



SoMM  
2018

2<sup>ND</sup>

SYMPOSIUM  
OF THE OCCITANIE NETWORK  
ON MONOCYTES-MACROPHAGES

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*INVITED SPEAKERS*

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FRIDAY, 30 NOVEMBER 2018 **TOULOUSE**

Auditorium Institut Universitaire du Cancer Toulouse (IUCT) Oncopole





# Charlotte Scott

(Martin Williams- ONSET group)

Department of Biomedical molecular biology

University of Gent, Gent, Belgium

Postdoctoral Researcher

**Link:** [ResearchGate](#)

## Research description:

ONTOGENY AND FUNCTIONAL SPECIALIZATION OF MYELOID CELL SUBSETS (ONSET)

Our research focuses on the development and functional specialization of macrophages (MΦs) and dendritic cells (DCs). To unravel the role of MΦs and DCs in the regulation of immune responses in vivo we have constructed novel DTR- or CRE- expressing knock-in mice that allow to deplete a particular MΦ or DC subset specifically in vivo or to knock-down genes of interests specifically within these cells. These novel knock-in mouse models include mice specific for liver resident Kupffer Cells and lung-resident Alveolar Macrophages. We are particularly interested in: (i) identifying the transcription factors that drive DC and MΦ development, (ii) unraveling how tissue-resident macrophages participate to the maintenance of tissue homeostasis and (iii) understanding how inflammation influences the development and function of DC and MΦ subsets.



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## KEYNOTE LECTURE I:

Zeb2 and maintenance of macrophage tissue-specific identities



# Arnaud Jacquiel

U1065, C3M, Nice, France

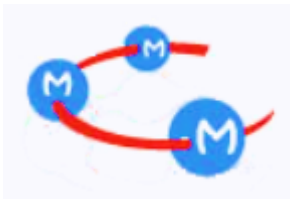
## Researcher CRCN INSERM

Links : [https://www.researchgate.net/profile/Arnaud\\_Jacquel](https://www.researchgate.net/profile/Arnaud_Jacquel)

<http://www.unice.fr/c3m/index.php/research-teams/patrick-auberger/>

### Research description:

DISSECTING THE MECHANISMS INVOLVED IN MACROPHAGE DIFFERENTIATION OF MONOCYTES AND THEIR ALTERATIONS IN CMML.



The differentiation of peripheral blood monocytes into resident tissue macrophages can be recapitulated ex vivo by CSF-1. We recently established that proper macrophagic differentiation of monocytes required both caspase and autophagy activation and pinpointed a novel and highly specific mechanism of caspase activation during this process. This may explain the limited number of cleaved substrates at sites different from those cleaved during apoptosis observed during monocyte differentiation. In this context, the main objectives of the project are to decipher this original mechanism of caspase activation, understand the specificity of cleavage and the role of the cleaved fragments and define the interplay between caspases and autophagy during CSF-1 mediated monocyte differentiation. We expect to highlight a new mode of caspase activation in a non-apoptotic context, i.e. the physiological differentiation of monocytes and more widely during hematopoietic cell differentiation. Finally, understanding these mechanisms of differentiation will allow us to better understand the pathophysiology of the chronic myelomonocytic leukemia (CMML) characterized by defect in monocyte differentiation.

## LECTURE I:

Molecular characterization of the mechanisms involved in macrophage differentiation of monocytes



# Dominique Baeten

Amsterdam, Netherland, UCB (originally Union Chimique Belge)

MD PhD, Professor.

Link : <https://www.ucb.com/>

## Research description :

Keeping a part-time position as Professor of Rheumatology at the University of Amsterdam, Dominique Baeten joined UCB in August 2016 where he first lead the New Patient Value Mission in the Immunology Patient Value Unit and is currently Vice-President and Head of the Immuno-Bone Therapeutic Area. His major interest is in early clinical development (bringing solutions to the right patient population) and translational medicine (linking unmet needs back to the cellular and molecular pathways).

His translational research focuses on the immunopathology of chronic inflammatory arthritis, in particular rheumatoid arthritis and arthritis). He aim to identify the immune alterations triggering, driving and perpetuating these diseases in order to a) understand the pathophysiology, b) develop biomarkers for prediction, diagnosis and prognosis (including treatment response), and c) develop and validate innovative targeted therapies.



## KEYNOTE LECTURE II

Macrophage-derived pro-inflammatory cytokines in chronic arthritis: how one single mediator can lead to completely different phenotypes



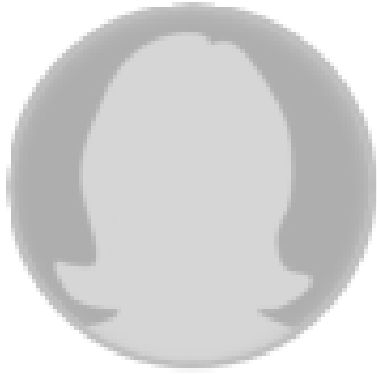
Inspired by **patients**.  
Driven by **science**.

*Inspirés par les **patients**. Guidés par la **science**.*

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# Audrey Varin

Stromalab, Toulouse, France

## Researcher

**Link:** <https://www.stromalab.fr/equipe2/interactions-csmimmunité/>

### Research Description :

Audrey Varin did her thesis in the laboratory of Prof. Georges Herbein at the University of Franche-Comté where she worked on the control of the replication of HIV in macrophages by the accessory proteins of HIV. Then, she left 2 years post-doc at Oxford University in the team of **Pr Siamon Gordon** where she worked on alternatively activated macrophages and their responses to different pathogens such as bacteria *Neisseria meningitidis* or H5N1 influenza virus, part of the work done at the University of Hong Kong. This work was continued for 1 year in the laboratory of Prof. Giorgi Trinchieri at the National Cancer Institute in Frederick, Maryland, USA. Since 2011, she is a researcher in the STROMALab unit in Toulouse where she work on the interaction between the innate immune system and mesenchymal stromal cells and its role in tissue regeneration.



## LECTURE II

Interaction with pro-inflammatory macrophages modulated immunosuppressive function of human mesenchymal stromal cells



# Elisa Gomez Perdiguero

Institut Pasteur, Paris, France

## Laboratory Director

**Link:** <https://research.pasteur.fr/fr/team/macrophages-and-endothelial-cells/>

### Research Description :

#### MACROPHAGES AND ENDOTHELIAL CELLS

Our team is interested in the role of 'resident' macrophages during development, homeostasis and tissue repair. Macrophages are professional phagocytic cells that scavenge dead/apoptotic cells, debris, macromolecules and pathogens, and also produce cytokines, both concurring to tissue homeostasis and repair. They belong to the innate immunity arm of the hematopoietic system.

Within the hematopoietic system that produces all blood cells, tissue 'resident' macrophages are a lineage of myeloid cells that arise from yolk sac-derived progenitors and that self-maintain in their tissue of residency, independently of adult hematopoietic stem cells (HSC).

Resident macrophages from the same lineage, such as liver Kupffer cells, brain microglia, epidermal Langerhans cells, lung alveolar macrophages..., display tissue-specific phenotypes, perform tissue-specific functions and have distinct gene expression profiles. Thus, resident macrophages are a unique system where the respective contributions of ontogeny and environment can be investigated. We will combine methods from the fields of immunology, developmental biology and angiogenesis to understand in vivo the development and lineage-specific function(s) of resident macrophages, thereby opening new venues of research into the interaction between macrophages and endothelial cells during development and in response to tissue damage.



## KEYNOTE LECTURE III

TBA (To be announced)



# Florence Perrin

INSERM U1198 - Université de Montpellier, France

Laboratory Director. Professor

Links : <http://www.ibn-lab.com/fr/equipe/1-florence-perrin>

## Research Description :

We investigate molecular mechanisms that underlie neurodegeneration processes in the context of spinal cord pathologies. We are particularly interested in spinal cord injury (SCI), a pathology with no current therapy.

One means of analyzing the molecular substrate of degeneration is to identify in each cell type of the spinal cord, changes induced in gene expression levels. In this line, we are developing a genomic integrative analysis to decipher the specific involvement of different cell populations as well as cell-cell interactions in mechanisms and pathogenesis of SCI and other spinal cord pathologies. Using this approach will allow the identification of cell specific candidate genes that may be involved in the mechanisms and pathogenesis of spinal cord diseases.

We have a specific interest in glial cells (such as astrocytes and microglia), the most abundant cells in the mammalian central nervous system. Yet our knowledge about their function in health and disease has been limited but during the last years, more and more evidence show that they are playing a crucial role in several spinal cord and brain pathologies.

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## LECTURE III

RNA-Seq analysis of microglia reveals time-dependent activation of specific genetic programs following spinal cord injury.





# Hal Drakesmith

University of Oxford, Oxford, UK

Associate professor of immunology

**Links**

<https://www.medsci.ox.ac.uk/study/graduateschool/supervisors/alexander-drakesmith>

[https://www.researchgate.net/profile/Hal\\_Drakesmith](https://www.researchgate.net/profile/Hal_Drakesmith)

**Research description:**

**IRON AND INFECTION**

My lab at the Weatherall Institute of Molecular of Medicine studies the role of iron in infectious diseases and the immune response. Iron is critical for the biochemistry of cells, and is needed equally by host and pathogen; indeed the 'battle for iron' is a key determinant of the outcome of infection. We study the molecular basis of this battle, focusing on hepcidin, the iron regulatory hormone (1, 2). Hepcidin controls iron homeostasis analogously to how insulin controls glucose, but unlike insulin, hepcidin is also an acute phase response gene and is upregulated by inflammation. This innate immune activity of hepcidin reflects the importance of iron regulation for host-pathogen interactions.



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## KEYNOTE LECTURE IV

Iron, hepcidin and macrophages: implications for innate and adaptive immunity





# H el ene Authier

IRD. Pharma-Dev, Toulouse, France

Associate Professor

*Links*

<http://www.pharmadev.ird.fr/annuaire/helene-authier2>

<https://clubmacrophage31.weebly.com/heacutelegravene-authier.html>

**Research description:**

INFLAMMATORY BOWEL DISEASES AND MACROPHAGES  
POLARIZATION

We are studying the role of macrophages in inflammatory bowel diseases and particularly their polarization and implication of C-type lectin receptors in the course of intestinal chronic inflammation. We identify signaling pathways and molecular mechanisms that control the M1/M2 polarization of macrophages.



## LECTURE IV

Dectin-1 on macrophages promotes inflammatory responses contributing to the severity of inflammatory bowel diseases



# Sandrine Henri

(Bernard Mallissen- group)

CIML, Marseille, France

Researcher CRCN INSERM

**Link :** <http://www.ciml.univ-mrs.fr/science/lab-bernard-marie-malissen/genetic-dissection-function-t-cells-and-dendritic-cells>



Sandrine HENRI is a resident scientist who joined the CIML Institute in 2002 after a post-doctoral training in Ken Shortman's laboratory (WEHI, Melbourne, Australia). As a project leader, she supervised postdoctoral fellow, PhD student and technical assistant and made major contributions to decipher dendritic cell and macrophage subsets within peripheral tissues (ie skin and gut) and to the understanding of their origins and functions. With the use of innovative knock-in mouse models, she is interested to further understand the role of each specific DC and macrophage subsets in the control of adaptive T cell responses and to apply this knowledge to improve vaccination and desensitization.

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## (KEYNOTE) LECTURE V

### Dermal Macrophages: Failure of the phagocytosis process