Radislav Sedláček

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LABORATORY OF

TRANSGENIC MODELS OF DISEASES

transgenesis, disease models, embryogenesis, proteases and their inhibitors

In the picture:
1. Shohag Bhargava | **2.** Veronika Grešáková | **3.** Jan Procházka | **4.** Petr Kašpárek | **5.** Jan Dvořák | **6.** Jolana Turečková | **7.** Olga Žbodáková | **8.** Ivan Štěpánek | **9.** Katarzyna Szczerkowska | **10.** Petr Kašpar PhD | **11.** Renata Turečková | **12.** Marie Indrová | **13.** Eliška Selingerová | **14.** Veronika Iatsuk | **15.** Olena Sapega | **16. Radislav Sedláček** | **17.** Michela Luciano

The laboratory has three subgroups that are interlinked by technologies used and mouse models studied to reveal gene functions in the complexity of the whole organism.
One subgroup focuses on proteases in physiology and disease, particularly on matrix metalloproteinases (MMP), a disintegrin and metalloproteinase (ADAM), and kallikreins (Klk). MMP and Klk proteases are partly responsible for controlling extracellular matrix-cell interactions affecting cell differentiation, survival, migration, and other processes. ADAM proteinases (ADAM 10, ADAM17) release ligands and their receptors from the cell surface, thus guiding bioavailability of many important regulatory molecules. We have created a number of mutants for Klk genes on the background of SPINK5, the major inhibitor of serine proteases, to reveal their complex network in the skin, especially in the development of the Netherton syndrome.
Ubiquitylation-mediated processes. Using mutant mouse models we are addressing the role of several new uncharacterized ubiquitin ligases in health and disease. A major focus of these studies is to understand the role of ubiquitylation in regulating the intestinal barrier function and to characterize links with human inflammatory bowel disease.
Early embryonic development, epigenetics, and meiosis. Using unique mouse models for the FAM208a gene we address the molecular mechanisms influencing cell fate decisions and the embryonic development, stem cell pluripotency. Other mouse models were generated to study the role of Fragile X mental retardation syndrome 1 neighbour gene (FMR1nb), especially in female reproduction during oocyte meiosis and maturation.

Selected recent papers:

Kasparek P, Ileninova Z, Haneckova R, Kanchev I, Jenickova I, Sedlacek R: A viable mouse model for Netherton syndrome based on mosaic inactivation of the Spink5 gene. **Biol Chem. 2016** Dec 1;397(12):1287-1292.

Brauer R, Tureckova J, Kanchev I, Khoylou M, Skarda J, Prochazka J, Spoutil F, Beck I M, Zbodakova O, Kasparek P, Korinek V, Chalupsky K, Karhu T, Herzig KH, Hajduch M, Gregor M, Sedlacek R: MMP-19 deficiency causes aggravation of colitis due to defects in innate immune cell function. **Mucosal Immunol. 2015** Nov 11. doi: 10.1038/mi.2015.117.

Prochazka J, Prochazkova M, Du W, Spoutil F, Tureckova J, Hoch R, Shimogori T, Sedlacek R, Rubenstein J L, Wittmann T, Klein O D: Migration of Founder Epithelial Cells Drives Proper Molar Tooth Positioning and Morphogenesis. **Dev Cell. 2015** Dec 21;35(6):713-24.


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