WELCOME TO THE SECOND UNIGE

• **June 6th 2019** •

• Grand Auditorium, École de Physique •

POST-DOC ASSOCIATION OF THE UNIVERSITY OF GENEVA

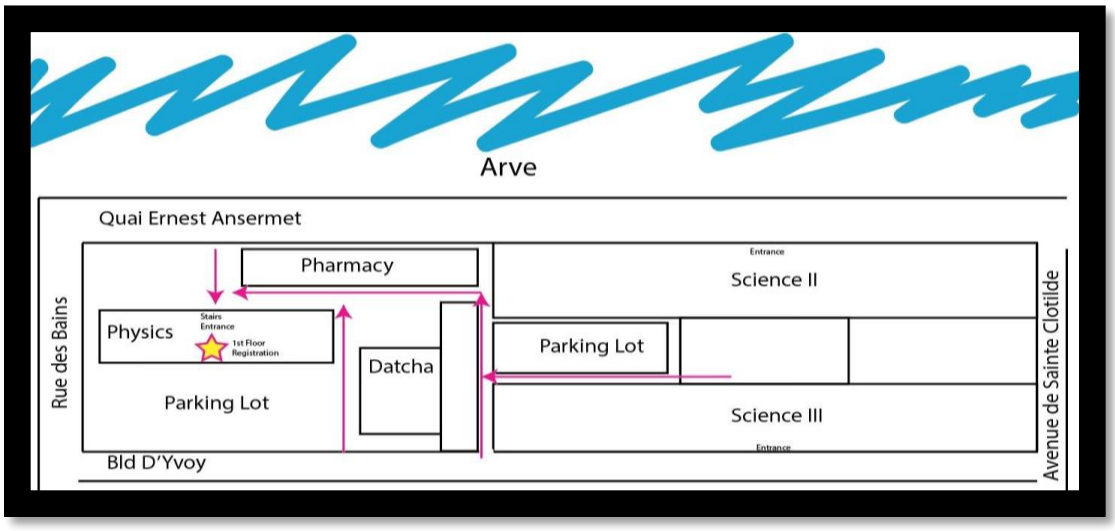
APDUG

CONFERENCE BOOKLET

How to get there ?

The conference will take place at “l’Ecole de Physique” in the “Grand Auditorium”.

Registration will be held starting from 8h30 at the main entrance in the first floor.



How to connect to Internet ?

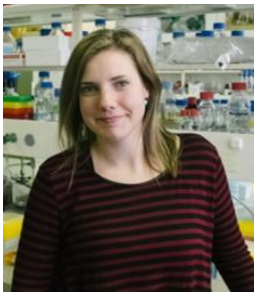
1) Connect your mobile device or computer to the SSID WIFI: guest-unige (make sure your browser accepts pop-up windows and javascript to run).
2) In your browser, open any URL or HTTP page (e.g. the University website: www.unige.ch). You will be directed to an e-portal where you can register and authenticate.
3) On the following page, click on the "connect" button.
   When you first log in, you will need to register on the system. To do so, please enter your mobile number and click the "Register" button.
4) You will receive a code via SMS, you need to enter on the next screen and click the "Login" button.
   You will be automatically redirected to the e-University website. Your access code is valid for 6 months, during which you will not need to request a new code.

Organizing Committee

Caterina Tomba - Manuela Leonardelli - Adai Colom - Priya Ramakrishna - Daniel Dilg

Aurelien Roux Lab - Roman Ulm Lab - Aurelien Roux Lab - Marie Barberon Lab - David Shore Lab

APDU Committee



Noemi Jiménez Rojo - Rita Mateus - Federico Miozzo - Clément Immarigeon - Alicia Daeden - Zena Hadjivasiliou - Marie Pireyre

– PROGRAM –

POSTDOC DAY

9h – 9h10 *Opening Remarks*

Part I - Who are your colleagues?

9h10 – 9h50 *Postdoc scientific talks (10min each)*

Valeria Cagno *(Microbiology and Molecular Medicine): "Broad spectrum virucidal strategies"*

Jinsheng Zhu *(Plant Biology): "How plant sense phosphate limitation? Inositol pyrophosphates are signaling molecules"*

Joachim Moser von Filseck *(Biochemistry): "Helical ESCRT-III heteropolymers with different binding interfaces and bending stiffnesses stabilise spiralling-tubular membrane deformation"*

Chris Rands *(Genetic Medicine and Development): "Functional and junk DNA in human and microbial genome sequences"*

9h50 – 10h30 ***Keynote speaker – BIOLOGY***

Giovanni D'Angelo – EPFL

"Making the plasma membrane: Lipid landscape transitions and metabolic biases in the biosynthetic pathway"

10h30 – 10h45 *Short break*

10h45 – 11h35 *Postdoc scientific talks (10min each)*

Clément Immarigeon *(Genetics and Evolution): "Size is not what matters - lesson from a tiny peptide involved in sperm competition"*

Michael Plank *(Molecular Biology): "Chemical genetics of AGC-kinases reveals shared functions of Ypk1 and Protein Kinase A"*

Liam Scarratt *(Inorganic and Analytical Chemistry): "What Makes SLIPS Slippery? Measuring effective slip on lubricated surfaces with colloidal probe AFM"*

Noemi Veraldi *(Pathology and immunology): "Heparan sulfate implication in genetic human disorders: the case of Multiple Osteochondromas"*
Julien Soudet *(Cell biology): "Fine chromatin-driven mechanism of transcription interference by antisense noncoding transcription"*

11h34 – 12h20 ***Keynote speaker – PHYSICS***
Magdalena Kowalska – CERN
"Polarized radioactive nuclei: from fundamental and nuclear physics, via material science, to biology and medicine"

12h30 – 14h00 *Poster Presentation and Llunch*

14h00 – 14h45 ***Keynote speaker – CHEMISTRY***
Olga Garcia Mancheño - Universität Münster
"Polarizing Catalytic Effects - in bonds, reactions and lifestyles"

14h45 – 15h15 *Postdoc scientific talks (10min each)*
Marine Laporte *(Cell biology): "Unravelling the molecular assembly of the centriole using expansion microscopy"*
Pietro Cacialli *(Pathology and immunology): "The endothelial niche detoxifies HSCs from ROS in the caudal hematopoietic tissue"*
Claudio Quilodran *(Genetics and Evolution): "Hybridization during density-dependent range expansion: European wildcats as a case study"*

15h15 – 15h30 *Short break*

Part II - Focus on your career:

15h30 – 15h35 ***LS_2 AM$_{2020}$*** *-* ***Elena Cardenal****, Scientific Officer*
PIs of Tomorrow, a first step to your professorship

15h35 – 15h45 ***Noemi Jimenez-Rojo and Rita Mateus****, APDU founders*

15h45 – 17h00 *Open discussion:*
Academic vs non-Academic career – possibilities and opportunities?
Monica Gotta *- Eccellenza Evaluation Committee Biology and Medicine*
Eliane Abou-Mansour *and* ***Alexander Waehry****: Service recherche UNIGE*

Mylène Docquier *(CMU, Geneva): iGE3 Genomics Platform Manager*
Helen Kimbell *(Frontiers, Lausanne): Journal Development Specialist*

17h - 20h *Best talk and Best poster prizes followed by Apéro*

Part I - Who are your colleagues?

Session 1 : 9h10-9h50

Valeria Cagno (Faculty of medicine - Department of Microbiology and Molecular Medicine): "Broad spectrum virucidal strategies"

Jinsheng Zhu (Plant Biology):
"How plant sense phosphate limitation? Inositol pyrophosphates are signaling molecules"

Joachim Moser von Filseck (Biochemistry):
"Helical ESCRT-III heteropolymers with different binding interfaces and bending stiffnesses stabilise spiralling-tubular membrane deformation"

Chris Rands (Genetic Medicine and Development):
"Functional and junk DNA in human and microbial genome sequences"

Broad spectrum virucidal strategies

Valeria Cagno, Caroline Tapparel University of Geneva, Switzerland

Samuel Jones, Ozgun Kocabiyik, Francesco Stellacci, EPFL, Switzerland

Ronan Le Goffic, INRA, France

Viral infections kill millions yearly. Most antiviral drugs are virus-specific and available for a restricted number of viruses. Likewise, in the recent epidemics of Ebola and Zika, despite huge efforts, no specific antiviral could be developed in time. An ideal strategy to fight viral infections is to develop broad-spectrum antivirals that irreversibly inhibit viral infectivity. We have previously developed gold nanoparticles mimicking the viral attachment receptor heparan sulfate and showing both broad-spectrum and antiviral activity with a virucidal mechanism.

We next designed sulfonic acid (CDHS) or sialic acid (CDSA) decorated biocompatible cyclodextrins that mimic cell attachment-receptors widely used by viruses and evaluated their activity against a broad panel of viruses in cells, in human-derived tissues and in mouse models. CDHSs show broad-spectrum virucidal activity against different heparan sulfate dependent viruses (HSV, RSV, HIV, HCV, DENV, ZIKV) and CDSA against sialic acid dependent viruses (Influenza A, Influenza B). For both molecules, the activity is maintained ex vivo in human cervicovaginal histocultures infected by HSV-2 and in respiratory tissues infected with RSV or Flu and in vivo in mice infected with HSV-2 and Flu. Therefore, the design of nanomaterials mimicking attachment receptors can be a valuable strategy to develop broad-spectrum and virucidal antivirals effective in vitro, ex vivo and in vivo. The approach proved effective against heparan sulfates and sialic acid-dependent viruses and can be extended to target other viral receptors.

How plant sense phosphate limitation? Inositol pyrophosphates are signaling molecules.

Jinsheng Zhu[1], Kelvin Lau[1], Robert K. Harmel[2], Robert Puschmann[2], Larissa Broger[1], Amit K. Dutta[3], Henning Jessen[3], Ludwig A. Hothorn[4,#], Dorothea Fiedler[2], Michael Hothorn[1,]*

1. Structural Plant Biology Laboratory, Department of Botany and Plant Biology, University of Geneva, Switzerland.
2. Leibniz-Forschungsinstitut für Molekulare Pharmakologie, Berlin, Germany & Department of Chemistry, Humboldt Universität zu Berlin, Berlin, Germany
3. Institute of Organic Chemistry, Albertstrasse 21, 79104, Freiburg im Breisgau, Germany.
4. Institute of Biostatistics, Leibniz University, Hannover, Germany

Many eukaryotic proteins regulating phosphate (Pi) homeostasis contain SPX domains. We have previously shown that these domains act as cellular receptors for inositol pyrophosphate (PP-InsP) signaling molecules, suggesting that PP-InsPs may regulate Pi homeostasis. Here we report that simultaneous deletion of two diphosphoinositol pentakisphosphate kinases VIH1 and 2 in Arabidopsis impairs plant growth and leads to constitutive Pi starvation responses. We demonstrate that VIH1 and VIH2 are bifunctional cytosolic enzymes able to generate and break-down PP-InsPs. Point-mutants targeting the kinase and phosphatase active sites have opposing effects on plant Pi content and Pi starvation responses, while VIH1 and VIH2 protein levels remain constant in different Pi growth conditions. Enzymatic assays reveal that ATP-Mg2+ substrate levels can shift the relative kinase and phosphatase activities of full-length diphosphoinositol pentakisphosphate kinases. Deletion of phosphate starvation response transcription factors rescue vih1 vih2 mutant phenotypes, placing diphosphoinositol pentakisphosphate kinases and PP-InsPs in plant phosphate signal transduction cascades. We propose that VIH1 and VIH2 relay changes in cellular ATP concentration to changes in PP-InsP levels, allowing plants to maintain cellular Pi concentrations constant and to trigger Pi starvation responses.

Helical ESCRT-III heteropolymers with different binding interfaces and bending stiffnesses stabilise spiralling-tubular membrane deformation

Joachim Moser von Filseck[1], Luca Barberi[2], Adam Frost[3], Martin Lenz[2] and Aurélien Roux[1,4]

1. *Biochemistry Department, University of Geneva*
2. *LPTMS, CNRS & Université Paris-Sud*
3. *Department of Biochemistry and Biophysics, University of California San Francisco*
4. *NCCR Chemical Biology, University of Geneva*

ESCRT-III proteins are key players in cellular membrane remodelling, such as intraluminal vesicle formation, viral budding, cytokinesis and others. Reconstituting ESCRT-III-mediated membrane deformation and fission in vitro has not delivered reliable results so far. In a bottom-up approach, we were able to identify a minimal subset of recombinant yeast ESCRT-III proteins (Snf7, Vps24, Vps2) that are sufficient for deforming flat membranes into stable spiralling tubes. The three proteins heteropolymerise into filaments on an artificial lipid bilayer, and their lateral interaction leads to the formation of rigid filament clusters that have different membrane binding interfaces with regard to their helical axis. These rigid clusters can serve as a helical protein scaffold shaping the underlying membrane. Membranes shaped by such a scaffold will minimise their global curvature by limiting the deformed surface area. Together, protein scaffolding and the energy cost of membrane curvature explain why helical membrane tubes are thermodynamically stable at high membrane tension. This combination of experimental and theoretical work highlights the versatility in geometry and membrane binding of ESCRT-III polymers and the membrane reorganisation described could eventually play a key role in ESCRT-III-mediated membrane fission.

Functional and junk DNA in human and microbial genome sequences

Chris Rands [1,2]

1. University of Geneva and Swiss Institute of Bioinformatics
2. Formerly at the University of Oxford

Despite the deluge of genomic data being produced, identifying the functional roles, if any, of DNA sequences remains challenging. Nearly 99% of the human genome does not encode proteins and it remains unclear whether the bulk of these remaining noncoding sequences have important functions or if they represent 'junk DNA'. By comparing the genome sequences of different animal species, we identified genomic regions that have evolved unexpectedly slowly, a signature of natural selection upon functional sequence. Through this, we estimated that approximately 8% of the human genome is functional and we inferred how rapidly different types of functional sequence 'turn over' as they are gained and lost over time (Rands et al., 2014).

Bacterial genomes contain proportionally less noncoding DNA than human genomes, but they have other sources of potential junk DNA. In particular, up to 20% of bacterial genomes consists of integrated viral sequences, termed prophages, whose functional relevance for the bacteria are often unknown. We found rare examples of prophages harbouring antibiotic resistance genes in an under studied group of bacteria, showing that prophages sometimes carry functional sequences that are beneficial for their bacterial host (Rands et al., 2018; Rands et al., 2019).

Overall, we demonstrate through bioinformatics analyses that human and microbial genomes contain a mixture of functional and junk DNA, and we identify and characterise some important sequences, providing complementary evidence to many other computational and experimental studies investigating genome function.

Rands, C.M., Brüssow, H., and Zdobnov, E.M. (2019) bioRxiv 606087.

Rands, C.M., Meader, S., Ponting, C.P., and Lunter, G. (2014) PLoS Genetics 10: e1004525.

Rands, C.M., Starikova, E.V., Brüssow, H., Kriventseva, E.V., Govorun, V.M., and Zdobnov, E.M. (2018). Environmental Microbiology.

Session 2 : 10h45 -11h35

Clément Immarigeon (Genetics and Evolution):
"Size is not what matters - lesson from a tiny peptide involved in sperm competition"

Michael Plank (Molecular Biology):
"Chemical genetics of AGC-kinases reveals shared functions of Ypk1 and Protein Kinase A"

Liam Scarratt (Chemistry):
"What Makes SLIPS Slippery? Measuring effective slip on lubricated surfaces with colloidal probe AFM"

Noemi Veraldi (Pathology and immunology):
"Heparan sulfate implication in genetic human disorders: the case of Multiple Osteochondromas"

Julien Soudet (Cell biology):
"Fine chromatin-driven mechanism of transcription interference by antisense noncoding transcription"

Size is not what matters - lesson from a tiny peptide involved in sperm competition

Clément Immarigeon, Yohan Frei, Rob Maeda, François Karch

Sperm competition occurs in polygamous species of mammals, birds, reptiles, insects etc… This drove evolution of striking and surprising features by which sperm outperform its rivals. Drosophila for example has giant sperm tails coated with Sex Peptide (SP), a small protein secreted by accessory glands (AGs)– the functional analog of mammalian prostate. SP triggers Post Mating Responses (PMR) in females, an ensemble of behavioral and physiological changes beneficial to the male reproductive success. The large quantity of SP and its slow release from stored sperms allows long-term PMR (LT-PMR).

Several LT-PMR phenotypes such as decreased female receptivity and sperm competition have been linked to a few genes expressed in AGs' Secondary Cell (SC), such as Abd-B and the ncRNA MSA. However, the rarity of this cell type (4% of the accessory gland) made them hard to fully comprehend.

We performed SC-specific transcriptome and translatome analysis to better understand the function of these cells. Strikingly, a handful of non-essential genes account for the vast majority of the RNAs and proteins of these cells. Gene Ontology analysis shows large enrichment for genes involved in reproduction, maintenance of PMR in females, sperm competition, protein synthesis and secretion. SCs thus appear as factories of secreted proteins transferred to females via seminal fluid.

Surprisingly, the MSA RNA is highly translated despite being annotated as non-coding. Careful analysis of its sequence reveals a conserved short Open Reading Frame (sORF) that might encode a micro protein (miP). We generated miP-GFP knock-in and revealed the production of a 9 amino-acids peptide in SCs. In addition, miP mutant males display a sperm competition phenotype. This sORF demonstrates that size does not matter for biological relevance, and that so called "non-coding RNAs" should be considered as potential source of functional tiny proteins.

Chemical genetics of AGC-kinases reveals shared functions of Ypk1 and Protein Kinase A

Michael Plank (1), Mariya Perepelkina (1), Stefania Vaga (2), Jacques Saarbach (3), Xiaoming Zo (1), Steven Haesendonckx (1), Ruedi Aebersold (2), Robbie Loewith (1)

(1) Department of Molecular Biology, University of Geneva, CH-1211, Geneva, Switzerland
(2) Department of Biology, Institute of Molecular Systems Biology, ETH Zürich, CH-8093, Zürich, Switzerland
(3) Department of Organic Chemistry, University of Geneva, CH-1211, Geneva, Switzerland

Protein phosphorylation cascades play a central role in the regulation of cell growth in response to nutrient availability and other environmental factors. Three enzymes of the AGC-kinase family take center stage in this process in S. cerevisiae: Protein kinase A (PKA) and Sch9 mainly respond to nutrient availability while Ypk1 is believed to primarily sense properties of the cell membrane. Several of their targets and the wiring of their signalling pathways have been mapped out by genetic and classical molecular biology approaches. Additionally, changes in the phospho-proteome upon kinase deletion have been determined. However, while transcriptomics upon chemical inhibition of some of these enzymes has been performed, acute changes in the phospho-proteome have not been systematically assessed.

In this study we inhibited the three kinases individually as well as PKA and Sch9 in combination via a chemical genetics approach and evaluated changes in the phospho-proteome. Sites hypo-phosphorylated upon PKA and/or Sch9 inhibition were preferentially located in RRxS/T motifs suggesting that many are directly phosphorylated by these enzymes. Interestingly, when inhibiting Ypk1 we not only detected several downregulated sites in the previously reported RxRxxS/T consensus motif, but also sites in an RRxS/T (but not RxRxxS/T) context.

We site-specifically validated phosphorylation changes on previously reported PKA target neutral trehalase Nth1, which carries one RxRRxS, one RxRxxS and one RRxS site in its N-terminal tail. Each of these sites is hypo-phosphorylated upon Ypk1 inhibition and this is associated with reduced trehalase activity.

Globally, this phospho-proteomics dataset illustrates how three major growth-regulatory kinases co-ordinately regulate cell growth. It further shows that Ypk1 is closer to PKA and Sch9 in its target profile than previously appreciated and may perform functions pr

What Makes SLIPS Slippery? Measuring effective slip on lubricated surfaces with colloidal probe AFM

Liam Scarratt[1,2], Liwen Zhu[1], Chiara Neto[1]

1. School of Chemistry and University of Sydney Nano Institute, The University of Sydney, NSW 2006, Australia
2. Department of Inorganic and Analytical Chemistry, University of Geneva, 1205 Geneva, Switzerland

Slippery Liquid-Infused Porous Surfaces (SLIPS), inspired by the pitcher plant, possess self-cleaning and anti-fouling properties, with the potential for drag reduction.[1,2] They achieve these properties by using micro- and nano-scale topography to trap a chemically compatible lubricant, creating a customizable liquid interface.[3] Understanding the nanoscale forces that stabilise the thin lubricant film, and the ability of these films to reduce drag is crucial for their practical use under shear.[4] Recently it has been shown that smooth surfaces are capable of retaining a lubricant layer, and the mechanism for this lubricant retention is unclear. Using colloidal probe atomic force microscopy, the flow of simple viscous liquids was studied over smooth lubricated surfaces, with the aim to quantify effective interfacial slip. The minimum lubricant film thickness that has slippery properties was quantified and related to macroscopic measurements of roll-off angles of water droplets over a partially dewetted lubricant film.[5] Further studies have been performed on the drag reducing properties of stable lubricant films with thicknesses up to 800 nm. This work provides insight regarding liquid flow in confined geometries and on effective ways to minimise hydrodynamic drag.

References

[1] Epstein, A. K.; Wong, T. S.; Belisle, R. A.; Boggs, E. M.; Aizenberg, J., Proc. Natl. Acad. Sci. U.S.A. 109 (33) 2012, 13182-13187.

[2] Ware, C. S.; Smith-Palmer, T.; Peppou-Chapman, S.; Scarratt, L. R. J.; Humphries, E. M.; Balzer, D.; Neto, C., ACS Appl. Mater. Interfaces, 10 (4) 2017, 4173-4182.

[3] Scarratt, L. R. J.; Hoatson, B. S.; Wood, E. S.; Hawkett, B. S.; Neto, C., ACS Appl. Mater. Interfaces, 8 (10) 2016, 6743-6750.

[4] Wexler, J. S.; Jacobi, I.; Stone, H. A., Phys. Rev. Lett., 114 (16) 2015, 168301.

[5] Scarratt, L. R. J.; Zhu, L.; Neto, C., Langmuir, 35 (8) 2019, 2976-2982.

Heparan sulfate implication in genetic human disorders: the case of Multiple Osteochondromas

Noemi Veraldi [1], Alessandro Parra [2], Elena Urso [3], Cesare Cosentino [3], Manuela Locatelli [4] , Serena Corsini [4], , Elena Pedrini [4], Annamaria Naggi [3] , Antonella Bisio [3], Luca Sangiorgi [5]*

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2. IRCCS—Istituto Ortopedico Rizzoli, Bologna, Italy
3. Istituto di Ricerche Chimiche e Biochimiche G. Ronzoni, Milan, Italy;
4. Department of Medical Genetics and Rare Orthopaedic Diseases—IRCCS, Istituto Ortopedico Rizzoli, Bologna, Italy
5. Department of Medical Genetics and Rare Orthopaedic Diseases & CLIBI Laboratory—IRCCS, Istituto Ortopedico Rizzoli, Bologna, Italy

Heparan sulfate (HS) is a ubiquitously expressed glycosaminoglycan constituted by repeating units of a glucosamine linked to an uronic acid, either D-glucuronic or L-iduronic acids, the canonical major repeating unit being N-acetyl-glucosamine linked to D-glucuronic acid. Structural variability relies on the different sulfation pattern obtained by combining sulfation in N- and O-positions and it is proven to be different depending on tissue and cellular microenvironment. In cartilage, HS play a pivotal role by participating in gradient formation of morphogens thus directing and regulating the growth of chondrocytes. A genetic disorder affecting cartilage and known as Multiple Osteochondromas (MO) is characterized my mutations in the genes EXT1 and EXT2 which encode for two glycosyltransferases that catalyze HS polymerization. The resulting decreased or absent expression of HS alters proper signaling network thus resulting in the formation of cartilage-capped bony outgrowths called exostoses for which there is no treatment. Although the link between HSs and the pathology is clear, nothing is known on the structural alterations that prevent HS from undergoing its physiologic pathway. In this work, osteochondroma (OC), peripheral chondrosarcoma (PCS, malignant tumor), and healthy cartilaginous human samples were processed to structurally characterize and compare HS from pathologic and physiologic conditions, and to examine the phenotypic differences that arise in the presence of either EXT1 or EXT2 mutations. Although deeper investigation is still necessary, the approach here applied pointed out, for the first time, structural differences among OC, PCS, and healthy HS chains extracted from human cartilaginous excisions, and could help in understanding how the structural features of HS are modulated in the presence of pathological situations also involving different tissues.

Fine chromatin-driven mechanism of transcription interference by antisense noncoding transcription

Jatinder Kaur Gill, Andrea Maffioletti, Varinia García-Molinero, Françoise Stutz and Julien Soudet

Eukaryotic genomes are almost entirely scanned by RNA polymerase II (RNAPII). Consequently, the transcription of long noncoding RNAs (lncRNAs) often overlaps with coding gene promoters triggering potential gene repression through a poorly characterized mechanism of transcription interference. In this study, we propose a global model of chromatin-based transcription interference in Saccharomyces cerevisiae (S. cerevisiae). By using a noncoding transcription inducible strain, we analyzed the relationship between antisense elongation and coding sense repression, nucleosome occupancy and transcription-associated histone modifications using near-base pair resolution techniques. We show that antisense noncoding transcription leads to -1/+1 nucleosome H3K18/H4 deacetylation associated with H3K36 trimethylation increase (H3K36me3). This results in the loss of -1/+1 nucleosome interaction with the RSC chromatin remodeler and subsequent sliding into the Nucleosome-Depleted Region (NDR) hindering Pre-Initiation Complex (PIC) binding. Finally, we extend our model by showing that natural antisense noncoding transcription significantly represses around 20% of S. cerevisiae genes through this chromatin-based transcription interference mechanism.

Session 3 : 14h45-15h15

Marine Laporte (Cell biology):
"Unravelling the molecular assembly of the centriole using expansion microscopy"

Pietro Cacialli (Pathology and immunology):
"The endothelial niche detoxifies HSCs from ROS in the caudal hematopoietic tissue"

Claudio Quilodran (Genetics and Evolution):
"Hybridization during density-dependent range expansion: European wildcats as a case study"

Unravelling the molecular assembly of the centriole using expansion microscopy

Marine Laporte, Davide Gambarotto, Maeva Le Guennec, Nikolai Klena, Virginie Hamel, Paul Guichard.

Centrioles are conserved organelles that coordinate fundamental biological processes including cell division, cellular signalling and cell motility. Centrioles are characterized by a nine-fold radial arrangement of microtubule triplets (MTTs). Strong cohesion between these MTTs is critical to resist forces applied by ciliary beating and the mitotic spindle. Failure to maintain this cohesion causes centrioles to disintegrate, resulting in cellular dysfunction and disease. How centrioles resist such forces to maintain their structural integrity and which proteins are involved in this process is poorly understood. Using cryo-electron tomography, we demonstrate that MTTs are bound together by a circular inner scaffold that maintains MTT cohesion under compressive forces. Moreover, we resolve the 3D structure of this inner scaffold, revealing a large helical lattice that covers 70% of the entire centriolar length. We undertook to unveil which centriolar proteins compose this inner scaffold using our newly developed method of Ultrastructure Expansion Microscopy (U-ExM) that allows a nanometric mapping of proteins to structural elements. Importantly, we identified a subset of centriolar proteins that localize to the inner scaffold and are known to be important for centriole integrity, assembly and elongation. We demonstrate that these proteins localize precisely adjacent to the inner surface of the microtubule wall and span up to 85% of the centriolar length. Moreover, we found that all of these proteins form a complex in vivo. Altogether, these results provide novel fundamental insights into the conserved mechanisms of centriole assembly, function and integrity.

The endothelial niche detoxifies HSCs from ROS in the caudal hematopoietic tissue.

Pietro Cacialli 1, Julien Y. Bertrand 1

1. Department of Pathology and Immunology, School of Medicine, University of Geneva, Switzerland.

Hematopoietic stem cells (HSCs) are responsible for sustaining hematopoietic homeostasis. In the adult, it has been well established that their functions can be affected by reactive oxygen species (ROS) that are produced endogenously through cellular metabolism or after exposure to exogenous stress. An increase of ROS can inhibit HSC self-renewal and induce HSC senescence, resulting in premature exhaustion of HSCs and hematopoietic dysfunctions. Here we show that ROS similarly affect HSCs during their expansion phase in the embryo. We show that connexins are important in regulating ROS levels in HSCs. Indeed, their inhibition increases the level of ROS in HSCs and induces their cell-death in the Caudal Hematopoietic Tissue (CHT) niche of zebrafish embryos. The loss of HSCs after connexin inhibition can be rescued by reduced-Glutathione (GSH) treatment. In cells, GSH levels can be modulated by several enzymes. Here, we show the importance of a new gene in this process: Gamma-interferon-inducible lysosomal thiol reductase (GILT/ifi30), an important enzyme for antigen presentation in the context of immunity, can rescue the HSC loss resulting from ROS toxicity. Unexpectedly, we found that ifi30 was highly expressed in endothelial cells (ECs) from the CHT, but not in HSCs. Endothelial-specific ifi30 overexpression increased HSCs expansion in the CHT. Moreover, we found a high increase of ROS in ifi30-deficient embryos, resulting in a defect of HSC expansion in the CHT. This defect was rescued by several anti-oxidants: GSH and N-acetyl-cysteine. Altogether, our data show that HSCs transfer ROS to the endothelial niche, where all the tools are expressed to detoxify the microenvironment. This new role of ifi30 seems to be conserved during human embryogenesis as most of immature hematopoietic progenitors are associated with IFI30/GILT expressing cells in the human fetal liver.

Hybridization during density-dependent range expansion: European wildcats as a case study

Claudio S. Quilodrán, Juan I. Montoya-Burgos, Mathias Currat

Department of Genetics and Evolution. University of Geneva, Switzerland.

Interbreeding between historically allopatric species with incomplete reproductive barriers may result when species expand their range. The genetic consequences of such hybridization depend critically on the dynamics of the range expansion. Hybridization models during range expansion have been developed but assume dispersal to be independent from neighboring population densities. However, organisms may disperse because they are attracted by conspecifics or because they prefer depopulated areas. Here, through spatially explicit simulations, we assess the effect of various density-dependent dispersal modes on the introgression between two species. We find huge introgression from the local species into the invasive one with all dispersal modes investigated, even when the hybridization rate is relatively low. This represents a general expectation for neutral genes even if the dispersal modes differ in colonization times and amount of introgression. Invasive individuals attracted by conspecifics need more time to colonize the whole area and are more introgressed by local genes, while the opposite is found for solitary individuals. We applied our approach to a recent expansion of European wildcats in the Jura Mountains and the hybridization with domestic cats. We show that the simulations explained better the observed level of introgression at nuclear, mtDNA and Y chromosome markers, when using solitary dispersal for wildcats instead of random or gregarious dispersal, in accordance with ecological knowledge. Using density-dependent dispersal models thus increase the predictive power of the approach. Finally, we also present a new spatially explicit software to explore the ecological and evolutionary dynamic of many organism: SPLATCHE3.

Poster Session

Mimicking tubular environments to study epithelial sensing to curvature

Caterina Tomba [(1)], Florian Maechler [(1)], Valeriy Luchnikov [(2)], Aurélien Roux [(1,3)]

(1) Biochemistry Department, University of Geneva
(2) Université de Haute-Alsace, CNRS, IS2M, Mulhouse
(3) NCCR Chemical Biology, University of Geneva

During embryogenesis, epithelial tissues fold to establish the final shape of the different organs. These shape changes at the tissue level involve processes at the cellular scale, like cell shape changes and proliferation. However, the understanding of the mechanisms underlying the formation of epithelial tubes remains elusive due to the complexity to access to both the tissue and the cellular scale observation in 3D environments.

We developed two complementary approaches based on microfabrication techniques to control the 3D cell organization in tubular environments and to quantitatively study the epithelial response to curved confinements. The main difference between the two methods is to be a passive or an active constraint. These two approaches provide simple and reproducible tools to control the mechanical properties of the cell environment and to quantify their effect on cell growth.

We expect our results will contribute to provide useful insights for new quantitative studies of cell adaptation to curved and more bio-mimetic environments.

The functioning and regulation of Hsp90 co-chaperones: old and new

Stacey Mattison, University of Geneva, Pablo Echeverría, University of Geneva, Didier Picard, University of Geneva

The Hsp90 chaperome is the quality control machine of the cell. It requires the assistance of numerous co-chaperones to function in full capacity. We demonstrated the functionality of a novel isoform of the co-chaperone AHA1 (Activator of 90 kDa heat shock protein ATPase 1); the Putative activator of 90 kDa heat shock protein ATPase homolog 2 (AHA2). Furthermore we demonstarted the Hsp90 isoform specifity of a subset of co-chaperones by mapping the dynamic epigenetic landscape of enhancers.

Enhancers are distal regulatory elements that can regulate expression of their associated genes in cis. Although many epigenetic modifications have been described to occur at these elements, their causative effect on enhancer activity remains poorly understood. In this project, I systematically investigate how the crosstalk between the epigenetic landscape of enhancers and local transcription factor occupancy regulates the expression of target genes. I use recently developed epigenome editing tools based on the CRISPR/Cas9-system to specifically and rapidly modulate individual epigenetic events as well as transcription factor binding at enhancers. Then, I will conduct time-course experiments to measure the fast and slow dynamic changes of the local epigenetic landscape and target gene expression. Importantly, by using such recently developed methods to manipulate the epigenome in living cells we will be able to transition from passive observations to active mechanistic and functional assays in a cellular context, hence allowing us to move from correlative studies to finding the causation behind the correlations. This project will result in a map to navigate the epigenetic landscape of enhancers in vivo, which will aid us in deciphering the fundamental principles of cellular diversity. Since our results will be interpreted in the context of the cell, this will furthermore yield potentially useful information for future clinical applications.

Uncovering the interplay between the growth-promoting transcription factor Sfp1 and the stress-responsive transcriptional activator Msn2

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Department of Molecular Biology, University of Geneva and Institute of Genetics and Genomics in Geneva (iGE3), Geneva, Switzerland The Split zinc-finger protein 1 (Sfp1) is a major orchestrator of transcriptional programs supporting cell growth in the budding yeast Saccharomyces cerevisiae, including the complete sets of ribosomal protein (RP) and ribosome biogenesis (RiBi) genes. Conversely, Msn2, another zinc-finger transcription factor, binds to and activates a large suite of genes associated with the environmental stress response (ESR).

Using the anchor-away technique, we were able to assess the immediate transcriptional consequences of Sfp1 nuclear depletion at a genome-wide level by RNA Polymerase II (RNAPII) ChIP-seq. Shortly after initiating Sfp1 depletion RP and RiBi genes are strongly down-regulated whilst, surprisingly, a large subset of ESR genes activated by Msn2 factor are up-regulated. Notably, anchoring away of Msn2 during growth in low glucose revealed the exact opposite trend: stress genes were down-regulated whilst RiBi and RP genes were mostly up-regulated.

Sfp1 and Msn2 both operate through the coactivator Tra1, a scaffolding subunit of the two major histone acetyltransferases in yeast, SAGA and NuA4. We established a functional link between Sfp1 and Tra1 by quantifying histone acetylation levels following nuclear depletion of Sfp1. Levels of H3-K9Ac and H4-Ac correlated with that of RNAPII genome-wide, i.e. increased acetylation was observed at Msn2 target genes upon Sfp1 depletion, whereas acetylation at Sfp1 target gene promoters decreased. We are currently testing the hypothesis that Sfp1 and Msn2 compete for limiting amounts of Tra1 (in either SAGA and/or NuA4 complexes), thus explaining how depletion of one activator can increase transcription through the other. This might help wild-type cells to more efficiently switch between growth- and stress-related transcriptional programs.

Induction of ligand promiscuity of cell-matrix receptor αVβ3 integrin by mechanical force

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Integrin-ligand interactions anchor cells in their extracellular environment. These interactions are described as promiscuous compared to the interaction between other cell adhesion receptors and their binding partners. αVβ3 integrin for example is reported to have at least 12 possible ligands like fibronectin (Fn), vitronectin (Vn), fibrinogen, and osteopontin. However, when cell behavior is analyzed in cell culture, integrin ligands are often presented as homogeneous substrates. In tissues, in contrast, cells experience highly complex environments, where changes in ligand type, geometry, and physical properties are frequent and accompany pathological changes. For example, increased expression of vitronectin is associated with different cancer types and fibrotic tissues are characterized by mechanical stiffening.

We introduce binary choice substrates in the micrometer range that allow cells to choose between different ligands. These substrates combined with cell culture experiments and superresolution microscopy reveal that αVβ3 integrin binds only to a subset of possible ligands when the cell is under low mechanical load. Stronger pulling by the cellular actin-myosin network leads to conformational changes in the protein structure of αVβ3 integrin that allow the binding of additional ligands. Thus, ligand promiscuity of αVβ3 integrin is induced by mechanical load on the matrix-integrin-actin axis.

Sensing of mechanical cues and responding to them is of increasing interest in cell biology. Certain pathologies like fibrosis and cancer are associated with tissue stiffening and a change in extracellular matrix composition. Our results indicate that this tissue stiffening might induce ligand promiscuity of αVβ3 integrin and thereby increase cell adhesion and favor cell signaling by cancer associated cells. This force-dependent binding to different ligands introduces a new layer of regulation for the interaction of cells with their environment.

D. discoideum flotillin homologues are essential for phagocytosis and manipulated by Mycobacteria during infection

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Tuberculosis is a human disease caused by the pathogen Mycobacterium tuberculosis (Mtb). Mtb is phagocytosed by macrophages, but is capable of manipulating the maturation pathway of its compartment (MCV). Moreover, Mtb damages the MCV thanks to factors secreted via the ESX-1 secretion system, allowing it to access the nutrients in the cytosol. We use the Dictyostelium discoideum – Mycobacterium marinum model to study host-pathogen interactions in mycobacterial infections.

The vacuolin protein family in D. discoideum is highly similar to flotillins in animals, which are membrane-associated proteins involved in signaling and recycling of plasma membrane cargoes. We showed that all three vacuolins are present on endocytic compartments and are associated with membranes. When vacuolins are absent, cells are not able to phagocytose different particles and take longer to recognize and adhere to particles. In addition, the recycling of plasma membrane proteins, involved in adhesion or membrane trafficking, is affected in absence of vacuolins. Moreover, when vacuolins are absent, expression of MyoI, a protein involved in phagocytosis and adhesion, is downregulated. These results suggest that vacuolins may be important for the correct localization or function of MyoI in phagocytosis and adhesion.

We also investigated the role of vacuolins during infection with M. marinum. We found that vacC is specifically induced during infection by mycobacteria. In absence of vacuolins, intracellular growth of M. marinum is impaired and it resides longer in an intact MCV. We suggest that vacuolin-rich microdomains facilitate the damaging of the MCV by allowing insertion of membrane-disrupting factors secreted by M. marinum, thus favoring its replication and access to the cytosol. We propose that vacuolins are important host factors that are highjacked by the pathogenic M. marinum to establish its infection.

Using Tn-Seq strategy in Mycobacterium marinum - Dictyostelium discoideum to understand the pathogenesis of Tuberculosis

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How do mycobacteria manipulate the fundamental processes of cell-autonomous immunity, especially the phagosome environment? How does the host overcome the infection? Which genes are involved in the establishment and maintenance of the infection?

This study aims at further developing D. discoideum – M. marinum as a powerful genetically tractable host-pathogen model to investigate pathogenesis of Tuberculosis.

We applied Transposon Sequencing (Tn-Seq) in M. marinum M strain to quantitate before and after selection in D. discoideum at 24hpi, 48hpi and 96hpi the essentiality and fitness cost, in terms of growth advantage and disadvantage of over 105 mutants.

Our first results indicate that ~10% of the genes are essential for growth in vitro, with ~86% shared between various conditions and ~50% with M. tuberculosis.

We identified that mutants in genes encoding the T7SS ESX-1, biotin biosynthesis and the response to iron starvation are impacted already at 24hpi. Finally, at 96hpi the lipids catabolism is mostly represented (e.g Mce transporters).

From our selection, surprisingly and contrary to M. tuberculosis, only mce1 mutants seem to be impacted during infection. Therefore, we compared the role of the two transporters Mce1 (fatty acid) and Mce4 (cholesterol) in M. marinum in broth and during infection. Using flow cytometry quantification of Bodipy-C14 in broth, we observe that the mce1 mutant incorporates less neutral lipids compared to mce4. Lipids identity was investigated by TLC showing less TAGs for both mce1 and mce4 mutants. By immunofluorescence, we notice that mce mutants seem to use different strategies to escape the phagosome. We also observe by high content microscopy that mce1 and mce4 mutants both have a growth advantage during infection of D. discoideum and macrophages.

Therefore, these preliminary results by using different approaches suggest that in M. marinum strain M mce1 and mce4 mutants use different strategies compared to M. tuberculosis.

Measuring lipid membrane properties of organelles and plasma membrane: From in vitro to tissues

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Membrane tension is thereby a major regulator of membrane remodeling cell processes, but has proved very hard to measure in vivo. FliptR (for Fluorescent LIPid Tension Reporter) can monitor changes of membrane tension by changing its fluorescence lifetime as a function of the twist between its fluorescent groups. We show that fluorescence lifetime depends linearly on membrane tension within cells, allowing for an easy quantification of membrane tension by fluorescence lifetime imaging microscopy (FLIM) and Fast-FLIM. We further showed using model membranes that this linear dependency between lifetime of the probe and membrane tension relies on a membrane-tension dependent lipid phase separation. We obtained a calibration curves that allow to measure accurately membrane tension using FLIM and Fast-FLIM in vivo. FliptR thus tremendously facilitates membrane tension measurements, now we are studying membrane tension on plasma membrane and in the organelles in cell processes and developing tissues.

Comparative cell biology of endocytosis in yeasts

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Clathrin-mediated endocytosis is a major membrane trafficking pathway in eukaryotes. The protein machinery that drives endocytosis is largely conserved throughout the eukaryotic tree of life. However, the endocytic process differs among species and we know little about the mechanistic implications of these differences. Here, we study interspecies differences aiming to understand the evolutionary pathways that led to the different variants of the endocytic process and to identify the conserved core mechanisms.

Budding yeast, Saccharomyces cerevisiae, and fission yeast, Schizosaccharomyces pombe, are two distantly related species from the phylum Ascomycota. Both yeasts are amenable for genetic manipulations and live cell imaging, and their endocytic machineries have been extensively studied. We fluorescently tagged endocytic proteins in both species and we used quantitative live cell and superresolution imaging to compare protein dynamics and localization patterns at endocytic sites.

We found that endocytic protein dynamics and the time it takes for the membrane invagination to grow are remarkably similar in the two species. However, the invagination grows ~60% faster in fission than in budding yeast. Therefore, in fission yeast, invaginations are longer and vesicles are released further away from the plasma membrane than in budding yeast. WASp homologs Wsp1 and Las17, respectively in fission and budding yeast, nucleate the branched actin network that propels the invagination growth. In both species, Wsp1 and Las17 molecules organize in a 70-nm wide ring region that surrounds the invagination. The number of Wsp1 molecules that occupy this region is up to two times higher than the number of Las17 molecules. We hypothesize that the different Wsp1 and Las17 densities might be responsible for the different invagination speeds.

This comparative study will allow us to identify which of the elements that regulate invagination growth can vary and how they affect endocytosis.

Investigating neurodegeneration in a novel model of Parkinson's Disease : a neuroprotective transcription factor conserved from the fly to mammals

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Parkinson's disease (PD) is the second most common neurodegenerative disorder, primarily caused by the specific and progressive loss of dopaminergic (DA) neurons in the Substantia Nigra. Despite the discovery of several genes implicated in PD, the scarcity of animal models showing robust degeneration of DA neurons has hindered the study of the pathology, and no protective or restorative therapies are currently available.

We have recently established a novel PD model in Drosophila, the Fer2 gene loss-of-function mutant, which recapitulates several hallmarks of PD, notably the specific loss of DA neurons, severe locomotor impairment and mitochondrial defects (Bou Dib et al., PLoS Genetics, 2014). Moreover, Fer2 over-expression in DA neurons is sufficient to rescue neurodegeneration in a number of genetic PD models and upon oxidative insults, further highlighting Fer2 importance for DA neurons protection.

Since Fer2 is a transcription factor, we hypothesized that it controls a specific set of target genes to exert its beneficial role. By combining Chromatin Immunoprecipitation coupled to sequencing (ChIPseq) and transcriptome analysis (RNAseq), we identified over 30 bona fide direct targets, bound and regulated by Fer2. A knock-down/over-expression screening functionally validated several of these genes as novel actors in DA neurons survival. Furthermore, profiling of DA neurons-specific transcriptome revealed Fer2 regulation of multiple components of the electron transport chain, pointing out a crucial role for mitochondria in Fer2-medited neuroprotection.

Current investigation of Fer2 mammalian counterpart, through its conditional inactivation in mouse, is revealing the importance of this gene for DA neurons gene expression and physiology, and for mouse locomotion. Elucidation of the genes and processes governed by Fer2 across evolution will provide novel insights into the degeneration of DA neurons, and potential new targets for therapeutic intervention.

Some shall pass: Molecular mechanism underlying adaptive response of nutrient barrier in Arabidopsis

Vinay Shukla

Nutrient acquisition in adequate amounts is an essential aspect of regular plant growth and development. Materials taken up by the plant roots are transported to aerial organs to provide the supplies for photosynthesis and other metabolic processes in vascular plants. In order to cope with the fluctuations in nutrient availability in soil, plant root growth and development require high plasticity. Similar to specialized epithelial cells of animal gut, endodermis cell layer of plant roots facilitates selective uptake of nutrients from the soil. Formation of the secondary cell wall by deposition of suberin lamellae is one such mechanism that provides another layer of selectivity to this nutrient barrier. It has been shown only recently that plant roots also efficiently regulate the status of suberin lamellae in order to permit or block the amounts of specific nutrients that are absorbed from the soil. Hormonal regulation of enhanced suberization through abscisic acid (ABA) has been well-studied, however, the downstream important players remained unknown. Therefore, we performed an in-depth analysis of nutrient stress dictating hormonal regulation of suberin lamellae. Our study led to the identification of primary transcription factors involved in ABA-dependent as well as independent suberization of the root. Our results also demonstrate the physiological relevance of the enhanced suberization in terms of plant survival to nutrient stress, which was not well understood from past studies. Our study introduces the novel regulators of nutrient barrier in plants and this will allow fine-tuning of crops and will lead to better yield without the usage of non-sustainable chemical fertilizers.

Our Sponsors

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