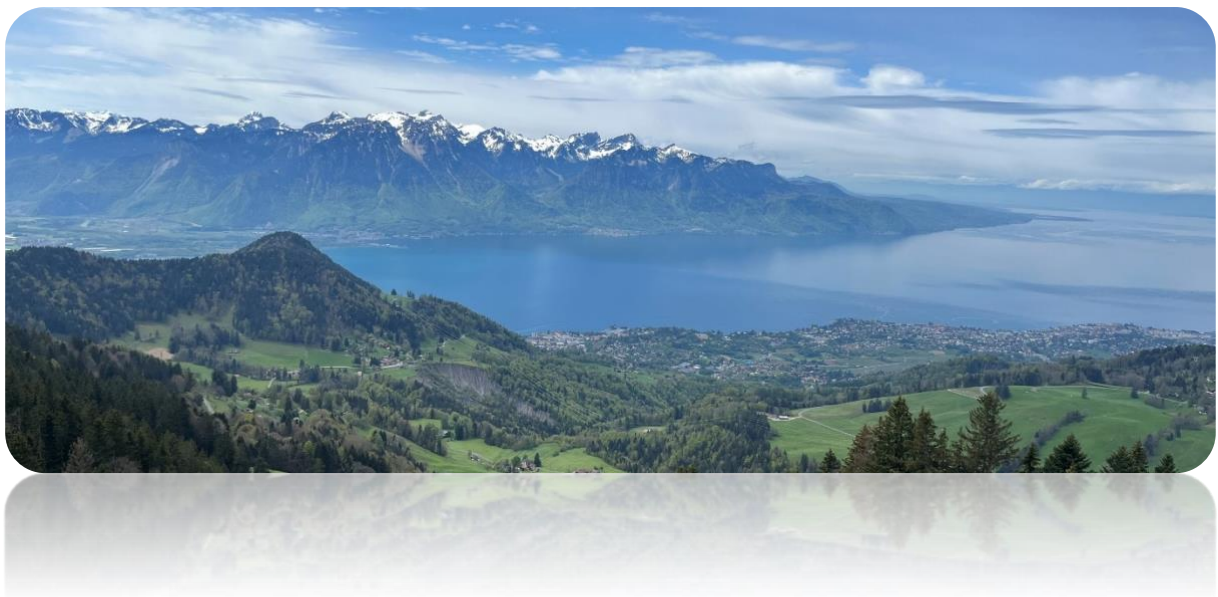


LAC LEMAN REGENERATIVE MEDICINE SYMPOSIUM (L²RM)

17-19 September 2025

César-Roux Auditorium, CHUV Lausanne, Switzerland

<https://www.habiblab.org/l2rm>



Organized by:

Shukry James Habib (DSB-UNIL) and Ulrike Toepel (FBM Doctoral School)

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LAC LEMAN REGENERATIVE MEDICINE SYMPOSIUM (L²RM)

SEPTEMBER 17

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| FROM 15:00 | REGISTRATION |
| 16:45-17:00 | OPENING WORDS BY SHUKRY J. HABIB, DSB-UNIL |
| 17:00-18:00 | SPECIAL KEYNOTE LECTURE: TOM KORNBERG, USCF, USA SIGNALING AT CELL-CELL CONTACTS IN DEVELOPMENT AND DISEASE |
| 18:30-21:00 | CONCERT + APÉRO |

SEPTEMBER 18

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| 09:00-9:35 CHAIR: NADEGE ZANO | INGE HERRMANN, DEPARTMENT OF MECHANICAL AND PROCESS ENGINEERING, ETH ZURICH INNOVATING MEDICAL MATERIALS FOR SURGICAL TISSUE REPAIR |
| 09:35-10:10 | TUSHAR DESAI, STANFORD UNIVERSITY, USA CELL TRANSPLANT THERAPY FOR ACUTE LUNG INJURY |
| 10:10-10:25 | DI CHEN FROM, ZHEJIANG UNIVERSITY EPITRANSCRIPTOMIC REGULATION RESTRICTS THE ENTRY OF HUMAN GERM CELL LINEAGE |
| 10:25-10:40 | COFFEE BREAK |
| 10:40-11:15 CHAIR: TATIANA PETROVA | FRANCESCA SPAGNOLI, KING'S COLLEGE LONDON, UK DECIPHERING MECHANISMS OF BETA CELL DEVELOPMENT FOR REGENERATIVE MEDICINE |
| 11:15-11:50 | LUDOVIC VALLIER, BERLIN INSTITUTE OF HEALTH, CHARITÉ, BERLIN, GERMANY CELL-BASED THERAPY AGAINST LIVER DISEASES AND BEYOND |
| 11:50-12:05 | GAURI RSHIKESAN PADUTHOL, EPFL A BIOENGINEERED HUMAN UROTHELIAL ORGANOID MODEL REVEALS THE URINE-UROTHELIUM INTERPLAY IN TISSUE RESILIENCE AND UPEC RECURRENCE IN URINARY TRACT INFECTIONS |
| 12:05-12:20 | JORGE MADRID-WOLF, READILY3D |
| 12:20-13:30 | LUNCH BREAK |
| 13:30-14:05 CHAIR OLAI A NAVEIRAS | ANJALI KUSUMBE, NANYANG TECHNOLOGICAL UNIVERSITY, SINGAPORE VASCULAR CONTROL OF TISSUE AGEING AND REGENERATION |
| 14:05-14:40 | BART DELPLANCKE, EPFL, LAUSANNE RESOLVING MESENCHYMAL STEM CELL HETEROGENEITY AND FUNCTION, ONE CELL AT A TIME |
| 14:40-14:55 | GABRIELA MICO DESDIN, UNIL, LAUSANNE GENERATION OF NOVEL MOUSE STRAIN FOR THE INDUCTION OF SAFE LONG-TERM HEMATOPOIETIC IN VIVO EPIGENETIC REPROGRAMMING |
| 14:55-15:10 | COFFEE BREAK |

LAC LEMAN REGENERATIVE MEDICINE SYMPOSIUM (L²RM)

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| 15:10-15:45 CHAIR: NICOLA VANNINI | BEATE LICHTENBERGER, MEDICAL UNIVERSITY OF VIENNA, AUSTRIA MODULATING EMBRYONIC SIGNALING PATHWAYS IN FIBROBLASTS TO ADVANCE SKIN REGENERATION |
| 15:45-16:20 | MAXIMINA YUN, CIMR-CHINESE INSTITUTES FOR MEDICAL RESEARCH, BEIJING, CHINA NOVEL MODELS IN AGEING RESEARCH |
| 16:20-17:30 | POSTER SESSION & APERO |
| SEPTEMBER 19 | |
| 09:00-9:35 CHAIR FREDDY RADTKE | ELIF EROGLU, KAROLINSKA INSTITUTE STOCKHOLM, SWEDEN MORE THAN A BARRIER: THE ROLE OF TIGHT JUNCTIONS IN SALAMANDER HEART REGENERATION |
| 09:35-10:10 | WOUTER RICHARD KARTHAUS, SWISS INSTITUTE FOR EXPERIMENTAL CANCER RESEARCH, EPFL, LAUSANNE ANDROGEN MEDIATED PLASTICITY AND REGENERATION IN THE PROSTATE |
| 10:10-10:25 | GIUSEPPE AIELLO, UNIL, LAUSANNE INCREASED REPRODUCIBILITY OF BRAIN ORGANIDS THROUGH CONTROLLED FLUID DYNAMICS |
| 10:25-10:40 | COFFEE BREAK |
| 10:40-11:15 CHAIR: SOPHIE VERRIER | ANGELICA PEREZ FORNOS, WESTERN SWITZERLAND UNIVERSITY COCHLEAR IMPLANTS CENTER, GENEVA UNIVERSITY HOSPITALS AND UNIVERSITY OF GENEVA VESTIBULAR IMPLANTS IN HUMANS, STEPS TOWARDS A CLINICAL APPLICATION |
| 11:15-11:50 | PATRIZIA D'AMELIO, CHUV, LAUSANNE MITOCHONDRIAL HEALTH AND REJUVENATION: UNLOCKING NEW APPROACHES TO AGE-RELATED DISEASES |
| 11:50-12:25 | EILEEN GENTLEMAN, KING'S COLLEGE LONDON & DSB-UNIL MODULAR HYDROGELS FOR ORGANOID-BASED DISEASE MODELLING |
| 12:25-13:30 | LUNCH BREAK |
| 13:30-14:05 CHAIR: NIKLAUS SCHAEFER | JEROME FEIGE, NESTLÉ INSTITUTE OF HEALTH SCIENCES & EPFL, LAUSANNE DISCOVERY & CLINICAL VALIDATION OF NUTRITIONAL ACTIVATORS OF MUSCLE STEM CELLS |
| 14:05-14:20 | CLÉMENCE BECHELLI, UNIL, LAUSANNE DIETARY MACRONUTRIEND REPROGRAMING ENHANCES VASCULAR REGENERATION VIA THE FGF21-ADIPNECTIN AXIS |
| 14:20-15:05 | GIANNI SOLDATI, SWISS STEM CELL FOUNDATION RESISTING COLD DEATH VIA CD38 PATHWAY BY ADIPOSE-DERIVED MESENCHYMAL STEM CELLS |
| 15:05-15:40 | SHUKRY J. HABIB, UNIL, LAUSANNE REGENERATIVE MEDICINE AT THE CROSSROADS: MERGING STEM CELL BIOLOGY, BIOENGINEERING, AND AGING RESEARCH |
| 15:40-15:45 | CLOSING WORDS, SHUKRY J HABIB |

KEYNOTE LECTURE: TOM KORNBERG, USCF, USA
SIGNALING AT CELL-CELL CONTACTS IN DEVELOPMENT AND DISEASE

Development creates a vast array of forms and patterns with elegant economy, using a small vocabulary of pattern-generating signaling proteins (BMPs, FGFs, EGFs, Wnts, and Hhs) in similar ways in many different contexts. Specialized filopodia called cytonemes transport these signaling proteins between cells, mediating cell-cell exchanges at contacts that are functionally similar to neuronal synapses. The roles and physical attributes of cytonemes have fascinating implications for the way signaling proteins disperse across tissues to elicit specific responses in both normal development and disease contexts.

TALK ABSTRACTS

- IN ALPHABETICAL ORDER OF PRESENTER -

PATRIZIA D'AMELIO, CHUV, LAUSANNE

MITOCHONDRIAL HEALTH AND REJUVENATION: UNLOCKING NEW APPROACHES TO AGE-RELATED DISEASES

The talk will explore the critical role of mitochondrial function in the aging process and its implications for geriatric medicine. Aging is a multifaceted phenomenon where biological age, reflecting the physiological state of an individual, can deviate significantly from chronological age. Mitochondrial dysfunction is a central factor in aging, contributing to key processes such as frailty, chronic inflammation (termed "inflammaging"), and immune system decline (referred to as "immunosenescence"). These mechanisms collectively exacerbate physical and cognitive decline in older adults.

The talk will underscore the need for well-designed, large-scale studies to establish optimal protocols for improving mitochondrial function in clinical settings. By addressing frailty, sarcopenia, and cognitive decline through targeted interventions, there is substantial potential to redefine trajectories of aging and enhance the quality of life in older adults.

BART DELPLANCKE, EPFL

RESOLVING MESENCHYMAL STEM CELL HETEROGENEITY AND FUNCTION, ONE CELL AT A TIME

In this presentation, I will first discuss our lab's recent work on resolving adipose tissue plasticity. Specifically, I will focus on a new anti-adipogenic stromal cell type that our lab has discovered, adipogenesis-regulatory cells (Aregs), which sheds new light on the regulation of adipose tissue homeostasis. We have made significant progress in identifying the developmental origin and function of Aregs, including an intriguing phenotypic switch around weaning, whose underlying mechanisms we are currently aiming to unravel.

In a short second part, I will explain how studying adipogenesis led us to develop a technology that allows us to study cellular differentiation heterogeneity, a hallmark of any cell programming process. This method, Live-seq, provides a powerful means to derive transcriptomes of individual cells while keeping these cells alive, thus uniquely enabling us to identify molecular determinants of this heterogeneity.

In the final part, I will discuss our efforts to unravel the regulatory mechanisms of mesenchymal stem cell (MSC) differentiation. I will introduce our newly developed single-cell assay, scTF-seq, which quantifies transcriptomic changes as a function of transcription factor (TF) dose in single cells. This method, using systematic TF overexpression, provides deep insights into TF dose-dependent cell fate determination and cellular reprogramming. I will illustrate this by discussing our scTF-seq atlas for 384 murine TFs in MSCs, highlighting the relevance of scTF-seq data in providing new insights into gene regulation, cellular reprogramming, and developmental biology, while opening new research avenues in tissue engineering.

TUSHAR DESAI, STANFORD UNIVERSITY, USA

CELL TRANSPLANT THERAPY FOR ACUTE LUNG INJURY

Outcomes of acute lung injury span full recovery, survival with residual fibrosis, and death from acute respiratory distress syndrome (ARDS). Patients requiring mechanical ventilatory support are at risk for developing ARDS, in part due to the detrimental effect of positive pressure ventilation. We developed a protocol for direct cellular reprogramming of somatic cells into multipotent embryonic lung progenitors capable of generating both airway and alveolar (gas exchange) epithelial lineages. Transplanting these induced lung progenitors (iLPs) endotracheally into the lungs of mice following severe acute lung injury showed moderate engraftment with regionally appropriate differentiation and significantly improved survival. Remarkably, survivors who received transplant had no residual fibrosis and preserved lung function, in contrast to survivors who did not receive cell therapy. If it can be clinically translated, lung progenitor cell transplant therapy has the potential to transform the care of patients with acute respiratory failure.

ELIF EROGLU, KAROLINSKA INSTITUTE STOCKHOLM, SWEDEN

MORE THAN A BARRIER: THE ROLE OF TIGHT JUNCTIONS IN SALAMANDER HEART REGENERATION

Adult heart is one of the least regenerative organs in the human body, with substantial fibrotic scarring in response to tissue damage. In contrast, aquatic salamanders, such as the newt, display unique heart regeneration capacities. Molecular and cellular basis of this regenerative capacity in the salamanders remains understudied. By combining genetic marker-independent lineage-tracing strategies with transcriptional profiling and loss-of-function methods, we previously reported that the epicardium, the outermost layer of the heart, of the highly regenerative salamander species *Pleurodeles waltl* has an intrinsic capacity to differentiate into cardiomyocytes. We furthermore found that disruption of epicardial tight junctions using the C-terminus of the *Clostridium perfringens* enterotoxin following cryoinjury impairs heart regeneration. We now show that tight junction integrity and remodelling are required for epicardial cell differentiation into cardiomyocytes in the injured heart and propose that tight junction protein Claudin-6 acts as a signaling hub coordinating downstream cellular responses. Salamanders constitute the evolutionarily closest species to mammals with an extensive ability to regenerate heart muscle and our results highlight the epicardium and tight junctions as key targets in efforts to promote cardiac regeneration.

JEROME FEIGE, NESTLÉ INSTITUTE OF HEALTH SCIENCES & EPFL, LAUSANNE

DISCOVERY & CLINICAL VALIDATION OF NUTRITIONAL ACTIVATORS OF MUSCLE STEM CELLS

The regenerative capacity of skeletal muscle relies on the activity of Muscle Stem Cells (MuSCs), a population of resident stem cells that enables myofiber repair following intense exercise or injuries, and contributes to tissue homeostasis and turnover. Aging and muscle diseases impair MuSC function, leading to altered regenerative capacity and progressive loss of skeletal muscle mass and strength. Using a high-content imaging screen of natural molecules, we identified nicotinamide (NAM) and pyridoxine (PN) as potent bioactive nutrients from vitamin B metabolism that synergistically stimulate MuSC activity, while having a history of safe human use. In human clinical cohorts or preclinical models, endogenous levels of NAM and bioactive PN decline during aging and inter-independently associate with muscle mass and walking speed. Oral treatment with a combination of NAM/PN accelerates muscle repair *in vivo* by stimulating MuSCs and enhances recovery of muscle strength. In a human randomized placebo-controlled clinical trial using eccentric contraction-induced muscle regeneration in healthy volunteers, oral NAM/PN supplementation was well tolerated and stimulated the myogenic activity of MuSCs detected via increased number of MyoD and Myogenin positive cells in muscle biopsies during recovery. Collectively, our results establish NAM/PN as a promising nutritional intervention that stimulates MuSCs, enhances muscle repair, and alleviates muscle decline during aging, and opens new opportunities to enhance muscle recovery and manage regenerative capacity in muscle disorders through nutrition.

EILEEN GENTLEMAN, KING'S COLLEGE LONDON & DSB-UNIL

MODULAR HYDROGELS FOR ORGANOID-BASED DISEASE MODELLING

Pathological matrix remodelling plays a central role in many human diseases, but is challenging to study as *in vitro* models often cannot replicate the complex 3D cell-matrix interactions that drive pathologies. In this seminar, I will discuss how we built a 3D model of the human gut that allowed us to uncover an unexpected role for a rare immune cell type called ILC1 in driving gut fibrosis in patients with inflammatory bowel diseases. We used molecular dynamics simulations to design PEG hydrogels that cross-link quickly, but can still mimic the stiffness of normal intestinal tissue. We then co-cultured encapsulated human intestinal organoids with ILC1, and using a combination of atomic force microscopy force spectroscopy and multiple particle tracking microrheology, found that ILC1 drive intestinal matrix remodelling through a balance of MMP9-mediated matrix degradation and TGFβ1-driven fibronectin deposition. Our findings demonstrate the potential of using hydrogels in disease modelling, and open the possibility of unravelling how pathological matrix remodelling contributes to disease.

INGE HERRMANN, DEPARTMENT OF MECHANICAL AND PROCESS ENGINEERING, ETH ZURICH

INNOVATING MEDICAL MATERIALS FOR SURGICAL TISSUE REPAIR

The Nanoparticle Systems Engineering Lab is focused on the design and development of nanoparticle-enabled approaches addressing clinical needs in disease etiology, diagnostics and drug delivery. We engineer nanoparticle-based systems based on design specs defined by clinical needs. By combining experimental and modelling approaches, we strive to obtain a holistic understanding of the materials-biology interplay across scale in order to rationally adjust the materials design. We have expertise in nanoparticle synthesis and functionalization, characterization and multiscale imaging of particle-based systems, as well as translational nanomedicine.

WOUTER RICHARD KARTHAUS, SWISS INSTITUTE FOR EXPERIMENTAL CANCER RESEARCH, EPFL
ANDROGEN MEDIATED PLASTICITY AND REGENERATION IN THE PROSTATE

Androgen deprivation therapy (ADT) or chemical castration is the cornerstone of prostate cancer treatment. In the normal prostate, ADT results in involution of the gland to ~90% of its original size. When androgen levels are restored, the prostate regenerates fully. The molecular effects of ADT and mediators of androgen driven regeneration are not fully understood. In this talk I will discuss recent advances we made in understanding these intricate processes. Using a combination single cell omic techniques, lineage tracing and organoid culture, we gain insight in 1) How differentiated cells acquire progenitor potential during ADT treatment 2) Which paracrine factors drive prostate regeneration and 3) how we can leverage this knowledge to enhance ex vivo culturing approaches.

ANJALI KUSUMBE, NANYANG TECHNOLOGICAL UNIVERSITY, SINGAPORE
VASCULAR CONTROL OF TISSUE AGEING AND REGENERATION

The vasculature plays a crucial role in regulating hematopoietic and bone regeneration. I will present an efficient pipeline for clearing and immunolabeling intact calcified and soft tissues, enabling rapid, high-resolution, single-cell imaging. This approach has allowed us to uncover the presence of lymphatic vessels in bones, challenging the longstanding belief that bones lack a lymphatic network.

Using high-resolution light-sheet imaging and cell-specific mouse genetics, we identified lymphatic vessels in both mouse and human bones. Further investigation revealed that these vessels expand in response to genotoxic stress, driven by VEGF-C/VEGFR-3 signaling and stress-induced IL-6. During lymphangiogenesis, proliferating lymphatic endothelial cells secrete CXCL12, a key regulator of hematopoietic and bone regeneration. Additionally, lymphatic vessel-derived CXCL12 promotes the expansion of mature Myh11+ CXCR4+ pericytes, which differentiate into bone-forming cells and contribute to both bone and hematopoietic recovery. In contrast, blood vascular endothelial cells play a central role in bone ageing and our results show that bone angiogenesis can be exploited to enhance the bone mass.

Our findings suggest that targeting vasculature could serve as a novel therapeutic strategy to enhance hematopoietic and bone regeneration, particularly in the context of stress, injury, and age-related decline in bone mass.

BEATE LICHTENBERGER, MEDICAL UNIVERSITY OF VIENNA, AUSTRIA
MODULATING EMBRYONIC SIGNALING PATHWAYS IN FIBROBLASTS TO ADVANCE SKIN REGENERATION

The skin is highly proliferative tissue with the capability for constant renewal and regeneration, a feature that is essential for survival as the skin forms a protective barrier against external insults and water loss. However, as we age, the skin's function declines, which is a major cause of morbidity and risk factor for skin fragility, infections, and impaired wound healing. Aging-dependent skin alterations are associated with significant changes of the connective tissue and dermal fibroblasts, which display an unprecedented plasticity and heterogeneity.

In adult mammalian skin, every injury will lead to a scar. This scar tissue is usually rigid and lacks elasticity and the skin's original resilience to external impacts, but also secondary appendages such as hair follicles and sebaceous glands. While it was long thought that hair follicles develop solely during embryogenesis, it is becoming increasingly clear that hair follicles can also regenerate within a wound. The ability of the skin to induce hair neogenesis following injury however declines with age. Since fetal and neonatal skin have the remarkable

capability to heal without scarring, the recapitulation of a neonatal state has been a primary target of regenerative research. We highlight how modulating dermal signaling or the abundance of specific fibroblast subsets could be utilized to induce *de novo* hair follicles within the wound bed, and, thus, to shift wound repair with a scar to scarless regeneration.

ANGELICA PEREZ FORNOS, WESTERN SWITZERLAND UNIVERSITY COCHLEAR IMPLANTS CENTER, GENEVA UNIVERSITY HOSPITALS AND UNIVERSITY OF GENEVA

VESTIBULAR IMPLANTS IN HUMANS, STEPS TOWARDS A CLINICAL APPLICATION

Vestibular implants are implantable devices that attempt to partially restore vestibular function to patients with severe bilateral vestibulopathy of peripheral origin, using electrical currents. Our group developed an original concept based on a modified cochlear implant in close collaboration with MED-EL (Innsbruck, Austria). We started implantations in humans in 2007 and, to date, 25 patients with severe bilateral vestibulopathy were implanted with these prototype devices. In this talk, we will cover the main results obtained so far in humans which are very encouraging. We will also discuss the main steps that we have undertaken to achieve a clinical application, hopefully soon.

GIANNI SOLDATI, SWISS STEM CELL FOUNDATION

RESISTING COLD DEATH VIA CD38 PATHWAY BY ADIPOSE-DERIVED MESENCHYMAL STEM CELLS

Adipose-derived mesenchymal stem cells (ASCs) are multipotent progenitors renowned for their ability to self-renew and differentiate into multiple cell types, including adipocytes, osteoblasts, chondrocytes, and myocytes. These cells are pivotal in regenerative medicine due to their extensive proliferation capacity while maintaining multipotency across numerous culture passages. Moreover, MSCs exhibit immunomodulatory properties, making them excellent candidates for addressing inflammatory diseases and facilitating tissue repair. ASCs have become central to preclinical and clinical research owing to their accessibility from diverse tissues and their therapeutic potential in tissue regeneration. Liposuction, a common method for obtaining ASCs yields significantly higher quantities of stem cells compared to bone marrow aspiration. The cryopreservation of ASCs has revolutionized regenerative medicine by ensuring long-term storage while retaining cell viability and functionality. Cryopreservation involves suspending cells in a cryoprotective agent, such as dimethyl sulfoxide (DMSO), to prevent ice crystal formation, followed by gradual controlled cooling and storage in liquid nitrogen at -196°C. This ensures rapid availability for autologous or allogeneic therapies, reducing the need for repeated harvesting and minimizing patient discomfort. Actual cryopreservation protocols preserve the differentiation potential, immunomodulatory properties, and a partial viability and count of post-thawed ASCs. The problem of post-thawing count and viability is much well known throughout all scientific domains and applicative medical fields and new strategies for ameliorating count and viability of post-thawed ASCs are mandatory for the use these cells are promised to, cell therapies. CD38 is a transmembrane glycoprotein involved in NAD⁺ metabolism and its intracellular level regulation and can be considered the main NAD-degrading enzyme in mammalian tissues. It functions primarily as an NAD⁺-dependent enzyme that catalyzes the conversion of NAD⁺ into ADP-ribose and nicotinamide, which are critical in regulating intracellular calcium levels and cellular stress responses. The expression and activity of the NADase CD38 increases with aging in terminally differentiated cells and therefore NAD declines as function of time. We show that CD38 expression is decreased in adipose-derived mesenchymal stem cells submitted to low temperatures when compared to terminally differentiated cell populations exposed to the same conditions. We also show that the death resistance of ASCs is probably due to

the increased intracellular concentration of NAD⁺ derived by the decreased expression of the NADase CD38 and that this resistance is mediated through Sirtuins.

FRANCESCA SPAGNOLI, KING'S COLLEGE LONDON, UK

DECIPHERING MECHANISMS OF BETA CELL DEVELOPMENT FOR REGENERATIVE MEDICINE

Cell identities are assigned by the interplay of extrinsic signals and intrinsic determinants. My research interests focus on the mechanisms regulating cell identity and plasticity in metabolic organs, such as the pancreas. We use multimodal approaches in mouse embryos and human models to elucidate how distinct cell types arise from common progenitors and crosstalk to each other and surrounding microenvironment to acquire specialized shapes and form functional organs. Is the establishment of distinct cellular identities and morphogenetic programs interdependent? How plastic are these cellular states? Can we harness cell plasticity towards novel regenerative therapies for diabetes? Ultimately, the long-term goal of our research is to translate these concepts into a better understanding of the pathogenesis as well as treatment options of diabetes.

LUDOVIC VALLIER, BERLIN INSTITUTE OF HEALTH, CHARITÉ, BERLIN, GERMANY

CELL-BASED THERAPY AGAINST LIVER DISEASES AND BEYOND.

The liver sustains reserves of iron, vitamins and minerals and detoxifies alcohol, drugs and other chemicals. The liver has also a function in synthesis by producing albumin which represents the most abundant protein in the plasma, and blood clotting factors. Finally, the liver has an essential metabolic function by storing glycogen and lipids. Most of these activities are managed by one cell type, the hepatocyte. However, additional cell types such as cholangiocytes also play essential roles in organ repair, tissue homeostasis and diseases. Diseases targeting the liver are life threatening and the only treatment for end-stage disease is organ transplantation. Such therapy entails high risk of surgical complications and indefinite immunosuppression associated with severe side effects. Furthermore, lack of organ donors greatly limits the number of patients who can benefit from this therapy. Thus, cell therapy using primary cells, Hepatocytes or cholangiocytes, have been proposed as an alternative for organ transplantation. However, this approach has been limited since adult primary hepatocytes can be grown only for a limited time in vitro without losing their functional activity. For this reason, production of hepatocytes from human induced pluripotent stem cells (hiPSCs) represent an advantageous option. Here, we will provide an update of the technologies available to produce liver cells in vitro and the different approaches currently available for improving their functionality. We will also describe the proof-of-concept applications in regenerative medicine against liver diseases and beyond.

MAXIMINA YUN, CIMR-CHINESE INSTITUTES FOR MEDICAL RESEARCH, BEIJING, CHINA

NOVEL MODELS IN AGEING RESEARCH

Extensive regeneration of the body plan is found in salamanders such as axolotls and newts, which exhibit the most extensive regenerative repertoire among vertebrates. Complex regeneration relies on the modulation of cellular plasticity for the generation of regenerative progenitors and can be achieved throughout lifespan, in contrast to mammals. Further to this, salamanders display additional noteworthy traits, namely remarkable longevity, defiance of the Gompertz law of mortality and apparent lack of traditional signs of age-related decay, constituting organisms of 'negligible senescence'. As such, they constitute valuable models for addressing the nature of organismal senescence and the interplay between regeneration and ageing.

Here, I will introduce the axolotl as a novel model in ageing research, discuss our lab's efforts towards understanding how salamanders regulate hallmarks of ageing through regeneration and lifespan, and elaborate on the potential of salamander models to illuminate the basis of negligible senescence.