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SoMM2023

6th Symposium of the network on monocytes & macrophages

November 24th 2023 in Paris

Macrophage Clubs

Macrophage Club Île-de-France

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6th Symposium of the Network on Monocytes & Macrophages

Paris – November 24, 2023

Amphithéâtre Vulpian, Université de Paris Cité
12 rue de l'école de Médecine – 75006 Paris

Université Paris Cité

Institut du Diabète

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Organizers: Camille BLERIOT, Elise DALMAS, Emmanuel GAUTIER, Florent GINHOUX, Julie HELFT, & Nicolas VENTECLEF

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6th Symposium of the Network on Monocytes & Macrophages
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PROGRAM

8h30 – 8h55	**Registration**
8h55 – 9h	**Symposium introduction** Camille Blériot (INEM, Paris) & Elise Dalmas (INEM, Paris)
9h – 10h30	**Session 1: Macrophage development and identities** Chairs: Julie Helft (Institut Cochin, Paris) & Camille Blériot (INEM, Paris)
9h – 9h25	Andreas Schlitzer (LIMES Bonn, Germany) *Molecular control of functional specialization of monocyte-derived macrophages*
9h25 – 9h50	Elisa Gomez-Perdiguero (Institut Pasteur Paris, France) *Contribution of macrophage ontogeny to health and disease*
9h50 – 10h15	Melanie Greter (Zurich University, Switzerland) *Regulation and Function of Macrophages at the Maternal-Fetal Interface*
10h15 – 10h30	Selected Abstract : Léa Di Maria (EnVI , Rouen) *Circulating monocytes from patients promote calcific aortic valve disease development*
10h30 – 11h	**Coffee break**
11h – 12h30	**Session 2: Macrophages in inflammation** Chairs: Laurent Yvan-Charvet (C3M, Nice) & Emmanuel Gautier (INSERM, Paris)
11h – 11h25	Johan Garaude (ImmunoConcept, Bordeaux) *Microbial viability orchestrates the metabolic reprogramming of macrophages*
11h25 – 11h50	Florian Sennlaub (Institut de la Vision, Paris) *How genetic risk variants of age-related macular degeneration shape inflammation*
11h50 – 12h15	Renato Ostuni (HSR Milano, Italy) *Transcriptional control of myeloid cell identity and activation*
12h15 – 12h30	*1 minute "Flash" communications* *12 selected abstracts*
12h30 – 14h	**Lunch/poster session**
14h – 15h30	**Session 3: Macrophages in diseases** Chairs: Elise Dalmas (INEM, Paris) & Nicolas Venteclef (INEM, Paris)
14h – 14h25	Stoyan Ivanov (Université Côte d'Azur, Nice) *Brown Adipose Tissue Myeloid Cell Diversity and Functions*
14h25 – 14h50	Alexandre Boissonnas (CIMI, Paris) *Visualizing the spatial organisation in situ of monocytes, septal and parenchymal macrophages by non-linear optical imaging*
14h50 – 15h15	Jerome Martin (CR2TI, Nantes) *Myeloid dysregulation in Crohn's disease*
15h15 – 15h30	Selected abstract: Clara Pierrot (C3M Nice) *Role of mitochondrial dynamics in the regulation of macrophages effector functions in acute and chronic inflammatory diseases*
15h30 – 16h	**Coffee break**
16h – 17h30	**Session 4: Brain macrophages** Chairs: Florent Ginhoux (IGR, Villejuif) & Aymeric SILVIN (IGR, Villejuif)
16h – 16h25	Claudia Pasqualini (IGR, Villejuif) *Microglia-sufficient brain organoids: from neurodevelopment to paediatric oncology*
16h25 – 16h50	Etienne Audinat (IGF, Montpellier) *Microglial TNFalpha increases excitatory synaptic transmission during epileptogenesis by activating purinergic signaling in astrocytes*
16h50 – 17h15	Morgane Thion (IBENS, Paris) *Early invaders of the brain: embryonic colonization and function of microglia*
17h15 – 17h30	Selected abstract: Frédéric Blanchard (RMeS , Nantes & Coll. Montpellier) *Spatial mapping of rheumatoid arthritis synovial niches reveals specific macrophage networks associated with response to therapy*
17h30 – 17h45	**Concluding remarks and departure**

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Selected Abstracts for Oral communication

Selected for communication Session 1

Circulating monocytes from patients promote calcific aortic valve disease development

Léa Di Maria * [1,2], Hugues Boël [1,2], Nicolas Perzo [1,2], Sylvanie Renet [1,2], Bryan Marais [2,3], Solenn Catherine [2,3], Thomas Levesque [1,2,3], Delphine Béziau-Gasnier [2,3], Hélène Eltchaninoff [2,3], Eric Durand [1,2,3], Sylvain Fraineau [1,2]

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Calcific Aortic Valve Disease (CAVD) is the most frequent valvular heart disease in developed countries. It is characterized by a progressive calcification of aortic valve leaflets mediated by myeloid cells, such as monocytes and macrophages. Previous studies have already demonstrated the role of macrophages in CAVD development. However, little is known about the implication of their precursor, monocytes. Thus, we hypothesized that circulating monocytes from CAVD patients have higher pro-calcific and pro-inflammatory capacities
To test this hypothesis, we explored the transcriptome of circulating monocytes from CAVD patients by transcriptomic analysis and assessed the inflammatory and pro-calcific potential of their secretome.

First, we analyzed the whole transcriptome of monocytes isolated from peripheral blood of patients without (NON-CAVD) or with CAVD by RNA sequencing (RNAseq) and RT-qPCR to identify dysregulated genes expression. Monocytes secretome composition was characterized by multiplex analysis and its potential to induce human valvular interstitial cells (hVIC) calcification was measured using O-cresolphtalein assay.

Our RNAseq analysis revealed that NON-CAVD and CAVD monocytes express significant higher and lower levels of genes involved in inflammation (TLR2, CX3CR1) and immunomodulation (IL4, KLF4) respectively. Interestingly, we also identified genes specifically up- and downregulated in monocytes from CAVD patients as respectively involved in inflammation/calcification (PDK4, ATP2B1) and immunomodulation (DDR1, IKBKE). Accordingly, CAVD-monocytes secretome was impoverished in immunoregulatory/anti-osteoblastogenic (IL4, CCL3) cytokines correlating with enhanced secretome-induced hVIC calcification.
We demonstrated that circulating monocytes from CAVD patients have higher pro-inflammatory and pro-calcific capacities that might promote CAVD development. Likewise, we identify genes as new therapeutic targets.

*Speaker

Keywords: Monocytes, Inflammation, Calcified aortic valve disease

Selected for communication Session 3

Role of mitochondrial dynamics in the regulation of macrophages effector functions in acute and chronic inflammatory diseases

Clara Pierrot * [1], Adelie Dumont [1], Lama Habbouche [1], Thibault Barouillet [1], Coraline Borowczyk [1], Béatrice Bailly-Maitre [1], Laurent Yavn-Charvet† [1]

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Introduction: Atherosclerosis is a chronic inflammatory disease of blood vessels and the first etiology of cardiovascular diseases. The accumulation of immune cells in atherosclerotic lesions is dominated by an infiltration of monocyte-derived macrophages, which are recruited to clean and repair the damage tissue (i.e, resolving and reparative functions). However, macrophages face a hostile lipid-rich and oxidative environment that impact their metabolism and subsequent functions.
Hypothesis: Besides clearance and repair functions, macrophage metabolic rewiring is also crucial for the prototype bacterial proinflammatory response. Thus, there is a growing interest in identifying specific metabolic reprogramming to control resolution pathways without risking the efficient metabolic mobilization of macrophages for bacterial infection, and vice versa. In that context, we identified a mitochondrial Rho GTPase (MIRO1, encoded by the RHOT1 gene), which is differentially regulated in inflamed and resolving macrophages.

Methods: We therefore generated mice with specific deficiency of Rhot1 in macrophages (LysM-Cre x Rhot1fl/flmice). These animals were challenged to either acute bacterial infection with *Escherichia coli* or chronic atherosclerosis inflammation. For Atherosclerosis, we transplanted the bone marrow of LysMCre x Rhot1fl/flmice into lethally irradiated Ldlr-/- mice, then fed a high-fat diet for 12 weeks.

Results: We first observed that LysMCre x Rhot1fl/fl mice were less susceptible to acute inflammation as revealed by enhanced phagocytosis, reduced inflammatory burden (i.e, plasma IL-6 and IL-1b levels) and improved survival. In contrast, Ldlr-/- mice transplanted with the bone marrow of LysMCre x Rhot1fl/fl mice developed accelerated atherosclerosis with more macrophage deposition and necrotic core in atherosclerotic plaques. *In vitro* experiments are ongoing to decipher the unique metabolic reprogramming at the origin of this response.
Conclusion: Our findings could have major relevance in the current dogma of the CANTOS trial, in which blocking IL1b signaling pathway limited the development of cardiovascular diseases while promoting more susceptibility to bacterial infection. Thus, this study hints at a therapeutic window to metabolically control macrophage functions.

Keywords: Macrophages, mitochondria, atherosclerosis, chronic, acute, inflammation

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Selected for communication Session 4

Spatial mapping of rheumatoid arthritis synovial niches reveals specific macrophage networks associated with response to therapy

Julien De Lima [1], Marie-Astrid Boutet [1], Olivier Bortolotti [2], Laure-Agnès Chépeaux [3], Yaël Glasson [3], Anne-Sophie Dumé [3], Adrien Le Pluart [4], Alessandra Nerviani [5], Liliane Fossati-Jimack [5], Henri-Alexandre Michaud [3], Jérôme Guicheux [4], Benoit Le Goff [4], Costantino Pitzalis [5], Gabriel Courties [2], Florence Apparailly [2], **Frédéric Blanchard** [*† 6]

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Rheumatoid arthritis (RA) is a chronic inflammatory autoimmune disease affecting peripheral joints and for which approximately 40% of the patients respond insufficiently to the available synthetic or biologic disease modifying anti-rheumatic drugs (DMARDs). The infiltration of the synovial membrane by lymphocytes and monocytes profoundly alters its homeostatic functions, leading to chronic joint inflammation and bone destruction. A better understanding of how DMARDs impact the complex synovial cell social network in relationship to response / non-response remains an unmet need to design more targeted and active therapeutic strategies. Here, we used imaging mass cytometry (IMC) to comparatively profile more than 115,000 cells in the synovial tissue of healthy, low inflammatory osteoarthritis and matched active early treatment-naïve RA patients at baseline and at 6 months after starting DMARDs treatment. We notably highlighted that tissue resident macrophages (LYVE1+CD206+) in perivascular synovial niches encompassing specific subsets of vascular cells, fibroblasts and immune cells vanished in active RA but were recovered in response to DMARDs treatment. Combined ligand-receptor analysis of single-cell RNA sequencing datasets identified that IL10, C-type lectin or TAM (TYRO3, AXL and MERTK) receptors were particularly involved in the restoration of these spatial cell interactions in the context of clinical remission. In addition to providing an unprecedented synovial spatial mapping, our work uncovered novel potential cellular and molecular targets for the development of therapies for RA.

*Speaker

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Abstracts for 1 minute Flash Oral communication (Session2)

Molecular mechanism employed by Human rhinovirus 16 to inhibit phagosome maturation in macrophage

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Alveolar macrophages (AM) survey the lung to prevent microbial infection. Yet, their functions can be impaired and bacterial infection may arise. A key factor to bacterial superinfection in patients with chronic pulmonary diseases are Human rhinoviruses (HRV). We previously demonstrated that HRV16 exposure impairs macrophages functions, among which phagocytosis (entry and maturation). In addition, we showed that the inhibition of phagosome maturation was dependent on a small GTPase of the Arf-like family, hereafter referred to as ARLx.
Here, we aimed to decipher how HRV16 can modulate macrophages responses. Using human monocytes derived macrophages (hMDMs), we showed that HRV16 triggered an interferon (IFN) and pro-inflammatory response in these cells. Interestingly, HRV16-mediated upregulation of ARLx and activation of IFN signaling was dependent on ICAM1, the receptor for HRV16 entry. Whereas ARLx was suggested to be an IFN stimulated gene, we showed here that this is not the case. We showed that HRV16-mediated ARLx induction was dependent on PKR (protein kinase R) using a specific inhibitor. PKR can activate several downstream pathways, among which c-Jun, ATF2 or IRF-3. We showed that ARLx induction by HRV16 was dependent on ATF2. Finally, we observed an increase of the epigenetic positive mark H3K27Ac on ARLx's promoter.
Altogether, we shed light on a new ICAM1-PKR-ATF2 axis regulating important phagocytic clearance function.

Keywords: alveolar macrophage, HRV, interferon, PKR, ATF2

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Talin1-dependent adherence to peritoneal mesothelium supports maturation of monocytes into GATA6+ large peritoneal macrophages.

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Peritoneal macrophages play key roles in health and disease. The major cell type in the peritoneal interstitial fluid of mice is the so-called large peritoneal macrophage (LPM) charaterized by selective dependence on the transcription factor GATA6. LPMs maintain themselves from embryonic progenitors by local proliferation in response to CSF1 produced by mesothelial cells and fibroblasts, but they also slowly repopulate from monocytes. Deletion of GATA6 in macrophages leads to increased apoptosis of LPMs and accumulation of macrophages with a distinct phenotype and transcriptome notably characterized by higher expression of LYVE1. Growing evidence suggests that these LYVE1+ peritoneal macrophages are a transitional state between monocytes and LPM called 'converting peritoneal macrophages' (CPM). However, little is known about the conditions that promote CPM differentiation to LPM. Here, we report that CPMs are tightly associated with peritoneal mesothelial cells in omentum and mesothelial lining of peritoneal organs. Using photoconversion and genetic fate mapping, we could demonstrate rapid trafficking of CPMs to and from omentum. Surgically removing omentum slows down maturation of monocyte derived CPMs into GATA6+ LCMs. Invalidating Talin1, an intermediate in inside-out signaling to activate integrins, also impacts maturation of monocyte-derived cells resulting in accumulation of LYVE1+ CPMs in peritoneal fluid. Importantly, impaired maturation of Talin1-deficient CPMs is highly associated with their incapacity to bind to mesothelium-rich peritoneal regions. Thus, we demonstrate that integrin-mediated interaction of monocyte-derived CPMs with their niche is crucial to support their maturation into GATA6+ LCMs.

Keywords: Peritoneal macrophages, Monocytes, Macrophage development

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Revealing how Intracellular Bacteria hijack Mitochondrial Metabolism during Infection

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As intracellular bacteria can obtain nutrients only from host cells, subverting the metabolism of the infected cell seems critical for these pathogens. We are investigating how the metabolism of human macrophages is altered during infection with intracellular bacteria. We are comparing *Salmonella enterica* Serovar Typhimurium and *Legionella pneumophila* infection of human monocyte-derived macrophages to learn whether the mitochondrial Electron Transport Chain and oxidative phosphorylation is altered during infection and if bacterial effectors play a role.

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Dynamics of macrophage polarization support Salmonella persistence in a whole living organism

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Numerous intracellular bacterial pathogens interfere with macrophage function, including macrophage polarization, to establish a niche and persist. However, the spatiotemporal dynamics of macrophage polarization during infection within host remain to be investigated. Here, we implement a model of persistent *Salmonella* Typhimurium infection in zebrafish, which allows visualization of polarized macrophages and bacteria in real time at high-resolution. While macrophages polarize toward M1-like phenotype to control early infection, during later stages, *Salmonella* persists inside non-inflammatory clustered macrophages. Transcriptomic profiling of macrophages showed a highly dynamic signature during infection characterized by a switch from pro-inflammatory to anti-inflammatory/pro-regenerative status and revealed a shift in adhesion program. In agreement with this specific adhesion signature, macrophage trajectory tracking identifies motionless macrophages as a permissive niche for persistent *Salmonella*. Our results demonstrate that zebrafish model provides a unique platform to explore, in a whole organism, the versatile nature of macrophage functional programs during bacterial acute and persistent infections.

Keywords: Macrophage – Polarization – Salmonella – Bacterial persistence – zebrafish

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Establishment of a primary hamster macrophage system to study susceptibility to leishmania donovani infection

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Macrophages are important innate immune cells that detect and eliminate invading microbes and instruct the immune system for appropriate adaptive responses. *Leishmania* resists macrophage cytolytic activities and exploit these cells as hosts for intracellular proliferation. Chronic infection in humans can be either asymptomatic or causing devastating immunepathologies. The host determinants and immune mechanisms underlying this dichotomy are largely unknown, even though pathways controlling the infection could inform on urgently needed, immune-therapeutic interventions. Mice are reported to control *Leishmania (L.) donovani* infection conversely to hamsters that develop progressive and lethal visceral leishmaniasis. Therefore, we propose to exploit these two rodent systems to investigate the role of *Leishmania*-macrophage interactions in disease outcome. C57BL/6 mice (m) and Golden Syrian hamsters (ham) were used as source of primary macrophages. The heterogeneity of peritoneal exudate cells and the limited number of peritoneal macrophages (PMs) recovered per animal primed us to establish a protocol for hamster bone marrow-derived macrophages (hamBMDMs) differentiation to obtain bulk quantities of highly homogenous cells for downstream analyses. PMs, used as a positive control for macrophage cell population, were recovered from the animals' peritoneal cavities while BMDMs were generated from precursor cells. Hamster bone marrow precursor cells were cultured during 6 days in presence of different sources of CSF1 (macrophage colony-stimulating factor 1) to determine the best condition to generate fully differentiated macrophages. The phenotype of hamBMDMs was compared to hamPMs or mBMDMs using (i) microscopic analysis of morphology, (ii) real time quantitative PCR (qRT-PCR) to monitor the expression of macrophage markers, and (iii) capacity to phagocytose zymosan particles. Hamster progenitor cells cultured in presence of human recombinant CSF1 differentiated into cells that were morphologically equivalent to hamPMs and that showed the same increased expression of macrophage markers (CD68, CD14, CSF1r) as mBMDMs when compared to their respective progenitor cells. The phagocytic capacity of hamBMDMs and mBMDMs were similar as judged by the uptake of fluorescent zymosan particles into lysotracker-positive vacuoles by 92% of the cells in both systems. Altogether, these results confirm the robustness of this protocol allowing the production of hamBMDMs. The susceptibility of mouse and hamster BMDMs to *L. donovani* infection was then tested using both hamster-isolated splenic amastigotes and their subsequently derived promastigotes parasites. We demonstrated by *in vitro* infection that only hamster but not mouse BMDMs allow robust proliferation of *L. donovani* parasites, supporting our initial hypothesis that the different disease outcome observed in the corresponding animal models may be determined by the initial *Leishmania*/macrophage interaction. To further characterize hamster and mouse BMDMs and potentially link the differences in susceptibility to *L. donovani* parasites with inherent features of these two macrophage populations, we compared by transcriptomic analysis the changes in gene expression profiles of uninfected mBMDMs and hamBMDMs between 24 hours and 3 days. We found 1198 and 1607 transcripts specifically

*Speaker

regulated only in mBMDMs or in hamBMDMs and 2268 modulated in both mouse and hamster BMDMs (FC> 1.5, padj< 0.05). Interestingly, among those, 73% displayed an inversed regulation profile associated with gene ontology enrichments of key biological processes known to be relevant for *Leishmania* establishment such as angiogenesis or production of pro-inflammatory molecules. We selected 4 transcripts (Nexn, Bap1, Itgal, Lyve1) based on their species-specific regulation profiles and observed, using qRT-PCR, the same expression trends in hamPMs and mPMs, indicating the drift as being an overall macrophage specific feature. In conclusion, we established a solid protocol for hamBMDMs obtention and revealed differencies in the transcriptional signatures of mouse and hamster macrophages that may participate in their resistance or susceptibility to *L. donovani* proliferation. Further analyses are required to validate the implication of these macrophage drifts in parasites establishment. Finally, using hamster and mouse BMDMs as host cells for *L. donovani* parasites, we will gain insight on how the parasite gain leverage of his host.

Keywords: LEISHMANIA DONOVANI, ANIMAL MODELS, BONE MARROW DERIVED MACROPHAGES, HOST, PATHOGEN INTERACTION

Synovial landscape unravels immune-stromal networks linking inflammation to fibrosis in osteoarthritis

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Synovial macrophages are the most abundant immune cell type residing in healthy synovial tissue. Osteoarthritis (OA) is the most common form of degenerative joint disease characterized by progressive cartilage degradation, bone remodeling, as well as synovial inflammation and fibrosis leading to joint dysfunction and pain. This persistent synovitis is usually associated with macrophage accumulation within the synovial tissue, but the characterization, the origin, and the contribution of each macrophage subset in OA remained understudied. Here, we investigated the macrophage diversity in mice with OA, the respective contribution of macrophages according to their origin, as well as their putative interplay with other components of the synovial niche. Single-cell RNA sequencing was performed on knee synovial cells isolated from mice with collagenase-induced osteoarthritis (CiOA) versus PBS-treated animals (CTL). Eleven distinct clusters of cells were identified, including three defined as macrophages. The first macrophage cluster highly expressed genes associated with residency (*Lyve1*, *Timd4*, and *Folr2*), the second cluster lacked these genes but expressed high levels of MHC-II-related genes (*H2-Ab1*, *H2-Eb1* and *Cd74*). The third macrophage cluster was enriched for genes associated to inflammation and fibrosis (*Il1β*, *Spp1*, *Lgals3*). Additionally, a single-cell RNA sequencing was conducted on macrophages isolated from healthy and OA human synovial tissue, and a strong similarity in the gene signatures of macrophage clusters was observed in both species. In OA, flow cytometry and immunofluorescence staining showed a significant recruitment of Tim4- monocyte-derived macrophages in the sublining layer, concomitant to a massive rearrangement of synovial cells implying activated FAP+ fibroblasts. Cell-cell communication inference analyses have identified a privileged interaction between macrophages and stromal cells, uncovering key mediators that likely contribute to chronic synovitis.

Keywords: osteoarthritis, inflammation, fibrosis, synovitis, macrophages

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Neuro-immune interaction of pancreatic islets in T2D.

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Tight regulation of hormone secretion by pancreatic islets is a foremost condition for proper management of glycemia. In the context of T2D, insulin and glucagon secretion are dysregulated and contribute to the development of chronic hyperglycemia. Pancreatic islets are innervated by different subsets of neurons necessary for proper regulation of endocrine hormone release through the secretion of neurotransmitters. Interestingly, innervation is increased in obese T2D mice models, suggesting that dysregulated nerve function in islets could contribute to T2D physiopathology. However, the role of Schwann cells (SC), the glia cells that are essential in maintaining nerve integrity and function, remains entirely unexplored. SC are known to communicate with macrophages in multiple ways. Yet, whether islet-resident macrophages (IRM) and SC interact and contribute to islet function has never been investigated. My central hypothesis is that the interplay between IRM and islet SC regulate endocrine hormone release and islet homeostasis through maintenance of neuronal integrity in physiology. Interference and dysregulation of this interplay may promote islet dysfunction and T2D. In this project I propose to harness new mouse models and the lastest imaging and transcriptomic technologies to characterize the crosstalk between IRM and SC network. Moreover, I aim to determine whether IRM and SC regulate islet neural network integrity and islet homeostasis at the molecular level at steady state and in context of insulin resistance. My work will shed light in the role of these rare cell populations in islet function and in T2D physiopathology.

Keywords: Macrophages, Schwann cells, pancreatic islets, type 2 diabetes

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Histone Lysine Demethysale 6B (KMD6B) maintains adipose tissue integrity and function by reprogramming perivascular adipose tissue macrophages upon energy surplus.

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Background: Obesity is associated with adipose tissue dysfunction leading to the onset and development of several pathologies including type 2 diabetes (T2D). The mechanisms underlying the development of insulin resistance and T2D include adipose tissue (AT) remodelling and inflammation together with altered secretion of adipokines and impaired vascularization. Epigenetic modifications in AT have been proposed to influence the susceptibility to T2D by interfering with the expression of genes involved in AT remodelling, including inflammatory and angiogenic genes, however, the underlying mechanisms are not fully understood. The epigenomic modifier KDM6B (Histone Lysine Demethylase 6B) has been recently identified by our team as a strong candidate and we hypothesize that KDM6B plays a key role in adipose tissue macrophage (ATM) plasticity and AT integrity upon energy surplus.
Methods: We used a genetically modified mouse model of Kdm6b deficiency restricted to myeloid cells (Kdm6b MKO). Mice were subjected to high-fat diet for 4 and 12 weeks to induce obesity, AT remodelling and insulin resistance. We analyzed metabolic parameters in combination with genome-wide molecular (scRNA-seq and RNA-seq) and epigenomic (scATAC-seq) analysis of ATM.

Results: Our data indicated that Kdm6b is differentially expressed in ATM subsets with a higher expression in perivascular macrophages (PVATM). Kdm6b expression in PVATM was reduced with obesity. Kdm6b deficiency in myeloid cells induced transcriptomic changes and cellular reprogramming of ATMs, associated with AT inflammation and impaired AT vascularization as well as systemic glucose intolerance and insulin resistance.
Conclusion: Our findings establish a protective role for KDM6B-mediated epigenetic modifications in PVATM upon energy surplus by maintaining AT vasculature and limiting inflammation. They highlight KDM6B modulation as a potential therapeutic strategy for immunometabolic impairment and disease.

Keywords: Obesity – T2D – Immunometabolism – Epigenetic modifier – Adipose tissue – Macrophages

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In situ identification of both IL-4 and IL-10 Cytokine-Receptor Interactions during tissue regeneration

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Abstract: Cytokines secreted by individual immune cells regulate tissue regeneration and allow communication between various cell types. Cytokines bind to cognate receptors and trigger the healing process. Determining the orchestration of cytokine interactions with their receptors on their cellular targets is essential to fully understanding the process of inflammation and tissue regeneration. To this end, we have investigated the interactions of Interleukin-4 cytokine (IL-4)/Interleukin-4 cytokine receptor (IL-4R) and Interleukin-10 cytokine (IL-10)/Interleukin-10 cytokine receptor (IL-10R) using in situ Proximity Ligation Assays in a regenerative model of skin, muscle and lung tissues in the mini-pig. The pattern of protein-protein interactions was distinct for the two cytokines. IL-4 bound predominantly to receptors on macrophages and endothelial cells around the blood vessels while the target cells of IL-10 were mainly receptors on muscle cells. Our results show that in situ studies of cytokine-receptor interactions can unravel the fine details of the mechanism of action of cytokines.

Keywords: IL, 4, IL, 10, receptor, interaction, regeneration, Proximity ligation assay

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Leishmania: A macrophage signalling malware

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Leishmania, the causative agent of Leishmaniasis exhibits at least two distinct developmental stages, the extracellular form in the insect vector and the intracellular form that differentiates and proliferates within the macrophage phagolysosomes of the mammalian macrophages. *Leishmania* releases parasitic factors, such as kinases, to subvert the macrophage functions. Host manipulation by *Leishmania* is a well-studied concept at the transcriptional and proteomic level but unexplored at the phosphoproteomic level. Yet, modulation of the host phosphorylation status provides for the parasite a way to induce rapid and efficient hacking of host signalling. Considering that phosphorylation might be functionally most relevant during infection events, for the first time, we investigated the effect of *Leishmania donovani* infection on the status of macrophage phosphorylation at 4, 24 and 48hrs post infection. Early analysis indicates an overall dephosphorylation of host proteins as infection progresses. Looking more specifically, we showed that pathways are modulated following a certain temporal pattern, moving from actin remodelling and RNA processing to nutrient processes followed by metabolism and immune pathways. These sequential changes may relate to the specific requirements of the parasite at different points of infection. Using the temporal dimension, we are actively working to uncover specific signatures of *Leishmania* infection in terms of "phospho-marks" on host proteins. Interestingly, during *Leishmania donovani* infection, we only identified few changes in the host proteome suggesting that *Leishmania*-induced host modulation occurs mostly at the PTM level rather than at the protein level in the first 48h of infection. Consequently, the seen protein level changes might hold immense importance for infection. In this notable list of proteins, ferritin light chain 1 (Ftl1) was found to be massively upregulated as infection progressed. Ferritin is a protein complex composed of light (Ftl1) and heavy (Fth1) chains and is one of the primary sites for iron storage in cells. In previous work, Ftl1 has been shown to interact with a secreted parasitic kinase making it a good candidate to explore. Moreover, it localises around the parasite or the parasitophorous vacuole during infection, a phenotype that is species specific. Since Ftl1 plays a role in iron storage, we hypothesize this localisation might be important for the parasite to obtain iron from the host and thus may have a role in *Leishmania* infection. Indeed, increasing iron concentration, significantly improves proliferation of *Leishmania* in macrophages. Overall, modulation of host signalling is a key process by which *Leishmania* prepares the host cell for its intracellular life.

Keywords: Host modulation, Omics, Iron homeostasis, Leishmaniasis

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Nuclear envelope disruption triggers hallmarks of aging in lung alveolar macrophages

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Chronological aging is characterized by gradual immune dysfunction, degrading protective immune responses, thus enhancing susceptibility to many diseases. Immune aging is characterized by chronic inflammation which itself contributes to immune dysfunction and organismal aging. Genomic instability has been proposed to play a central role in driving the aging process, however, the intracellular mechanisms that compromise genome stability during aging in immune cells are ill-defined. Immune cells are highly migratory and constantly go through narrow spaces that lead to cellular, and nuclear, deformation. Hence, mechanical forces induce transient nuclear envelope (NE) rupture and DNA damage, with largely unknown consequences. Lamin A/C, an essential component of the Lamin meshwork, provides essential mechanical protection to the nucleus. Mutations in the Lamin A/C gene have been associated with decreased genome stability, increased NE rupture, and accelerated aging phenotypes. Whether Lamin A/C provides protection against aging in the immune system is not known. Here, we aimed to probe for the physiological consequences of NE rupture in the immune system. AM migrate within constricted spaces *in vivo*, in dimensions that induce NE rupture and DNA damage. Using *in vivo* models, we show that Lamin A/C ablation in immune cells results in selective depletion of lung alveolar macrophages (AM) and a heightened susceptibility to lung pathologies. Lamin A/C-deficiency

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in AMs induces constitutive NE rupture, DNA damage and p53-dependent senescence. Lamin A/C-deficient AM display an upregulated lysosomal signature with CD63 expression, and we find that CD63 is required to clear damaged DNA in macrophages and may constitute a new essential anti-inflammatory pathway in aging. Strikingly, Lamin A/C-deficient AM recapitulate features of aging. We propose that induction of genomic instability by NE disruption represents a mechanism of aging in AM.

Keywords: aging, alveolar macrophages, nuclear envelope rupture, DNA damage

New endotypes of type 2 diabetes based on immune-inflammatory profiles

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Type 2 diabetes (T2D), which accounting for 90% diabetic patients, is a heterogeneous set of diseases with an extensive clinical diversity. Additionally, inflammation has been recognized for its key role in the development of T2D and its vascular complications. Circulating monocyte activation contributes to tissue alterations in T2D. In this context, Ahlqvist's team classified diabetes into 5 subgroups based on GAD antibodies, age, BMI, HbA1c and HOMA indexes with distinct clinical and metabolic phenotypes. Futhermore, a German team found that the numbers of circulating immune cells populations are different between the Ahqvist's subgroups, suggesting different blood immune cells may be associated with different T2D endotypes. For that reason, we perform our study to cluster the newly-diagnosed T2D patients based on their clinical features and immune-inflammatory circulating cell profiles.
We recruited 410 newly-diagnosed T2D patients in Lariboisière and Bichat hospital from 2016 to July in 2023. We used k-mean method based on age, BMI, HbA1c and immune circulating cell counts (leucocytes, neutrophils, monocytes and lymphocytes) to cluster these patients into 4 endotypes that each of them has specific characteristics. Cluster 2 has the highest number of blood immune cells and the highest age. Meanwhile, cluster 4 has the lowest number of blood immune cells and the lowest insulin resistance. Cluster 1 has the relatively high immune cell counts, the lowest age and high BMI. And cluster 3 has low immune cell counts, highest HbA1c and lowest insulin secretion.
Our result highlight the circulating immune cell counts might be used to define new T2D endotyes. For the future plan, we utilize multiomic approaches to analyze the inflammatory pathway in circulating immune cell subpopulations in endotypes.

Keywords: type 2 diabetes, cluster, inflammation

*Speaker

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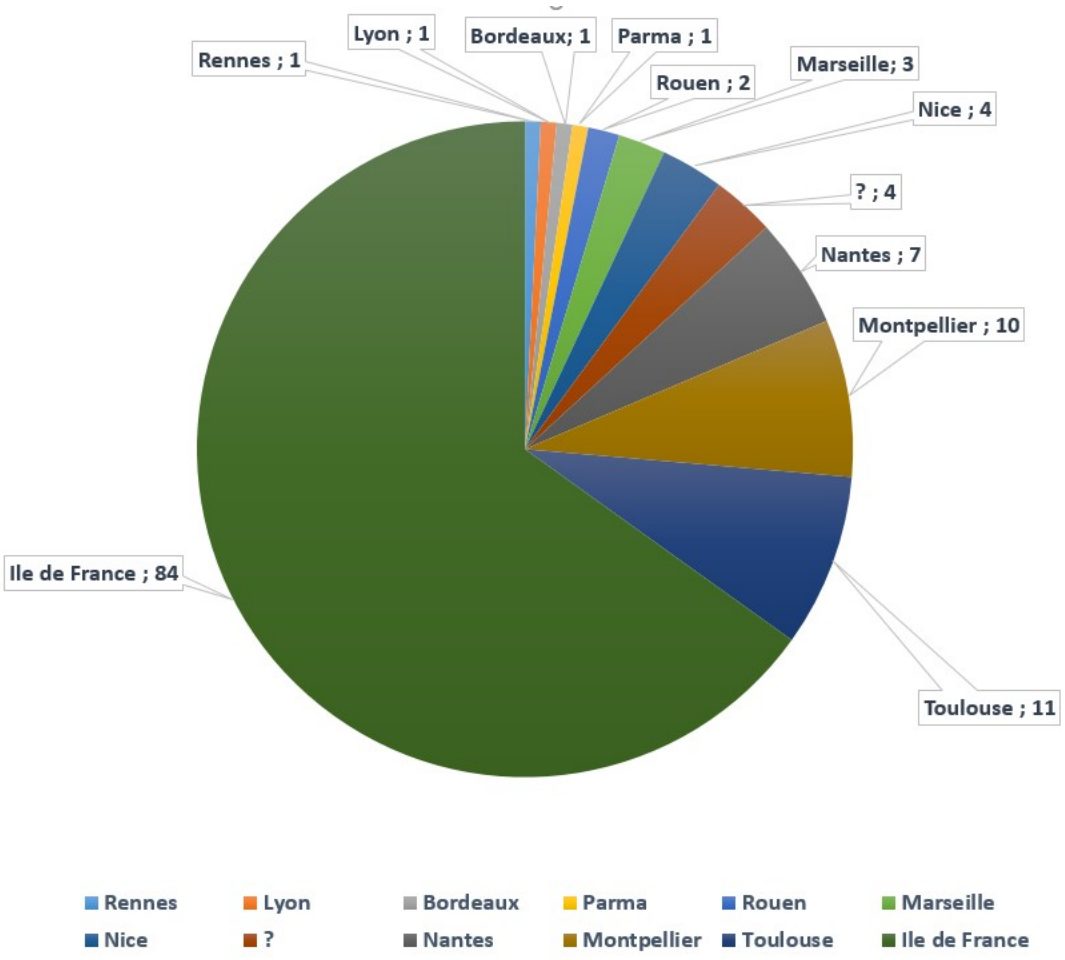

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