
16:15 – 16:45 (20+10)	**Sanja Škulj** *Vetmeduni, Vienna*	"Molecular Dynamics Simulations of Membrane Systems and Transmembrane Protein UCP1"
Dinner		

Guest Lecturer presentation on 03.03.2023, 15:00

Redox signaling, redox metabolic shuttles and changes of mitochondrial cristae upon insulin secretion

Petr Ježek, Department of Mitochondrial Physiology, Institute of Physiology of the Czech Academy of Sciences, Prague, Czech Republic

In pancreatic β-cells, NADPH-oxidase 4- (NOX4-) mediated cytosolic H2O2 release represents redox signaling essential for glucose-stimulated insulin secretion (GSIS) [1]. At least three metabolic shuttles activated upon GSIS enable that the matrix superoxide/H2O2 release declines, since equivalents of unmade NADH are transferred to the cytosolic NADPH [2]. For IS stimulated by fatty acids (FAs) and branched-chain ketoacids (BCKAs) [1], β-oxidation creates superoxide/H2O2, which provides mt-PM redox signaling from mitochondria to plasma-membrane which enables i) closing ATP-sensitive K+-channels (KATP), together with elevated ATP; and for FASIS also ii) activation of mitochondrial (H2O2-) redox-activated phospholipase iPLA2γ/PNPLA8, which cleaves FAs from mitochondrial phospholipids and supplies metabotropic GPR40 receptors, amplifying insulin secretion [3]. These mechanism were investigated in pancreatic islets (PIs) isolated from wt and PNPLA8 knockout (KO) mice by various techniques. Redox signaling was inferred from FASIS blockage by 10 nM SkQ1, a mitochondrial matrix-targeted antioxidant, and by overexpressed cytosolic catalase. Moreover, bulky cristae existing in low glucose in INS1E cells and PIs became narrow upon GSIS and IS stimulated by BCKAs [4]. Speculatively, redox signal might participate/reflect cristae morphology changes.

[1] Plecitá et al. Diabetes 69, 1341–1354 (2020).

[2] Plecitá et al. Antioxid. Redox Signal. 33, 789–815 (2020).

[3] Ježek J. et al. Antioxid. Redox Signal. 23, 958–972 (2015).

[4] Ježek J. et al. Antioxid. Redox Signal. (2023) review accepted

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