



Katedry genetiky a biochémie PriF UK
a občianske združenie *NATURA*



Vás pozývajú na 92. prednášku v rámci Kuželových seminárov:

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Peroxisome biogenesis: studies in yeast

ktorá sa uskutoční **18. októbra 2013** (piatok) o **14:00**

v miestnosti **CH1-222** Prírodovedeckej fakulty UK

Hostiteľ: Juraj Gregáň

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Andreas Hartig received his PhD. in Chemistry from the University of Vienna in 1979. Postdoctoral years at the NIH, Bethesda, USA, at Rutgers University, Piscataway, USA, and in Vienna at the Department of Biochemistry with the late Prof. Dr. Helmut Ruis, stimulated his interest in cellular and molecular biology of yeast. Since 1986 he is member of the faculty at the Department of Biochemistry. The main research interests of Dr. Hartig are the biogenesis of peroxisomes and the translocation of proteins and metabolites across membranes.



Eukaryotic cells contain intracellular to separate metabolic pathways. This spatial separation ensures optimal flux of metabolic intermediates and increases the efficiency of the metabolism. Peroxisomes are highly versatile organelles and essential for life. They participate in many metabolic processes, most notably the degradation of fatty acids and the glyoxylate cycle. Synthesis of organelles and their degradation has to be tightly regulated in agreement with the metabolic status of the cell. Accordingly, peroxisomes need to be maintained in sufficient number to ensure metabolic homeostasis. A network of interacting proteins guarantees the biogenesis of functional peroxisomes, the transport of peroxisomal matrix proteins across the organellar membrane, and the control of size, shape and number of these compartments. Dispensable peroxisomes are degraded in a process called pexophagy. Employing yeast as model system Hartig group's aim is to elucidate the molecular mechanisms leading to new peroxisomes either through proliferation of already existing ones or via a *de novo* biogenesis pathway through fission from the endoplasmic reticulum (ER). Currently, the main interest of the group is focused on the mechanism of the *de novo* biogenesis initiated at the ER. Proteins exclusively involved in the biogenesis of peroxisomes are called peroxins (Pex-proteins). Among these the Pex11 protein is a membrane elongation factor, and in yeast, we showed that this protein acts only on already existing peroxisomes leading to proliferation. Two distantly related yeast proteins, Pex25p and Pex27p, play similar roles at the peroxisomal membrane and, in addition, participate in the *de novo* biogenesis. The Pex3 protein is the only peroxin demonstrated to accumulate under certain conditions at the ER and later be transferred to peroxisomes. Distinct vesicles emanating from the ER may slowly mature into peroxisomes or may fuse with each other or already existing peroxisomes to form mature organelles. The priming event at the ER, the proteins involved and the molecular mechanism are so far unknown, and will be the focus of our future work.

Selected publications:

Huber, A., Koch, J., Kragler, F., Brocard, C, Hartig, A. (2012). A subtle interplay between three Pex11 proteins shapes *de novo* formation and fission of peroxisomes. *Traffic* 1:157-167.

Taschner, A., Weber, C., Buzet, A., Hartmann, R.K., Hartig, A., Rossmannith, W. (2012). Nuclear RNase P of *Trypanosoma brucei*: a single protein in place of the multicomponent RNA-protein complex. *Cell Rep* 2:19-25.

Koch, J., Pranjic, K., Huber, A., Ellinger, A., Hartig, A., Kragler, F., Brocard, C. (2010). *PEX11* family members are membrane elongation factors that coordinate peroxisome proliferation and maintenance. *J. Cell. Sci.* 123: 3389-3400.