



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



Vás pozývajú na **108. prednášku** v rámci Kuželových seminárov:

**Tomáš Doležal**

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## **WHY IS OUR IMMUNE SYSTEM SELFISH?**

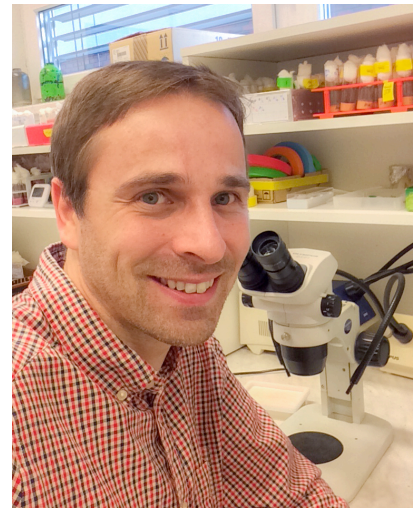
ktorá sa uskutoční **10. novembra 2017** (piatok) o **14:00**

v miestnosti CH1-222 Prírodovedeckej fakulty UK

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2015 associate professor Faculty of Science, University of South Bohemia in České Budějovice  
2003 PhD. University of South Bohemia in České Budějovice  
1999 M.Sc., Charles University in Prague  
Since 2016 Head of the Department of molecular biology and genetics  
2012-2014 visiting assistant professor, Stanford University School of Medicine, USA  
2003-2005 University of California, Irvine, USA (Peter Bryant lab)  
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### Lecture annotation:

Immune cells must either rapidly proliferate or at least activate a plethora of cellular actions upon their activation and therefore they significantly change their metabolism. This metabolic change is associated with increased glycolysis and glucose consumption, resembling thus Warburg effect observed in tumor cells and rapidly proliferating cells. Glucose must be rerouted from other tissues towards immune system to supply immune cells with enough energy during immune response. Recently, a theoretical concept of selfish immune system was proposed, in which insulin resistance caused by pro-inflammatory cytokines was seen as a way of immune system to usurp energy from the rest of the organism. Although this might be a vital strategy during acute response, it may become pathologic during chronic inflammation. Experimental evidence supporting this concept has been missing. We use simpler and genetically-well tractable model organism, the fruit fly *Drosophila melanogaster*, to investigate regulation of systemic metabolism during immune response. We induce the immune response either in fly larva by parasitoid wasps which inject their eggs into the larvae or by bacterial infection in the adult flies. We have found that immune cells behave selfishly releasing signals which suppress consumption of energy by other tissues. Such selfishness of immune system during its activation is crucial for the effectivity of immune response. When we block the selfish signals, the host resistance drastically drops. However, immune cells also limit their own selfishness by producing negative feedback regulators and this is important to prevent an excessive release of energy from reserves which are limited.

### Selected publications:

- Dolezal T** (2015) - Adenosine: a selfish-immunity signal? **Oncotarget** - Immunology and Microbiology Section 6 (32), 32307-32308.
- Bajgar A, Kucerova K, Jonatova L, Tomcala A, Schneedorferova I, Okrouhlik J, **Dolezal T** (2015) Extracellular Adenosine Mediates a Systemic Metabolic Switch during Immune Response. **PLoS Biol** 13(4): e1002135.
- Novakova M and **Dolezal T** (2011). Expression of *Drosophila* adenosine deaminase in immune cells during inflammatory response. **PLoS ONE** 6(3): e17741.
- Fenckova M, Hobizalova R, Fric Z, **Dolezal T** (2011). Functional characterization of ecto-5'-nucleotidases and apyrases in *Drosophila melanogaster*. **Insect Biochem Mol Biol** 41(12): 956-967.
- Dolezal T**, Kucerova K, Neuhold J, Bryant PJ (2010). Casein kinase I epsilon somatic mutations found in breast cancer cause overgrowth in *Drosophila*. **Int J Dev Biol** 54: 1419 – 1424.
- Dolezal T**, Dolezelova E, Zurovec M, Bryant PJ (2005). A Role for Adenosine Deaminase in *Drosophila* Larval Development. **PLoS Biol** 3(7): e201.
- Dolezal T**, Gazi M, Zurovec M, Bryant PJ (2003). Genetic analysis of the ADGF multigene family by homologous recombination and gene conversion in *Drosophila*. **Genetics** 165: 653-666.
- Zurovec M, **Dolezal T**, Gazi M, Pavlova E, Bryant PJ (2002). Adenosine deaminase-related growth factors stimulate cell proliferation in *Drosophila* by depleting extracellular adenosine. **PNAS** 99(7): 4403-4408.