

2026

LOUIS-JEANTET SYMPOSIUM

Membrane Lipids and Quality Control



PROGRAMME

- 8:15 – 8:50** **Registration and welcome coffee**
- 8:45 – 8:50** **Antoine Geissbühler**, *Dean of the Faculty of Medicine, University of Geneva*
Opening
- 8:50 – 8:55** **Denis Duboule**, *President of the Board of Trustees of the Louis-Jeantet Foundation*
Welcome
- 8:55 – 9:00** **Gisou van der Goot and Giovanni D'Angelo**, *EPFL, CH*
Introductory Remarks

- Chair** **Marlen Knobloch**, *University of Lausanne, CH*
- 9:00 – 9:30** **Scott Dixon**, *Stanford University, USA*
Membrane lipids and non-apoptotic cell death
- 9:30 – 10:00** **Andreas Linkermann**, *Heidelberg University, DE*
Control of Ferroptotic Cell Death Propagation
- 10:00 – 10:30** **Prisca Liberale**, *ETH Zürich, CH*
Lipid territories gate cell fate by modulating signalling decoding

10:30 – 11:00 COFFEE BREAK

- Chair** **Wanda Kukulski**, *University of Bern, CH*
- 11:00 – 11:30** **Aurelien Roux**, *University of Geneva, CH*
Molecular basis for cellular compartmentalization by an ancient membrane fission mechanism
- 11:30 – 12:00** **Franck Perez**, *Institut Curie/CNRS, Paris, FR*
Entering and leaving the endoplasmic reticulum years

12:00 – 13:30 LUNCH

- Chair** **Miriam Stoeber**, *University of Geneva, CH*
- 13:30 – 14:00** **Alf Honigsmann**, *Technical University Dresden, DE*
Lipid transport across the enterocyte brush-border membrane
- 14:00 – 14:30** **Marja Jäätelä**, *Danish Cancer Institute and University of Copenhagen, DK*
Targeting lysosomal membranes in cancer therapy
- 14:30 – 15:00** **Mikael Simons**, *Technical University Munich, DE*
Innate immune and metabolic regulation of CNS remyelination

15:00 – 15:30 COFFEE BREAK

- Chair** **Omayya Dudin**, *University of Geneva, CH*
- 15:30 – 16:00** **Ed Tate**, *Imperial College London & The Francis Crick Institute, UK*
Protein lipidation: new technologies and therapeutic opportunities
- 16:00 – 16:30** **Diana Pinheiro**, *IMP Vienna, AT*
Embryonic Origami: The forces shaping the vertebrate gastrula
- 16:30 – 16:40** **Gisou van der Goot and Giovanni D'Angelo**, *EPFL, CH*
Concluding remarks

ORGANISING COMMITTEE



Gisou van der Goot

EPFL, CH



Giovanni D'Angelo

EPFL, CH

THE LOUIS-JEANTET SYMPOSIUM

First held in 2012, the Louis-Jeantet Symposium is chaired each year by leading scientists — former Louis-Jeantet Prize winners, members of the Scientific Committee, or other distinguished researchers — who, together with invited speakers, highlight advances in their fields and discuss current challenges. These symposia offer

a unique opportunity for Master's students, PhD students, and post-doctoral researchers to engage with leading scientists and expand their networks. Over the years, the programme has explored a wide range of topics, including cancer, stem cells, immunity, developmental biology, and mRNA biology.

THE LOUIS-JEANTET FOUNDATION

Founded in 1983 in Geneva, the Louis-Jeantet Foundation advances medicine by supporting innovative projects in both fundamental and clinical research. Today, it is recognised as one of Europe's leading foundations in this field.

The Foundation awards the Louis-Jeantet Prize for Medicine and the Collen-Jeantet Prize for Translational Medicine, which recognise outstanding researchers working in member countries of the Council of Europe. These

prizes are not intended to crown completed work, but rather to foster the continuation of promising projects across all areas of the life sciences related to human health.

Deeply rooted in its home city, the Foundation also provides annual funding to the Faculty of Medicine at the University of Geneva, enabling the recruitment of full professorial and tenure-track positions and strengthening local biomedical research.



**Scott J. Dixon**

Stanford University, USA

Membrane lipids and non-apoptotic cell death

How lipids contribute to the execution of cell death remains poorly understood. In our work we have found that polyunsaturated phospholipids play a key role in the execution of ferroptosis, an iron-dependent form of non-apoptotic cell death. More recently, we have identified a series of structurally related molecules that can induce non-apoptotic cell death, via either ferroptosis or another lipid-dependent mechanism that is distinct from ferroptosis. These new lethal molecules share an overlapping requirement for the protein S-acyltransferase ZDHHC5, and the lipid metabolic enzyme trans-2,3-enoyl-CoA reductase (TECR) to cause cell death. TECR is canonically involved in the synthesis of very long chain fatty acids but, in this context, seems to promote non-apoptotic cell death by synthesizing a saturated long-chain fatty acid, palmitate. We speculate that this palmitate is used by ZDHHC5 and related enzymes to palmitoylate certain membrane proteins that drive non-apoptotic cell death. I will present our latest insights into the nature of these unusual lethal-dependent mechanisms and how this is altering our view of the role of membrane lipids in cell death regulation.

Biography

Scott is a Professor in the Department of Biology at Stanford University. As a postdoctoral fellow at Columbia University, he trained as a cancer cell biologist and chemical biologist in Brent Stockwell's lab. Scott has made several important contributions to the field of cell death, including the discovery of ferroptosis. His lab is currently focused on the relationship between membrane lipids and non-apoptotic cell death. In the lab, Scott currently mentors a mix of twelve graduate students, postdoctoral fellows and clinical fellows. Outside Stanford, Scott serves on the CCB study section at NIH and co-organizes the FASEB ferroptosis meeting.

www.dixonlaboratory.com





Andreas Linkermann
Heidelberg University, DE

Control of Ferroptotic Cell Death Propagation

Necrotic zones in tissues occur in a wide variety of diseases. Ferroptosis, an iron-promoted necrosis driven by lipid peroxidation, has been identified as a key cell death modality in these conditions. Cells undergoing ferroptosis are unique in that they can induce death in their neighbours. I will discuss salient aspects of ferroptosis propagation on the molecular, cellular and tissue levels. Cell death propagation is restricted by several ferroptosis-suppression systems. Glutathione peroxidase 4 (GPX4) and ferroptosis-suppressor protein 1 (FSP1) are now well established as master regulators, but various additional systems dictate ferroptosis sensitivity. I will discuss how these mechanisms contribute to the suppression of cell death propagation, and how they cause various tissues to be more or less resistant to ferroptosis propagation. Understanding tissue-specific dynamics is critical to interpret the beneficial effects and limitations of future therapeutic approaches for ferroptosis-driven diseases.

Biography

Andreas Linkermann is a German nephrologist and professor of internal medicine . Since October 2024, he has held the Chair of the 5th Department of Internal Medicine at Heidelberg University Hospital – UK Mannheim GmbH , part of the Mannheim Faculty of Medicine at Heidelberg University . His research focuses on regulated cell death, particularly ferroptosis and necroptosis , and he is a Fellow of the American Society of Nephrology (FASN).

<https://www.umm.de/v-medizinische-klinik/forschung/lab-members/linkermann-lab/>





Prisca Liberali
ETH Zürich, CH

Lipid territories gate cell fate by modulating signalling decoding

Many tissues are organized into supracellular structures that emerge through coordinated cell fate decisions that maintain the balance between cell proliferation and differentiation. This balance is directed by multiple morphogens. Yet, how continuous and fluctuating morphogen signals generate robust and spatially confined tissue architectures remains unclear. Here, we show that lipid composition contributes an additional spatial layer regulating signalling decoding during tissue morphogenesis. Using spatial lipidomics and high-content imaging in the mouse intestine and intestinal organoids, we uncover spatially segregated lipid territories that align with crypt–villus domains. These territories emerge dynamically during regeneration and reflect shared morphogen-driven states rather than cell-type identity. Among these, the territory identified by the (glyco)sphingolipid GM1 emerges as a defining feature of the crypt domain and a central regulator of morphogenesis. We show that Wnt signaling transcriptionally regulates GM1 biosynthesis, establishing the first direct link between a morphogen gradient and the formation of a supracellular lipid territory across cells. Uniform expansion of GM1 beyond its endogenous domain altered crypt–villus organization and impaired differentiation without altering the spatial restriction of the Wnt gradient itself. Mechanistically, loss of GM1 spatial confinement disrupted signaling compartmentalization across the crypt–villus axis, resulting in sustained YAP activity, altered TGF β /MAPK signaling, and persistence of a progenitor-like state. Together, our findings identify lipid territories as a supracellular layer of organization that gates how cells interpret signals to coordinate cell fate decisions with tissue architecture. More broadly, this work suggests that membrane lipid composition functions as an interface linking morphogen signalling to transcriptional and mechanical states during tissue morphogenesis.

Biography

Prisca Liberali trained initially as a physical organic chemist before transitioning to molecular cell biology for her PhD. During her postdoctoral work, she pioneered single-cell methods to explore cell-to-cell variability and its impact on complex cellular behaviors. Currently, she is a group leader at the Friedrich Miescher Institute and Professor at ETH Zürich's Department of Biosystems Science and Engineering (D-BSSE). Her research investigates self-organisation in collective cell behaviors, particularly how biochemical and mechanical cues drive coordinated tissue organization across different biological scales. Prisca's lab combines innovative experimental, quantitative, and statistical methods to unravel the principles underlying tissue architecture. She has received multiple awards, including the EMBO Gold Medal, the Suffrage Science Award, and the 2024 Basel City Science Prize.

<https://liberalilab.org/>



**Aurelien Roux**

University of Geneva, CH

Molecular basis for cellular compartmentalization by an ancient membrane fission mechanism

The emergence of intracellular membrane compartments depends on membrane fission. In eukaryotes, membrane fission often relies on ESCRT-III, a protein complex conserved across all domains of life. However, the molecular mechanism by which ESCRT-III drives membrane fission and whether fission represents an ancestral ESCRT-III activity predating eukaryogenesis remain unknown. Here we show that ESCRT-III_A from *Heimdallarchaea*, the closest relatives of eukaryotes, drives membrane fission through insertion of an N-terminal amphipathic helix, named Hofund, into the membrane. The Vps4 ATPase remodels ESCRT-III_A oligomers upon ATP hydrolysis to generate dynamic local constrictions that couple Hofund-mediated membrane insertion to fission. In eukaryotes, Hofund-like function is partitioned across A-type ESCRT-III paralogs, and fission-defective mutants of yeast A-type ESCRT-III subunits can be rescued by the *Heimdall* Hofund motif. Therefore, ESCRT-III-mediated membrane fission by coupling membrane constriction to membrane insertion likely arose in ESCRT-III_A of an archaeal ancestor of eukaryotes, revealing an ancient mechanochemical principle linked to the emergence of eukaryotic intracellular compartmentalization.

Biography

Aurélien Roux studied biology at the Ecole Normale Supérieure de Lyon, France. He received his PhD in 2004 studying how lipids can be sorted by membrane curvature in collaboration with Patricia Bassereau and Bruno Goud. He then moved to Yale for his post-doctoral work with Pietro de Camilli, reconstituting *in vitro* dynamin mediated membrane fission, which he continued as a CNRS staff scientist. He was appointed assistant professor of Biochemistry in Geneva in 2010 where he is now Full Professor.

<https://www.orelrouxlab.org/>



**Franck Perez**

Institut Curie/CNRS, Paris, FR

Entering and leaving the endoplasmic reticulum years

To regulate their homeostasis, cells need to address specific proteins and lipids toward particular compartments in a tightly controlled manner. Golgi-dependent trafficking plays a crucial role in these processes. Although our understanding of the mechanisms of intra-Golgi transport and sorting has significantly improved over the past two decades, the underlying complexity of transport routes, encompassing mechanisms and dynamics, is only beginning to be revealed.

Over the years, we have developed several versatile assays to monitor and quantify the transport of cargoes to the cell surface, including the RUSH assay and the CATCHFIRE system. These assays serve as powerful tools for studying and perturbing cell dynamics in real-time. Recently, we leveraged these tools to gain a deeper understanding of the translocation of specific secretory cargo within the ER, identifying a delayed translocation pathway linked to the nature of the signal peptide. We also studied the formation of gel-like domains in the ER and the partitioning and export of cargoes to downstream compartments. Last we analyzed how the ER can produce de novo Golgi complexes and identified a novel coordinated "Golgi regulon" gene program.

Biography

Franck Perez is Research Director at CNRS and Chairman of the "Cell Biology and Cancer" Unit of the Institut Curie in Paris. His group is studying intracellular trafficking with an important focus on the development of new methods and tools. For example, his group created a novel trafficking assay (RUSH) which is now a standard in the field and a novel fluorogenic capture system (CATCHFIRE). He strongly invested in the development of recombinant antibodies and developed a library based on a humanized nanobody scaffold. He also has a strong technological transfer activity, filed a dozen patents, and co-funded several biotech.

<https://institut-curie.org/person/franck-perez>



**Alf Honigmann**

Technical University Dresden, DE

Lipid transport across the enterocyte brush-border membrane

For efficient uptake and processing of dietary lipids in the intestine, enterocytes must rapidly route absorbed lipids from the apical membrane to the endoplasmic reticulum (ER). While uptake of free fatty acids has been extensively studied, the mechanism how complex lipids traverse the apical cytoplasm to reach the ER remains unresolved. Here, we use bifunctional lipid imaging in enterocyte tissue culture to quantify the transport kinetics and specificity of bile-acid-solubilized phospholipids. We find that phospholipids reach the ER within seconds after apical delivery, suggesting a non-vesicular transport route. Using whole cell FIB-SEM we found no direct contacts between the apical membrane and the ER, consistent with the known exclusion zone for organelles formed by the dense sub-apical cytoskeleton. To identify the involved transport machinery, we are combining lipid imaging with genetic perturbations. Together, our results suggest that an unconventional, non-vesicular, non-contact-side, long-range transport mechanism underlies apical lipid homeostasis in enterocytes.

Biography

Alf Honigmann studied molecular cell biology at the University of Osnabrück and obtained his PhD in experimental biophysics working on lipid phase separation in the group of Richard Wager in 2010. He then joined the group of Stefan Hell at the MPI for Biophysical Chemistry in Göttingen to study cell membrane organization using super-resolution microscopy. In 2015 he became an independent group leader at the MPI of Cell Biology and Genetics in Dresden, where he worked on cell adhesion and membrane organization. Since 2022 he is full professor for biophysics at the Technical University of Dresden.

<https://tu-dresden.de/cmcb/biotech/forschungsgruppen/honigmann>



**Marja Jäättelä**

Danish Cancer Institute and University of Copenhagen, DK

Targeting lysosomal membranes in cancer therapy

Being originally discovered as cellular recycling bins, lysosomes are today recognized as versatile signaling organelles that control a wide range of cellular functions that are essential not only for the well-being of normal cells but also for malignant transformation and cancer progression. In addition to their core functions in waste disposal and recycling of macromolecules and energy, lysosomes serve as an indispensable support system for malignant phenotype by promoting cell growth, cytoprotective autophagy, drug resistance, pH homeostasis, invasion, metastasis and genomic integrity. On the other hand, malignant transformation reduces the stability of lysosomal membranes rendering cancer cells sensitive to lysosome-dependent cell death. Notably, many clinically approved cationic amphiphilic drugs widely used for the treatment of other diseases accumulate in lysosomes, interfere with their cancer-promoting and cancer-supporting functions, and destabilize their membranes thereby opening intriguing possibilities for cancer therapy. Here, I will discuss the emerging evidence that supports the supplementation of current cancer therapies with lysosome-targeting cationic amphiphilic drugs and the molecular mechanisms by which they disturb lysosomal function and permeabilize lysosomal membranes.

Biography

Marja Jäättelä received MD and DMSc degrees from the University of Helsinki in 1989 and 1990, respectively. Following postdoctoral training in Copenhagen, New York and Ann Arbor, she was appointed the head of the Apoptosis Laboratory at the Danish Cancer Institute in 1997 and promoted to the Head of Cell Death and Metabolism Unit in 2012. She has also served as professor at the University of Copenhagen since 2003. Her research focuses on cellular metabolism and lysosomal biology in cancer, and her work is best characterized as a successful combination of uncompromised and innovative basic science with strong translational interest.

https://icmm.ku.dk/english/research-groups/marja_jaattela/



**Mikael Simons**

Technical University Munich, DE

Innate immune and metabolic regulation of CNS remyelination

Myelin is essential for nervous system function, and its loss, as observed in demyelinating diseases, disrupts axonal conduction and contributes to neurodegeneration. Demyelination triggers tissue damage and activates an innate immune response. While this response can promote tissue repair and remyelination, under certain pathological conditions it becomes dysregulated, resulting in persistent, non-resolving inflammation that impairs remyelination and drives neurodegeneration. I will discuss mechanisms that govern the transition from a self-limiting, regenerative immune response to a chronic, self-sustaining pathological state. To investigate these mechanisms in multiple sclerosis, we generated a high-resolution single-cell molecular and spatial atlas of chronic inflammation. Integrative profiling revealed the molecular landscape of glial and immune cells, their disease-associated states, and the microenvironments in which they reside. At the rims of chronic active lesions, we identified CD8⁺ T-cell niches associated with inflamed, foamy microglia characterized by a prominent interferon response and dysregulated lipid metabolism. We investigated the functional consequences of this altered microglial state in experimental models. Our findings revealed that impaired cholesterol efflux promotes the accumulation of lipid-laden phagocytes, which actively sustain inflammatory processes. I will discuss how disrupted lipid metabolism contributes to chronic active inflammation, how these metabolic changes may interfere with regenerative responses and remyelination.

Biography

Mikael Simons is a Professor for Molecular Neurobiology at the TU Munich, the Site Speaker and Head of Clinical Research & Clinical Trial Unit Munich at the German Centre for Neurodegenerative Diseases in Munich (DZNE/Munich). He is board-certified clinical neurologist with specialized expertise in neuroimmunological and neurodegenerative diseases. The main research focus is on glial biology and neuroinflammation in health and disease.

<https://www.mikael-simons-lab.de/>



**Ed Tate**

Imperial College London & The Francis Crick Institute, UK

Protein lipidation: new technologies and therapeutic opportunities

Our lab develops novel chemical biology approaches to enable drug discovery against post-translational modification (PTM) pathways and intractable targets, with a strong emphasis on enabling new translational opportunities. Our work integrates chemical proteomics, high-throughput screening technologies, and chemical probe discovery to illuminate PTM-driven mechanisms that underpin cancer progression, tumour immune evasion, and infectious disease.

Through systematic interrogation of protein lipidation pathways (e.g. N-myristoylation, S-acylation), we have established programmes focused on oncogenic signalling and anticancer immunity which have advanced from fundamental mechanistic studies to clinical development. For example, our identification of ultrapotent N-myristoyltransferase (NMT) inhibitors with strong anti-proliferative and cytotoxic activity across diverse tumour models has led to collaborations exploring the unique multimodal mechanism of action of NMT inhibition across protein secretion, induction of ferroptosis, mitochondrial respiration and synergy with MYC deregulation, as well as its role in viral infection.

Our interest also extends across new therapeutic modalities, including targeted protein degradation, molecular glues, and antibody-drug conjugates (ADCs), where we develop both mechanistic insights and drug candidates with direct relevance to oncology. Translation of our cancer-focused drug discovery work has been enabled through spinouts including Myricx Bio (<https://myricxbio.com/>), which is pioneering next-generation ADC payloads based on inhibitors of protein lipidation, and Siftr Bio (<https://siftr.bio/>), which is applying our activity-based probe technologies to functional profiling of patient-derived cancer tissues. These companies have raised over \$120 million to take the next step towards impact for cancer patients.

Biography

Ed Tate is GSK Chair in Chemical Biology in the Department of Chemistry at Imperial College London and a Satellite Group Leader at the Francis Crick Institute. His group has pioneered chemical proteomic technologies to interrogate post-translational modifications and enable new therapeutic strategies, leading to major academic, translational and industrial impact. He is Director of the Imperial Centre for Drug Discovery Science and academic founder of Myricx Bio and Siftr Bio.

<http://www.imperial.ac.uk/tate-group/>



**Diana Pinheiro**

IMP Vienna, AT

Embryonic Origami: The forces shaping the vertebrate gastrula

Embryogenesis requires rapid diversification of tissue shapes. The amnion, a critical extraembryonic tissue throughout pregnancy, arises directly from the pluripotent epiblast in primates, and must transition from columnar-to-squamous architectures to shape the amniotic sac. How a common lineage resolves into juxtaposed, yet starkly divergent shapes remains unknown. By combining scalable morphometric analysis, direct force measurement and functional perturbations in human and non-human models, we investigated the biophysical and molecular mechanisms underlying this sharp morphogenetic bifurcation.

Biography

Diana Pinheiro is a cell and developmental biologist studying the interplay between morphogen signalling, cell fate and mechanics during vertebrate gastrulation. Her lab combines the power of the zebrafish embryo, as the main *in vivo* model along with stem cell-derived *in vitro* models for human gastrulation. Diana is a group leader at the Research Institute of Molecular Pathology (IMP) at the Vienna Biocenter in Austria since 2022.

<https://www.imp.ac.at/groups/diana-pinheiro>

