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• **May 17th 2018** •

• Grand Auditorium, École de Physique •

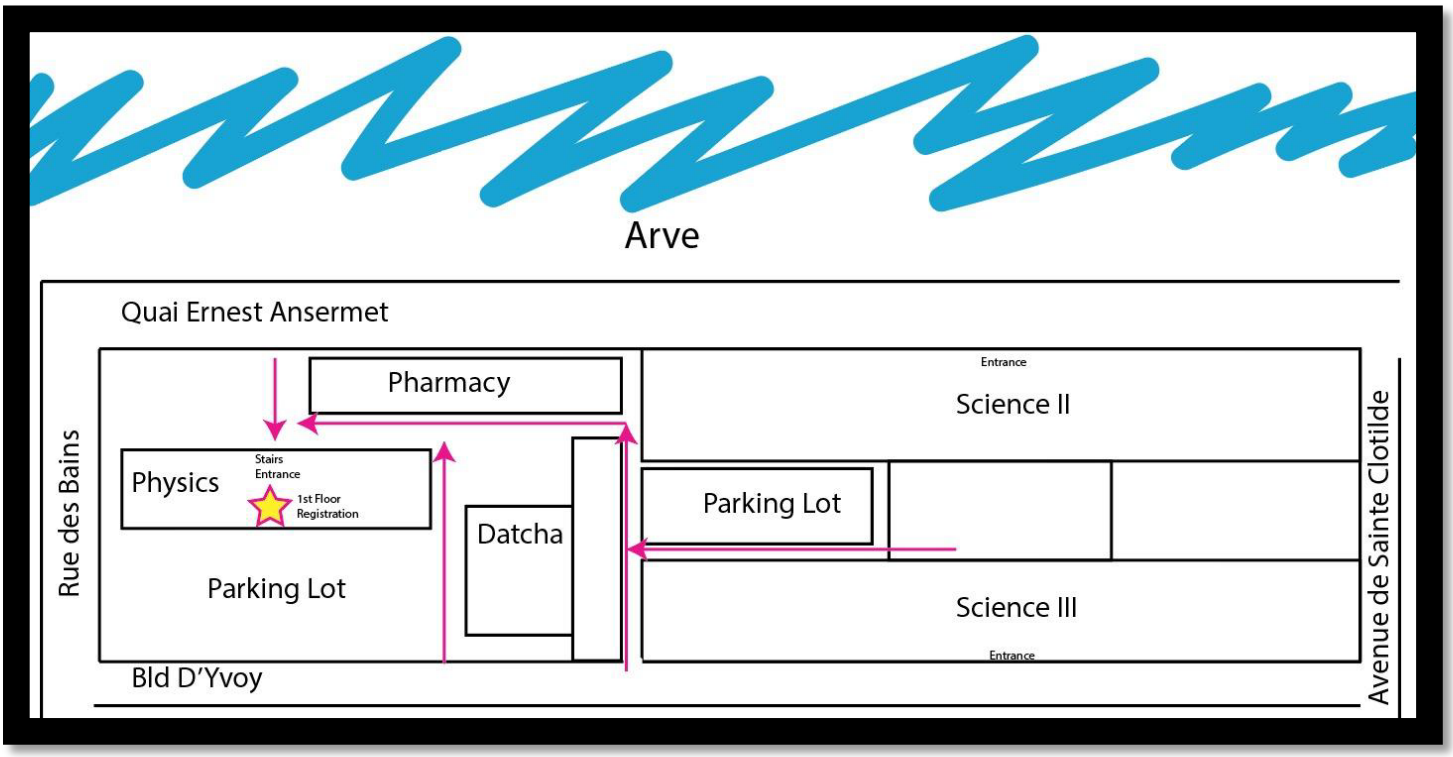
POST-DOC ASSOCIATION OF THE UNIVERSITY OF GENEVA

APDUG

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.



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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.
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Organizing Committee

+ Special thanks:

We are really grateful for the help of Louise Lefrançois who volunteered to organize the catering, Nicolas Roggli for his help with the registration webpage and Margot Riggi for her valuable contribution to our visuals.

Our Sponsors

Many thanks to all the institutions who contributed financially to make this event happen.

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– PROGRAM –

POSTDOC DAY

8h30 - 9h00 *Registration and poster set up*

9h00 - 9h10 *Opening Remarks*

Part I - Who are your colleagues?

9h10 - 10h00 *Postdoc scientific talks:*

Viola Puddinu: *From the trench to the open-field battle: Insight from an immune-excluded model of gastric cancer*

Andrea Picco: *An integrative fluorescence microscopy approach to reconstruct 3D architecture of protein complexes in vivo*

Dumitru Dumcenco: *2D materials beyond graphene*

Caroline Arous: *Analysis of the adhesion-mediated control of insulin secretion in response to glucose and autocrine insulin/IGF2-signaling in pancreatic b-cells*

10h00 - 10h45 ***Keynote speaker – BIOLOGY: Prof. Alain Chedotal*** *(Institut de la Vision, Paris)*
"3D analysis of embryonic development: of mice and men"

10h45 - 11h00 *Coffee Break*

11h00 - 12h00 *Postdoc scientific talks:*

Davide Gambarotto: *Imaging beyond super-resolution limits using ultrastructure expansion microscopy (UltraExM)*

Giulia Cheloni*: United we stand, divided we fall: Chlamydomonas strategy to endure micropolluants toxicity*

Eddie Rodriguez-Carballo*: The HoxD cluster is a dynamic and resilient TAD boundary controlling the regulation of antagonistic regulatory landscape*

Elena Cardenal-Muñoz*: CnrD and CueA, two novel autophagy receptors*

***Marie Pireyre**: Light perception by plants: UV-B induces specific transcriptional networks in Arabidopsis thaliana*

12h00 - 12h05 ***The PIs of Tomorrow**, LS^2 Competition – Carolin von Schoultz, PhD*

12h05 - 13h50 *Lunch and Poster presentation.*

Part II - Focus on your career:

13h50 - 14h35 ***Keynote speaker – PHYSICS: Prof. Chiara Mariotti***
CERN, Geneva
"The story of a discovery: How we found the long-sought-after Higgs boson"

14h35 - 14h50 ***Postdoc mentoring program presentation***
Prof. Aurélien Roux

14h50 - 15h35 ***Seeking opportunities outside academia – INDSUTRY: Prof. Piergiorgio Pettazzoni** (Lab Head at Roche, Basel)*
"Targeted degradation of oncogenic proteins, a new paradigm in drug discovery"

15h35 - 15h50 *Coffee Break*

15h50 - 16h35 ***Keynote speaker – CHEMISTRY: Prof. Michael Graetzel** (EPFL, Lausanne)*
"Energy beyond oil"

16h35 - 17h30 *Open discussion:*

Becoming a Professor – How to succeed in a selection process.
Prof. Howard Riezman, Prof. Nicolas Winssinger,
Prof. Paul Guichard, Prof. Marie Barberon

17h30 - 17h35 ***Concluding Remarks and best Poster/Talk Awards***

17h35 - 20h00 ***Apéro***

Part I - Who are your colleagues?

Session 1: 9h10-10h00

From the trench to the open-field battle: Insights from an immune-excluded model of gastric cancer.

Viola Puddinu[1], BetülTaşkoparan[1], Nathalie Steinhoff[2] and Carole Bourquin[1].

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[2] Chair of Pharmacology, Department of Medicine, Faculty of Science, University of Fribourg, 1700 Fribourg, Switzerland.

Gastric cancer is the fifth most frequent type of cancer worldwide and the third leading cause of cancer-related death. In the last years immunotherapy, which aims to activate the immune system against tumors, has shown great success in treating certain types of advanced cancer and constitutes a promising approach to treat patients with gastric cancer. Nevertheless, only 40 % of patients respond to immunotherapy, underlying the need to better understand the mechanisms of anticancer immunity. Among other factors, the pre-existing activation of the immune system against cancer is necessary for a good response. The most responsive patients have so called “inflamed” tumors, which are infiltrated with immune cells, such as T lymphocytes. Other patients have “immune-excluded” cancers, where T cells are confined to the boundaries of the tumor, leading to reduced responsiveness. Since the presence of T cells in tumors is critical, it is important to use proper models to study the recruitment of T cells to immune-excluded tumors. We work with SV40 T Ag transgenic mice, which develop spontaneous tumors in the stomach. Tumors in these mice grow gradually from premalignant lesions to invasive carcinoma, as is usually the case in patients. Using flow cytometry and fluorescence microscopy, we found that immune cells, including T cells, preferentially localize in the tumor-surrounding stroma rather than in the center of the tumor, indicating that these mice constitute a valid preclinical model to study immune-excluded gastric cancer. We are now investigating treatment modalities to enhance T-cell recruitment to the tumors in these mice.

An integrative fluorescence microscopy approach to reconstruct the 3D architecture of protein complexes in vivo.

Andrea Picco[1,5], Ibai Irastorza-Azcarate[2,5], Tanja Specht[4], Dominik Böke[4], Irene Pazos[3], Anne-Sophie Rivier-Cordey[1], Damien P. Devos[2], Marko Kaksonen[1] and Oriol Gallego[3]

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[2] Centre for Organismal Studies, Heidelberg University, Heidelberg, Germany and Centro Andaluz de Biología del Desarrollo (CABD), Universidad Pablo de Olavide-CSIC, Carretera de Utrera km1, 41013 Sevilla, Spain.
[3] Institute for Research in Biomedicine (IRB Barcelona), The Barcelona Institute of Science and Technology, c/ Baldiri Reixac 10, 08028 Barcelona, Spain.
[4] Cell Biology and Biophysics Unit, European Molecular Biology Laboratory (EMBL), Meyerhofstrasse 1, 69117 Heidelberg, Germany
[5] Co-first author

The structural characterization of protein complexes in their native environment is challenging but crucial for understanding the mechanisms that mediate cellular processes. We developed an integrative approach to reconstruct the 3D architecture of protein complexes in vivo. We applied this approach to the exocyst, a hetero-octameric complex of unknown structure that is thought to tether secretory vesicles during exocytosis with a poorly understood mechanism. We engineered yeast cells to anchor the exocyst on defined landmarks and determined the position of its subunits termini at nanometer precision using fluorescence microscopy. We then integrated these positions with the structural properties of the subunits to reconstruct the exocyst together with a vesicle bound to it. The exocyst has an open hand conformation made of rod-shaped subunits that are interlaced in the core. The exocyst architecture explains how the complex can tether secretory vesicles placing them in direct contact with the plasma membrane.

2D materials beyond graphene.

Dumitru Dumcenco

Department of Quantum Matter Physics, University of Geneva.

Two-dimensional (2D) materials consisting of layers bonded by the weak van der Waals interactions. Since the exfoliation of graphene in 2004, they have been receiving a great attention because of the qualitative changes in their physical properties by thinning down to a monolayer thickness. Beyond graphene, the family of 2D materials contains a wide selection of compositions including most elements of the periodic table. This makes it possible to achieve the materials with a variety of electronic properties, such as metals, semimetals, insulators, and semiconductors with the direct and indirect bandgaps extending from the ultraviolet to infrared spectral range. In addition, the excellent compatibility with the current thin film manufacture techniques in the semiconductor industry can facilitate the integration of 2D materials with traditional electronic materials, such as Si. It means that 2D materials have a potential to play a significant role in the future nanoelectronics, optoelectronics, and the development of novel ultrathin and flexible devices. Magnetic order in 2D semiconducting layered materials is extremely rare, and this recently generated a great interest in a variety of layered magnetic compounds. In case, if their magnetic properties can be retained down to a monolayer thickness, such materials could have applications in the magnetoelectronics, like data storage and sensing. In this talk, the elaboration and characterization of various compounds including the magnetic family are going to be presented and discussed.

Analysis of the adhesion-mediated control of insulin secretion in response to glucose and autocrine insulin/IGF2-signaling in pancreatic β-cells.

Caroline Arous[1], Karim Bouzakri[2], Philippe Halban[3] and Bernhard Wehrle-Haller[1]

[1] Department of Cell Physiology and Metabolism, Centre Médical Universitaire, University of Geneva, Switzerland.
[2] UMR DIATHEC, EA 7294, Centre Européen d'Etude du Diabète, Université de Strasbourg, Strasbourg, France.
[3] Department of Genetic and Development, Centre Médical Universitaire, University of Geneva, Switzerland.

Elevated basal insulin secretion under fasting conditions and insufficient glucose-stimulated insulin secretion (GSIS) by β-cells of pancreatic islet are important hallmarks of diabetes. Recent data proposed an improvement of β-cell function (GSIS and survival) when β-cells are anchored to the pancreatic tissue, called extracellular matrix (ECM), by β1-integrin-containing focal adhesions (FA). Integrin-dependent FAs are sites of mechanical linkage between the ECM and the actin cytoskeleton at cell membrane. To these FAs, adapter proteins such as focal adhesion kinase (FAK), and paxillin are recruited to mediate intracellular signaling. We have previously shown that glucose-induced remodeling of the actin cytoskeleton and FAs is involved in the regulation of insulin secretion in β-cell. However how adhesion are regulated is still unknown. We wondered whether the autocrine release of insulin/IGF2 itself could be a FAs modulator. Indeed, insulin/IGF2 signaling appears to be an important FA regulator in other cell types. Although the autocrine role of insulin/IGF2 on insulin secretion is still controversial, few evidences suggest glucose intolerance and elevated insulin secretion during fasting is prevent by activation of Insulin/IGF1 receptors (IR/IGF1R) signaling. We have studied the involvements of IR/IGF1R-PI3K-AKT signaling in the FAK-paxillin-mediated β-cells secretory functions. Our results showed that the activation of the IR/IGF1R-PI3K-AKT1 pathway enhances GSIS by a partial modulation of the adhesion remodeling. In contrast, under low-glucose condition (fasting condition), the IR/IGF1R-PI3K-AKT2 pathway blocks insulin secretion, while regulating FAK-paxillin-dependent FA signaling and associated F-actin remodelling. Interestingly, we have also confirmed that basal insulin secretion regulation is a FAK dependent signaling. Our data propose that autocrine insulin/IGF2-mediated signaling forks at the AKT1 and AKT2 level to mediate adhesion specific signaling in a glucose dependent manner to regulate insulin secretion. A potential dysfunction of the IR/IGF1R-AKT1/2-adhesion signaling pathway could explain why diabetic patients exhibit hyperinsulinemia under fasting conditions and insufficient GSIS.

Session 2: 11h00-12h00

Imaging beyond the super-resolution limits using ultrastructure expansion microscopy (UltraExM)

D. Gambarotto, F. U. Zwettler, M. Cernohorska, D. Fortun, S. Borgers, J. Heine, J. G. Schloetel, M. Reuss, M. Unser, E. S. Boyden, M. Sauer, V. Hamel and P. Guichard.

Centrioles lie at the core of the centrosome and template cilia or flagella in most of animal cells. Electron microscopy and more recently cryo-electron microscopy allowed us to disclose the centriole architecture, which has been very well conserved throughout evolution. Centrioles are cylinders composed by sets of microtubules arranged in a nine-fold radial symmetry. Along their long axis, three distinct regions with specific structural features can be recognized: a 100nm proximal part including the cartwheel, a 250-300nm central core decorated by the Y-shaped linker and a 50-100nm distal part including the appendages. Proteomic analyses and functional approaches revealed that the centriole requires more than 100 different proteins. One of the current challenges is now to map these proteins to specific structural features of the centriole in order to better understand their role in centriolar assembly and function. In the past five years, the use of super-resolution microscopy helped to spatially localize some proteins of the centriole and others of the pericentriolar material that surrounds the centriole. However, mapping proteins to specific structural features of the centriole is still a challenge. To circumvent this gap, we are developing a new approach based on the innovative method of Expansion microscopy (ExM). In ExM, a specimen is embedded and crosslinked into a swellable polymer network that can physically expand. Importantly, upon polymer expansion, the specimen expands as well, up to a 4.5-fold in an isotropic manner. Here, we present a novel expansion method named UltraExM (Ultrastructure Expansion Microscopy) that is amenable to precise mapping of centriolar proteins, with a particular interest to the tubulin modifications in the microtubule wall of the centriole. Taken together, we expect UltraExM to generate the first unprecedented detailed protein cartography of centrioles.

United we stand divided we fall: Chlamydomonas strategy to endure micropollutant toxicity

Giulia Cheloni[1], Michel Goldschmidt-Clermont[2] and Vera I Slaveykova[1].

[1] Environmental biogeochemistry and ecotoxicology group, Department F.-A. Forel for Environmental and Aquatic Sciences (DEFSE), UNIGE.
[2] Chloroplast Molecular Genetics group. Department of Botany and Plant Biology (BIVEG).

Anthropic impact on aquatic environments results in the release of chemicals in water bodies; such micropollutants (MPs) represent a threat to aquatic organisms. Phytoplankton play a key role in biogeochemical cycles of elements and toxic effects of micropollutants on these organisms may have important consequences on the entire aquatic food web. Phytoplankton have evolved multiple strategies to maintain cellular homeostasis and endure adverse environmental conditions however, still little is known about micropollutant induced stress response pathways and phenotypic plasticity under exposure to chemical stress. In our work, we investigate how, in response to MP exposure, the motile green alga Chalmydomonas reinhardtii switches from unicellular to colonial lifestyle with the formation of small clusters of cells called palmelloids. Despite the deep knowledge of C.reinhardtii cell biology, the mechanisms that drive palmelloid colony formation were poorly investigated. Results obtained so far revealed that palmelloid formation is induced by the presence of sub-lethal MP (Copper, Cadmium, Paraquat, PFOS) concentrations. Microscopic observations highlighted the presence of multiple cell wall-like envelopes and that palmelloid formation is associated to the retention of the daughter cells within the mother cell wall. Cells keep growing and dividing within the palmelloid colony when MPs are present, whereas cells revert to the unicellular lifestyle when colonies are harvested and resuspended in pristine medium. Palmelloid formation is not associated to cellular toxic effects, suggesting that cells within the colony are protected from the MP toxicity. Altogether, our results indicated that palmelloid formation is a cellular response to stress that allows C. reinhardtii to face adverse environmental conditions. C. reinhardtii is considered the unicellular reference for the investigation of the evolution of multicellularity in the plant kingdom. The mechanistic investigation of palmelloid colony formation is expected to have far-reaching evolutionary implications.

The HoxD cluster is a dynamic and resilient TAD boundary controlling the segregation of antagonistic regulatory landscapes.

Eddie Rodríguez-Carballo[1], Lucille Lopez-Delisle[2], Ye Zhan[3], Pierre J. Fabre [2#], Leonardo Beccari 1, Imane El-Idrissi[1], Thi Hanh Nguyen Huynh[1], Hakan Ozadam[3], Job Dekker[3] and Denis Duboule[1,2,4].

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[2] School of Life Sciences, Ecole Polytechnique Fédérale de Lausanne, 1015 Lausanne, Switzerland.
[3] Program in Systems Biology, Department of Biochemistry and Molecular Pharmacology, University of Massachusetts Medical School, Howard Hughes Medical Institute, Worcester, Massachusetts 01605, USA
[#] Present address: Department of Basic Neurosciences, University of Geneva, Switzerland.

The mammalian HoxD cluster lies between two topologically associating domains (TADs) matching distinct, enhancer-rich regulatory landscape. During limb development, the telomeric TAD controls the early transcription of Hoxd gene in forearm cells, whereas the centromeric TAD subsequently regulates more posterior Hoxd genes in digit cells. Therefore, the TAD boundary prevents the terminal Hoxd13 gene to respond to forearm enhancers, thereby allowing proper limb patterning. To assess the nature and function of this CTCF-rich DNA region in embryo, we compared chromatin interaction profiles between proximal and distal limb bud cells isolated from mutant stocks where various parts or this boundary region were removed. The resulting progressive release in boundary effect triggered inter-TAD contacts, favoured by the activity of the newly accessed enhancers. However, the boundary was highly resilient and only a 400kb large deletion including the whole gene cluster was eventually able to merge the neighbouring TADs into a single structure. In this unified TAD, both proximal and distal limb enhancers nevertheless continued to work independently over a targeted transgenic reporter construct. We propose that the whole HoxD cluster is a dynamic TAD border and that the exact boundary position varies depending on both the transcriptional status and the developmental context.

CnrD and CueA, two novel autophagy receptors

Elena Cardenal-Muñoz[1], Imen Ayadi[1], Jason S. King[2,3], Thierry Soldati[1].

[1] Department of Biochemistry, Faculty of Science, University of Geneva, Switzerland.
[2] Centre for Membrane Interactions and Dynamics, Department of Biochemical Sciences, University of Sheffield, United Kingdom
[3] Bateson Centre, University of Sheffield, United Kingdom.

Autophagy is a catabolic pathway that eukaryotic cells use to digest and recycle cytoplasmic components. Under conditions such as nutrient starvation or oxidative stress, cells generate double-membrane vesicles to engulf damaged organelles and misfolded proteins in so-called autophagosomes, which fuse with lysosomes to degrade their content. Cells also induce a similar process, called xenophagy, to specifically capture and kill intracellular pathogens. This pathway relies on receptors that are selectively recruited to the microbes, which are decorated with "eat-me" signals such as ubiquitin, to engulf them into autophagosomes for killing and degradation. We study xenophagy in the amoeba Dictyostelium. We have previously demonstrated that Mycobacterium marinum, a close relative of M. tuberculosis, antagonistically induces an early autophagic response while repressing its autophagic digestion. In addition, the homolog of the mammalian autophagy receptor p62/SQSTM1 links ubiquitinated bacteria to autophagosomal membranes. However, and very interestingly, Atg8-positive structures and membranes are still recruited to bacteria in the absence of p62, suggesting that other xenophagy receptors exist in Dictyostelium. Here we studied two proteins, CnrD and CueA, as potential xenophagy receptors. They contain CUE domains for binding to ubiquitin, and conserved protein regions for the interaction with the autophagosomal marker Atg8 (the Dictyostelium ortholog of LC3). Moreover, they accumulate in aggregates in autophagy-null cells, are degraded upon autophagy induction, and co-localize with autophagosomal membranes and ubiquitinated mycobacteria independently of p62. Surprisingly, the intracellular bacteria load only increases slightly in p62- cells, while it decreases in cnrD- cells. This leads us to hypothesize that the three receptors have complementary but also divergent functions. We will discuss the putative roles of these xenophagy receptors in supporting (by providing nutrients or membranes to the bacteria-containing compartment) or counteracting (by targeting bacteria to autolysosomes) the mycobacterial infection.

UV-B in light induces specific transcriptional regulation in plants

Marie Pireyre*, Eleni Tavridou* and Roman Ulm.

University of Geneva, Department of plant biology and botany.

Plant are sessile organisms and to adapt to their ever-changing environment, they have developed complex strategies to adapt accordingly. Their plasticity is illustrated by their high diversity of transcription factors (TFs); in Arabidopsis between 6% and 10% of genes encode TFs, in contrast to only 5% in humans. Those TFs are parts of sophisticated regulatory mechanisms allowing plants to "feel" and respond accordingly. Perception of the whole light spectra is a major process allowing the plant to optimize its growth. It is controlled by numerous photoreceptors, each sensitive to specific wavelength and activating downstream transcription factors. For example, PIF4/5 transcription factors interact with PHY photoreceptors in response to red and far-red light in the processus of shade avoidance (Pfeiffer et al. 2014). On the opposite, UVR8 photoreceptor senses UV-B and activates downstream TFs, HY5 and HYH. UV-B responses in hy5hyh mutants are almost completely abolished (Yin and Ulm. 2017). Increasing evidences suggest that these pivotal TFs ellicit opposing responses in light sensing. Using RNAseq analysis, we identify a set of genes that are UV-B induced independently of HY5 and HYH, suggesting potential alternative factors triggering gene expression. Promoter analysis of those genes revealed enrichment for G-box motif and PIFs binding site. Moreover over 50% of those genes have been previously reported as direct targets of PIF4/5 (Pfeiffer et al. 2014). We analyzed PIFs proteins behaviour in response to UV-B, our results pointing towards repression of PIFs proteins by UVR8 photoreceptor or competition for promoter binding with HY5 in response to UV-B. Our data suggests that UVR8 negatively regulates PIF4/5 protein stability and their capacity to bind promoters while it stabilizes HY5 and enhances its binding to UV-B responsive genes. Taken together, our results shed light on the molecular interaction between HY5 and PIF4/5 for transcription regulation under UV-B.

Poster Session

Poster I

RNA-seq profiling of the infection of Dictyostelium by Mycobacterium marinum reveals an integrated host response to damage and stress

Nabil Hanna, F. Burdet, A. Melotti, H. Hilbi, P. Cosson, M. Pagni, T. Soldati.

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[2] SIB, Vital-IT, Génopode building, Lausanne, Switzerland.
[3] Faculty of Medicine, Department of Cell Physiology and Metabolism, University of Geneva, Switzerland
[4] Institute of Molecular Life Sciences, University of Zurich, Switzerland.

Tuberculosis remains the most pervasive infectious disease and the emergence of multiple drug-resistant strains emphasizes the need for more efficient and better drug treatments. We use an integrated approach to dissect and model the relationship between mycobacteria and their host. The experimentally versatile Dictyostelium discoideum – Mycobacterium marinum infection model provides a powerful and ethically un-concerning system to study mycobacteria pathogenicity. We are using the technological developments in high throughput RNA-sequencing to determine transcriptional signatures triggered by mycobacteria infection. We investigated how the host Dictyostelium fine-tunes its gene expression in response to M. marinum infection by assessing its transcriptomic profile during early and late time-points of infection (1, 3, 6, 12, 24 and 48 hours post infection). Differential gene expression and GO Term analysis shows a clear "host damage" signal, especially in specific pathways involved in membrane repair (ESCRT) and bacteria elimination (autophagy). Maybe more unexpectedly, we also obtain signatures of cell cycle regulation (cytosolic large & small ribosomal subunits) and metabolic adaptations (lipids transport). Preliminary RNA-seq data from intracellular M. marinum showed an upregulation of virulence factors (esxA and esxB) as well as of genes involved in bacterial metabolism and persistence in the host (icl1). Although the damage to the membrane of the mycobacteria-containing vacuole leads to a well-orchestrated early autophagic response, the regulation of TORC1 and autophagy genes (atg1, atg8,..) remains unclear. Based on our RNA-seq data, we will investigate the function of a WIPI3 homolog in autophagy signalling and autophagosome formation during Mycobacterium infection. The altered transcriptional signatures caused by the addition of a small collection of anti-infective compounds, previously identified by screening on an amoeba – M. marinum infection model, will help to identify their molecular targets and lead

to an extensive bio-informatic analysis to build metabolic maps and decipher novel regulatory pathways.

Poster 2

Epithelium adaptation to external curvature *in vitro*

C. Tomba, F. Maechler, A. Trushko, I. Di Meglio, N. Chiaruttini, A. Roux

University of Geneva, Department of Biochemistry, Switzerland

Deformation of flat epithelia into a given shape is specific to each organ and its function in the organism. For instance, during the gut formation, an initially smooth gut tube is formed and then intestinal villification can take origin by muscle constriction (Shyer et al., Science, 2013). Once the villi shape is established, it is maintained throughout of life. Therefore, gut cells have to function in regions of different curvature. Despite of the growing evidence of the interplay between external forces, mechanotransduction and organ morphology, little is known about cell adaptation to external geometrical constraints. For this study, we have developed two complementary techniques to control epithelium curvature and to investigate its possible role in cell growth and organisation. In the first case we induce an initially flat epithelium on PDMS substrates to deform into a given curvature; in the second case we investigate epithelial cell growth encapsulated in alginate tubes. These systems have the advantage to provide simple tools to control the physical cell environment and to isolate the effects of its properties on cell growth. In particular, our researches focus on quantitative studies of epithelial monolayer adaptation, e.g. in terms of cell shape and proliferation.

Poster 3

Lipid membrane properties during cell adhesion and migration.

Adai Colom, Pau Guillamat, Caterina Tomba, Stefan Matile, Aurélien Roux.

University of Geneva, Department of Biochemistry, Switzerland

Cells and organelles are delimited by lipid bilayers, which high deformability is essential to many cell processes such as motility, endocytosis and cell division. During these deformations, lipid membranes experience stretches causing membrane tension. Membrane tension is thereby a major regulator of membrane remodeling cell processes, albeit very hard to measure in vivo. Here we show that using a planarizable push-pull fluorescent probe called FliptR (Fluorescent LIPid Tension Reporter) we can monitor changes of membrane tension and lipid packing during cell adhesion and migration.

Poster 4

Genome Wide Mutagenesis strategies in Dictyostelium discoideum and Mycobacterium marinum to decipher the conserved genetic basis of mycobacteria intracellular infections.

Louise H Lefrançois[1], Tom Mendum[2], Rachel Shrimpton[2], Frédéric Burdet[3], Marco Pagni[3], Graham Stewart[2] and Thierry Soldati[1].

[1] University of Geneva, Science II, Switzerland.
[2] FHMS, University of Surrey, Guildford, United Kingdom.
[3] Swiss Institute of Bioinformatics, Lausanne, Switzerland.

This study aims at further developping Dictyostelium discoideum - Mycobacterium marinum as a powerful genetically tractable host-pathogen model to investigate both partners during the infection. The central feature of mycobacteria infection is an ability to control the activities of phagocytic cells. Therefore, some of our scientific concerns are to understand how 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? To answer these questions, we have applied Transposon sequencing (Tn-Seq) in M. marinum, an unbiased, genome-wide strategy that combines saturation insertional mutagenesis and high throughput sequencing. This approach allows us to precisely identify the localization and relative abundance of insertions in pools of Tn mutants with a high dynamic range. The essentiality and fitness cost, in terms of growth advantage and disadvantage of over 105 mutants are quantitatively compared before and after in vitro and in vivo selections, in D. discoideum and in macrophages. Our first results using Tn-Seq in the M. marinum strain M, allowed us to identify that ~ 10% of the genes are essential for growth in broth. In addition to those, we have identified ~ 9% additional genes essential to survive 48 hours during infection of D. discoideum, including well known virulence factors (e.g ESX1, PDIM, mce …). In a recent effort, we are expanding our use of the Tn-Seq method to highlight pathways involved in drug resistance, by performing selection of the Tn pool in the presence of different drugs in broth and during infection of D. discoideum. These promising and innovative approaches will allow a comprehensive definition of the host and pathogen genes important for the intracellular interactions during infection in D. discoideum and macrophages.

Poster 5

Implication of TRPC1 in SOCE activated by the new STIM1L isoform.

Agnieszka Dyrda, Maud Frieden

University of Geneva, Switzerland, Dept. of Cellular Physiology and Metabolism.

Depletion of the endoplasmic reticulum (ER) Ca2+ store leads to a Ca2+ entry called store-operated Ca2+ entry (SOCE), which is due to the activation of Orai1 channel gated by STIM1. Our laboratory identified a new splice variant of STIM1, called STIM1L (long) that has an extra 106 aa in the C-term part. We documented that STIM1L is as efficient as STIM1 in eliciting SOCE, but still very little is known about the channel(s) gated by STIM1L. To answer this question, the whole-cell configuration of patch clamp technique and fluorescent recording of intracellular calcium fluctuations using Fura2-AM dye were applied. Currents recorded in HEK293T cells transiently overexpressing Orai1 and STIM1 or STIM1L proteins unexpectedly reviled also the outward currents which together with the values of the reversal potentials point to the participation of other channels than Orai1 in the process. Our strong candidate was TRPC1 and its involvement in ER refiling was tested next. Indeed, elimination of TRPC1 reviled the typical for calcium release-activated current (CRAC) signature of the I/V curve, strongly suggesting the participation of endogenous TRPC1 in intracellular Ca2+ stores refilling. These results were confirmed using Fura2-AM measurements. Based on the above we conclude that TRPC1 actively participate in SOCE phenomenon and the activation of the non-selective cationic current is responsible for maximal activation of SOCE mediated by STIM1L isoform. As these molecules are also expressed in muscle tissue, it remains to be determined whether they are also involved, together with other TRPCs, in human skeletal muscle SOCE. Keywords: TRPC1 channel; STIM1 isoform; store-operated Ca2+ entry; patch-clamp; calcium imaging. Funding: Swiss National Foundation (Grant 310030_166313), FSRMM, Foundation Marcel Levaillant. References: Darbellay B et al, 2011, JCB Antigny F et al., 2017, BBA

Poster 6

From the trench to the open-field battle: Insights from an immune-excluded model of gastric cancer

Viola Puddinu[1], BetülTaşkoparan[1], Nathalie Steinhoff[2], and Carole Bourquin[1].

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Gastric cancer is the fifth most frequent type of cancer worldwide and the third leading cause of cancer-related death. In the last years immunotherapy, which aims to activate the immune system against tumors, has shown great success in treating certain types of advanced cancer and constitutes a promising approach to treat patients with gastric cancer. Nevertheless, only 40 % of patients respond to immunotherapy, underlying the need to better understand the mechanisms of anticancer immunity. Among other factors, the pre-existing activation of the immune system against cancer is necessary for a good response. The most responsive patients have so called “inflamed” tumors, which are infiltrated with immune cells, such as T lymphocytes. Other patients have “immune-excluded” cancers, where T cells are confined to the boundaries of the tumor, leading to reduced responsiveness. Since the presence of T cells in tumors is critical, it is important to use proper models to study the recruitment of T cells to immune-excluded tumors. We work with SV40 T Ag transgenic mice, which develop spontaneous tumors in the stomach. Tumors in these mice grow gradually from premalignant lesions to invasive carcinoma, as is usually the case in patients. Using flow cytometry and fluorescence microscopy, we found that immune cells, including T cells, preferentially localize in the tumor-surrounding stroma rather than in the center of the tumor, indicating that these mice constitute a valid preclinical model to study immune-excluded gastric cancer. We are now investigating treatment modalities to enhance T-cell recruitment to the tumors in these mice.

Poster 7

An integrative fluorescence microscopy approach to reconstruct the 3D architecture of protein complexes in vivo.

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The structural characterization of protein complexes in their native environment is challenging but crucial for understanding the mechanisms that mediate cellular processes. We developed an integrative approach to reconstruct the 3D architecture of protein complexes in vivo. We applied this approach to the exocyst, a hetero-octameric complex of unknown structure that is thought to tether secretory vesicles during exocytosis with a poorly understood mechanism. We engineered yeast cells to anchor the exocyst on defined landmarks and determined the position of its subunits termini at nanometer precision using fluorescence microscopy. We then integrated these positions with the structural properties of the subunits to reconstruct the exocyst together with a vesicle bound to it. The exocyst has an open hand conformation made of rod-shaped subunits that are interlaced in the core. The exocyst architecture explains how the complex can tether secretory vesicles placing them in direct contact with the plasma membrane.

Poster 8

Uncovering the interplay between the growth-promoting transcription factor SFP1 and the stress-responsive transcriptional activator MSN2.

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The Split Zinc Finger protein SFP1 is known to be the main orchestrator of the growth transcription program in the budding yeast Saccharomyces cerevisiae. Using the anchoring away technique, we were able to assess the immediate transcriptional consequences at a genome wide level by RNA pol II Chipseq. After anchoring away SFP1 for 20 minutes, ribosome biogenesis (Ribi) and Ribosomal protein (RPG) genes are strongly downregulated whilst a large subset of genes of the stress response program governed by MSN2 are upregulated. Anchoring away MSN2 revealed the exact opposite trend: stress genes were downregulated whilst Ribi and RPGs were mostly upregulated. In addition, Chromatin endogenous cleavage (ChEC) experiments revealed an opposite genome wide occupancy of SPF1 and MSN2. Measuring H3k9Ac and H4Ac levels genome wide showed that there is a strong correlation between RNA pol II and histone acetylation levels as SFP1 is anchored away. SFP1 has been shown to interact with Tra1, the common subunit of histone acetyltransferase SAGA and NuA4. In addition, the activation of Tra1 is MSN2 dependent. We hypothesize that Tra1 is a common co-activator of SFP1 and MSN2, thus preventing the simultaneous activation of growth promoting and stress response programs.

Poster 9

Analysis of the adhesion-mediated control of insulin secretion in response to glucose and autocrine insulin/IGF2-signaling in pancreatic β-cells.

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Elevated basal insulin secretion under fasting conditions and insufficient glucose-stimulated insulin secretion (GSIS) by β-cells of pancreatic islet are important hallmarks of diabetes. Recent data proposed an improvement of β-cell function (GSIS and survival) when β-cells are anchored to the pancreatic tissue, called extracellular matrix (ECM), by β1-integrin-containing focal adhesions (FA). Integrin-dependent FAs are sites of mechanical linkage between the ECM and the actin cytoskeleton at cell membrane. To these FAs, adapter proteins such as focal adhesion kinase (FAK), and paxillin are recruited to mediate intracellular signaling. We have previously shown that glucose-induced remodeling of the actin cytoskeleton and FAs is involved in the regulation of insulin secretion in β-cell. However how adhesion are regulated is still unknown. We wondered whether the autocrine release of insulin/IGF2 itself could be a FAs modulator. Indeed, insulin/IGF2 signaling appears to be an important FA regulator in other cell types. Although the autocrine role of insulin/IGF2 on insulin secretion is still controversial, few evidences suggest glucose intolerance and elevated insulin secretion during fasting is prevent by activation of Insulin/IGF1 receptors (IR/IGF1R) signaling. We have studied the involvements of IR/IGF1R-PI3K-AKT signaling in the FAK-paxillin-mediated β-cells secretory functions. Our results showed that the activation of the IR/IGF1R-PI3K-AKT1 pathway enhances GSIS by a partial modulation of the adhesion remodeling. In contrast, under low-glucose condition (fasting condition), the IR/IGF1R-PI3K-AKT2 pathway blocks insulin secretion, while regulating FAK-paxillin-dependent FA signaling and associated F-actin remodelling. Interestingly, we have also confirmed that basal insulin secretion regulation is a FAK dependent signaling. Our data propose that autocrine insulin/IGF2-mediated signaling forks at the AKT1 and AKT2 level to mediate adhesion specific signaling in a glucose dependent manner to regulate insulin secretion. A potential dysfunction of the IR/IGF1R-AKT1/2-adhesion signaling pathway could explain why diabetic patients exhibit hyperinsulinemia under fasting conditions and insufficient GSIS.

Poster 10

Effets du vent et de la viscosité sur les ondes océaniques : réduction à un dynamique 2D presented at the 20th Rencontre du Non-linéaire.

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The study of ocean waves is currently focused either on their statistical properties or on the experimental detection of specific solutions of universal models such as the nonlinear Schrödinger equation (NLS) or its generalizations (High-order NLS, HONLS), with the aim of assessing their validity and limits. The recently developed model including dispersive forcing and damping is considered. We propose a truncated three-wave mode, which allows us, for a single unstable mode, to derive a dynamical system for the Stokes' wave, its sidebands, along with the difference between the latter and their total energy. For small perturbations we can trace the resulting evolution on a one degree-of-freedom phase portrait. By exploring the space of parameters, we conclude that viscosity can either give an energy loss and destroy the homoclinic structure of the NLS with a doubling in the period of recurrence, or, thanks to the interplay with a strong wind, may also correspond to an energy growth (on a limited bandwidth) and to the attraction towards singly periodic recurrence orbits inside the separatrix, these latter associated to a permanent down-shift. The phase-space topology of HONLS allows us to characterize the relative impact of wind and viscosity in the most advanced water tank facilities.

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Poster 11

United we stand divided we fall: Chlamydomonas strategy to endure micropollutant toxicity

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Anthropic impact on aquatic environments results in the release of chemicals in water bodies; such micropollutants (MPs) represent a threat to aquatic organisms. Phytoplankton play a key role in biogeochemical cycles of elements and toxic effects of micropollutants on these organisms may have important consequences on the entire aquatic food web. Phytoplankton have evolved multiple strategies to maintain cellular homeostasis and endure adverse environmental conditions however, still little is known about micropollutant induced stress response pathways and phenotypic plasticity under exposure to chemical stress. In our work, we investigate how, in response to MP exposure, the motile green alga Chalmydomonas reinhardtii switches from unicellular to colonial lifestyle with the formation of small clusters of cells called palmelloids. Despite the deep knowledge of C.reinhardtii cell biology, the mechanisms that drive palmelloid colony formation were poorly investigated. Results obtained so far revealed that palmelloid formation is induced by the presence of sub-lethal MP (Copper, Cadmium, Paraquat, PFOS) concentrations. Microscopic observations highlighted the presence of multiple cell wall-like envelopes and that palmelloid formation is associated to the retention of the daughter cells within the mother cell wall. Cells keep growing and dividing within the palmelloid colony when MPs are present, whereas cells revert to the unicellular lifestyle when colonies are harvested and resuspended in pristine medium. Palmelloid formation is not associated to cellular toxic effects, suggesting that cells within the colony are protected from the MP toxicity. Altogether, our results indicated that palmelloid formation is a cellular response to stress that allows C. reinhardtii to face adverse environmental conditions. C. reinhardtii is considered the unicellular reference for the investigation of the evolution of multicellularity in the plant kingdom. The mechanistic investigation of palmelloid colony formation is expected to have far-reaching evolutionary implications.

Poster 12

Structural Plant Biology

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Plants are non-sessile organisms meaning they must adapt to their environment wherever that might be. They must be aware of their surroundings by integrating various inputs to control their growth and development. In our lab we are interested in: (1) cell-surface signaling controlling plant growth, development, response to pathogens and interactions promoting symbiosis, (2) inositol pyrophosphate-mediated signaling and polyphosphate synthesis and degradation in plants, and (4) UVB-signal transduction. We combine an integrated genetic approach with quantitative biochemistry to shed light on how plants have adapted to colonize almost every environment on the planet.

Poster 13

Asymmetric Mechanics during Asymmetric Cell Division of the Sensory Organ Precursors

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In Drosophila Sensory Organ Precursors (SOP), asymmetric cell division gives rise to two daughter cells with different cell size, the PIIB being smaller than its sibling the PIIA daughter cell. Most of the studies of cytokinetic mechanics focus on the equatorial constriction ring and the positioning of the spindle, however an actomyosin cortex is also present at the poles of dividing cells. We show that in wild type SOP, an asymmetry of cortical actin is built during division, with more cortical actin in the bigger PIIA daughter. By using nanobody targeting experiments, we investigate how this polar actin cortex regulates cell shape to generate a proper asymmetric cytokinesis in SOPs. We demonstrate that this polar cortex has a big contribution to the final cytokinesis shape and that perturbing this asymmetry of cortical actin leads to daughter cell size inversion.

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