

Colloquia in Cellular Signaling

Venue: Medical University Vienna, Center for Physiology and Pharmacology,
Institute of Physiology, Schwarzschanerstrasse 17, 1090 Vienna, "gr. HS. Physiology".
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Friday 05.05.2017 12:00 Host: Helmut Kubista

Mitochondrial calcium signalling in health and disease

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Abstract:

The over-arching goal of work in my lab is to illuminate the place of mitochondria in cell physiology and their roles in cell health and disease. We are fascinated by the ways that cell signalling pathways and mitochondria interact. In this seminar, I will focus on the relationship between mitochondrial function and cellular calcium signalling in health and in disease. Calcium signals are fundamental to cell physiology, playing essential roles in contraction, motility, secretion, fertilisation and many other functions. Calcium signalling pathways shape mitochondrial function to meet the bioenergetic demands of the cell, while changes in mitochondrial function impact cell calcium signaling, physiology and function. I will introduce basic principles and discuss some recent studies that reveal mechanisms that define mitochondrial calcium handling in health and consequences of dysfunction in disease.

Calcium signaling as a mediator of cell energy demand and a trigger to cell death.
Bhosale G et al, *Ann NY Acad Sci.* 2015 Sep; 1350:107-16.

The regulation of neuronal mitochondrial metabolism by calcium.
Llorente-Folch I et al, *J Physiol.* 2015 Aug 15; 593 (16):3447-62.

Loss-of-function mutations in MICU1 cause a brain and muscle disorder linked to primary alterations in mitochondrial calcium signaling.
Logan CV et al, *Nat Genet.* 2014 Feb; 46 (2):188-93.

Ischaemic accumulation of succinate controls reperfusion injury through mitochondrial ROS.
Chouchani ET et al, *Nature.* 2014 Nov 20; 515 (7527):431-5.

Pathological consequences of MICU1 mutations on mitochondrial calcium signalling and bioenergetics.
Bhosale G et al, *Biochim Biophys Acta.* 2017 Jan 26. pii: S0167-4889 (17) 30023-X.