27th European Drosophila Research Conference

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ABSTRACT BOOK (do not print)

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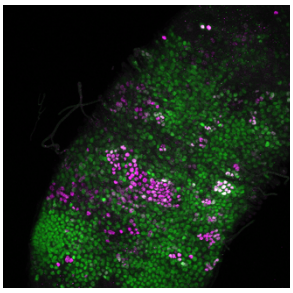
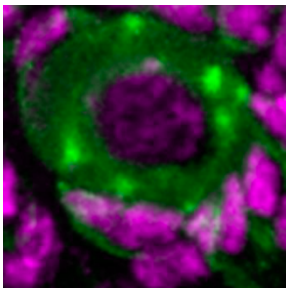
Cancer models workshop





Drosophila **research has a long history of contributing to our understanding of the mechanisms of cancer initiation and propagation. These studies range from molecular mechanisms of DNA repair to polarity and neoplastic growth. The goal of the workshop will be to bring together scientists interested in these very diverse aspects of modelling cancer using *Drosophila* and to create cross-discipline dialogue spanning distinct cellular contexts from stem cells to epithelial cells.**

Organisers: Renata Basto & Allison Bardin (Institut Curie, Paris, France)

13h-13h15: Allison Bardin
"Nucleotide sharing through gap junctions buffers replication stress".
Department of Genetics and Developmental Biology, Institut Curie, Paris, France.

13:15-13h30: Manon Budzyk
"Gen nuclease is essential for the proliferation of non-programmed polyploid cells".
Basto lab, Cell Biology and Cancer department, CNRS and Institut Curie, Paris, France.

13h30-13h45: Brian Calvi
"Unscheduled endoreplication impairs the growth and function of cells and tissues".
Indiana University, Bloomington, USA.

13h45-14h: Wu-Min Den
"Sex dimorphic and systemic regulation of tumor growth by Upd2-JAK/STAT signaling**".**
Department of Biochemistry and Molecular Biology, Tulane University School of Medicine, New Orleans, USA.

14h-14h15: Kaustuv Ghosh
"Chromosomal Instability-induced Cell Invasion through Caspase-driven DNA Damage".
Milan lab, Institute for Research in Biomedicine (IRB Barcelona), The Barcelona Institute of Science and Technology, Barcelona, Spain.

14h15-14h30: Tatsushi Igaki
"Non-cell autonomous tumor progression by unfolded protein response"
Graduate School of Biostudies, Kyoto University, Kyoto, Japan.

14h30-14h45: Anne-Marie Martinez
"Transient loss of Polycomb components induces an epigenetic cancer fate".
Institute of Human Genetics, Montpellier, Montpellier, France

14:45-15h: Marta Mira-Osuna
"Contribution of septate junction components to apical and basal extrusion of protumoral cells"
Le Borgne lab, Institute of Gentics & Development of Rennes, Rennes, France.

15h-15h15: Mirka Uhlirova
"Immunosurveillance, understanding the crosstalk between immune cells and epithelial tumors".
Institute for Genetics Cologne Excellence Cluster on Cellular Stress Responses in Aging-Associated Diseases (CECAD), University of Cologne, Cologne, Germany.

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In vivo dissection of behaviorally-relevant neuronal relaxin signaling pathway

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Relaxin-like peptides (relaxins) belong to the insulin-like family of peptides. Instead of acting via receptor tyrosine kinases as insulin and insulin-like growth factors do, relaxins typically exert their biological effects by binding to G protein-coupled receptors. The relaxin signaling pathway is found both in invertebrates and vertebrates, including humans, and plays important roles in the reproductive, circulatory, skeletal, renal, and nervous systems. The therapeutic potential of human relaxins, for instance, are being explored due to their vasodilator, antifibrotic, and antidepressant properties. However, the biology of relaxin receptors is not fully understood. Here, our primary aim is to identify new conserved regulatory mechanisms of relaxin receptor activity. For this, we are using a relaxin-receptor-dependent Drosophila phenotype to screen for new relaxin pathway components and regulators. In Drosophila, lack of the relaxin-like peptide, Dilp8, or its neuronal G protein-coupled receptor, Lgr3, compromises the formation of the puparium, a sort of cocoon generated by the larva from its own external cuticle to protect itself from desiccation and predators during metamorphosis. During puparium formation, or pupariation, the cuticle is actively remodeled by stereotyped muscle contractions and then hardened enzymatically. Reduced Lgr3 receptor signaling in six ventral nerve cord interneurons leads to abnormally shaped puparia, a phenotype that can be easily, cheaply, and quickly scored by eye at the same time that it is highly informative about the integrity of the Dilp8-Lgr3 relaxin signaling pathway, the presence and integrity of the critical Lgr3-positive interneurons, and the complex behavior they mediate. Taking advantage of this, we are performing a large cell-type-specific RNAi screen in vivo using a UAS-inducible RNAi stock collection for genes expressed in the central nervous system. In this presentation we will provide an update on the _˜1500 genes screened up to now and an initial dissection of the candidate hits identified.

Keywords: Relaxin, Drosophila, genetic screen, pupariation, Lgr3 receptor

*Speaker