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# Abstracts
# Plenary Lectures

## PL. 1

### Pathophysiology of the mitochondrial permeability transition

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The mitochondrial permeability transition (PT) is a $Ca^{2+}$-dependent increase of mitochondrial inner membrane permeability to solutes with molecular masses up to about 1500 Da [1]. Its occurrence is always accompanied by depolarization, while onset of matrix swelling, depletion of matrix pyridine nucleotides, outer membrane rupture and release of intermembrane proteins including cytochrome *c* depend on the open time. The PT is due to the reversible opening of a high-conductance, voltage-dependent channel in the inner mitochondrial membrane, the PT pore (PTP). In spite of many efforts, its molecular identity remains unknown (reviewed in [2]). In this lecture I shall cover the essential aspects of PTP pathophysiology, with specific emphasis on the role of matrix cyclophilin D [3]; the mechanism of action of cyclosporin A [4]; the modulation by the proton electrochemical gradient [5] and redox effectors [6]; and the consequences of PTP opening as a key to understanding its role in cell dysfunction and death. From this analysis the PTP emerges as a viable target for therapeutic intervention in cancer [7] and degenerative diseases [8].

### References

[1] Hunter D.R *et-al., J. Biol. Chem.* **251:** 1976 5069–5077.
[2] Bernardi P *et-al.,* 2006 *FEBS J.* **273** 2077–2099.
[3] Giorgio V *et-al.,* 2010 *Biochim. Biophys. Acta* doi:10.1016/j.bbabio.2009.12.006.
[4] Basso E. *et-al.,* 2008 *J. Biol. Chem.* **283:** 26307–26311.
[5] Bernardi P, 1992, *J. Biol. Chem.* **267:** 8834–8839.
[6] Petronilli V. *et-al.,*1994, J. *Biol. Chem.* **269:** 16638–16642.
[7] Rasola A. *et-al.,* 2010 *FEBS Lett.* doi:10.1016/j.febslet.2010.02.022.
[8] Merlini L *et-al.,* 2008 *Proc. Natl. Acad. Sci. U.S.A.* **105** 5225–5229.

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## PL. 2

### UCP1 and mitochondrial uncoupling

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Uncoupling protein 1 (UCP1) remains as the prototypic — and possibly only — physiologically relevant uncoupling protein (although several closely related proteins do exist). Despite its unchallenged uncoupling function, agreement has still not been reached as to how the uncoupling is accomplished, with hypotheses ranging from it actually being a proton translocator to it being a fatty acid anion transporter. In several respects, recent developments have changed the classical views concerning UCP1 gene expression and function. Whereas expression of the UCP1 gene was earlier considered to be fully under adrenergic control, it is now clear that also agents working through PPAR-gamma can in themselves induce UCP1 gene expression. Similarly, while it was earlier accepted that a cell expressing UCP1 was a brown adipocyte, it has become clear that UCP1 is expressed in adipocytes ("brite adipocytes") that do not possess all the properties of classical brown adipocytes. Although an absence of UCP1 has been accepted to lead to an absence of nonshivering thermogenesis, it was thought until recently that diet-induced thermogenesis was unaffected. It is now clear that also diet-induced thermogenesis is fully UCP1-dependent — and the absence of UCP1 causes or aggravates obesity. Finally, whereas it has been the accepted view that UCP1 and brown adipose tissue are only found and active in newborn humans, it is now evident that a significant fraction of adult humans also possess brown adipose tissue and that UCP1 activity thus may be of significance for metabolic efficiency in adult humans.

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## PL. 3

### Mitochondrial stress signaling

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Mitochondria are crucial for a wide spectrum of cellular processes. Their involvement not only encompasses the energy metabolism, but also apoptosis, cell growth, differentiation, movement, signaling and proliferation. Thus, any malfunction of mitochondria can have profound consequences for cell physiology. Severe mitochondrial malfunctions, leading to changes in $\Delta\Psi$, are termed the mitochondrial stress and trigger magnitude of cellular stress responses. Cellular calcium metabolism and mitochondrial dynamics (balance of fusion/fission processes) are modified by the mitochondrial stress.