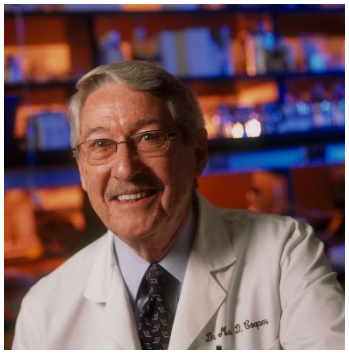
2015 LOUIS-JEANTET SYMPOSIUM

October 14 2015
Centre Medical Universitaire (CMU), Geneva
Auditorium 250



Max D. Cooper

Georgia Research Alliance Eminent Scholar; Professor, Department of Pathology and Laboratory Medicine, Emory University School of Medicine, Atlanta

Wednesday, October 14 2015, 09:00 – 09:30

The evolution of adaptive immunity in vertebrates

All living organisms have innate immune systems which they use for self-defense, but only vertebrates have a lymphocyte-based adaptive immune system that allows recognition of specific pathogens and protective memory against a second encounter. Alternative types of receptors for antigens have been defined for jawed and jawless vertebrates. The lymphocytes in jawless vertebrates (lampreys and hagfish) use leucine-rich-repeat based variable lymphocyte receptors (VLR) for antigen recognition, whereas T and B lymphocytes in all the jawed vertebrates use immunoglobulin-based receptors for the same purpose. Three lamprey VLR loci, VLRA, VLRB and VLRC, undergo assembly and expression in a clonally diverse fashion by separate populations of lymphocytes that resemble thymus-derived γδ and αβ T lymphocytes and bone marrow-derived B lymphocytes. This phylogenetic analysis suggests that prototypic T and B cell lineages evolved in a common vertebrate ancestor ~500 million years ago, prior to the convergent evolution of VLR and BCR/TCR types of antigen receptors. The question of when innate lymphoid cells evolved will also be addressed in this presentation.

Biography

Max Cooper, Professor of Pathology and Laboratory Medicine at Emory University, conducts comparative studies of lymphocyte development in order to better understand the differentiation defects in immunodeficiency diseases, lymphoid malignancies and autoimmune diseases. Research highlights include the definition of T and B cell lineages, identification of the hematopoietic tissue origin of B lineage cells, demonstration of isotype switching by IgM^+ lymphocytes, description of intestinal lymphoid follicle-associated epithelial "M" cells and their transcytotic function, demonstration that *TCR* excision circles in peripheral T cells can be used to monitor thymic function, and discovery of an alternative adaptive immune system in jawless vertebrates.

http://vaccines.emory.edu/faculty/cooper_max.html
http://pathology.emory.edu/CooperLab/

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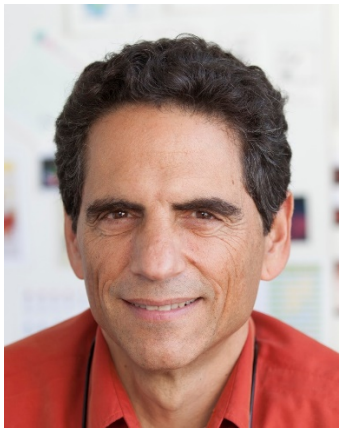

Ed Palmer

Prof. Experimental Transplantation Immunology, Departments of Biomedicine and Nephrology, University Hospital-Basel, University of Basel

Wednesday, October 14 2015, 09:30 – 10:00

T cell tolerance is built on stochastic events: the numbers favor tolerance, just not all the time

To generate a state of T cell tolerance, a developing thymocyte has to “read” the affinity of its T cell receptor (TCR) for a self-antigen. How the TCR measures antigen affinity and initiates negative selection has has been an open question for some time. The principle of affinity as well as the stochastic mechanism used by the TCR to establish an affinity threshold for central tolerance will be presented. In addition, how imperfections in negative selection lead to the escape of autoimmune T cells and the generation of autoimmunity will also be covered. Finally, the implications of stochastic processes in biology and medicine will be briefly discussed.

Biography

Ed Palmer received his MD and PhD degrees from the University of Rochester, NY, USA in 1980. After a post-doctoral fellowship at the Salk Institute, he started his own laboratory at the University of Colorado School of Medicine in 1984. In 1993, he joined the Basel Institute for Immunology as a permanent member, where he worked until the Institute was closed in 2001. He subsequently joined the Medical Faculty of the University of Basel as Professor of Experimental Transplantation Immunology. Ed Palmer has spent his scientific career working on TCR signaling, T cell tolerance and autoimmunity.

https://biomedizin.unibas.ch/nc/research/research-group-details/home/researchgroup/transplantation-immunology-and-nephrology/

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Michael L. Dustin

Kennedy Institute, NDORMS, University of Oxford, Oxford, UK; Skirball Institute, New York University School of Medicine, New York

Wednesday, October 14 2015, 10:00 – 10:30

New roles for T cell receptor through extracellular vesicles

The immunological synapse is a specialized cell-cell junction characterized by central accumulation of antigen receptors surrounded by adhesion molecules. We performed correlated light and electron tomography studies on the immunological synapse formed by T cells with supported lipid bilayers (SLB). Surprisingly, the compartment enriched the T cell antigen receptor (TCR) and major histocompatibility complex (MHC), was not a simple close contact region, but was instead an extracellular compartment packed with TCR enriched extracellular vesicles derived from the plasma membrane, also referred to as ectosomes. We have subsequently found that these synaptic ectosomes are produced in an ESCRT dependent manner and bud from the plasma membrane to fill this central extracellular compartment in proportion of the MHC-peptide complexes. Microvesicle formation contributes to TCR down-regulation and can participate in activation of the antigen-presenting cell. The role of ESRCT was further verified by demonstrating the TSG101 interacting protein HIV Gag can disrupt incorporation of TCR into central microvesicles in the immunological synapse. We further have found that the enveloped virus restriction factor tetherin is highly concentrated in and near the synaptic ectosomes and may account for apparent tethering of the vesicles to the T cell in immunological synapses formed by human T cells. Our results suggest that tetherin also restricts the release of synaptic ectosomes and may generate unique TCR specific signals with co-stimulatory character. This would represent a costimulatory signal that would be linearly related to MHC-peptide complexes, which is critical information in T cell help for B cells in antibody production.

Biography

Dr. Dustin has a B.A. from Boston University (1984) and a Ph.D. from Harvard University (1990). He trained further at Washington University School of Medicine (1990-93) and then joined the faculty (1993-2000). His work there led to discoveries related to the T cell immunological synapse. He moved to the Skirball Institute at New York University School of Medicine in 2001 where he focused on in vivo testing of the immunological synapse hypothesis. He moved to the Kennedy Institute of Rheumatology at the University of Oxford in 2013 to focus on translation of the immunological synapse.

http://www.kennedy.ox.ac.uk/research/group/mdustin

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Andreas Diefenbach

Research Centre of Immunology, Johannes Gutenberg University Mainz, Germany

Wednesday, October 14 2015, 11:00 – 11:30

Brave new world of innate lymphoid cells

Innate lymphoid cells (ILCs) are a recently discovered family of innate lymphocytes that are substantially represented at mucosal surfaces and have been implicated in the protection of epithelial barriers. Three groups of ILCs can be discriminated based on the expression of distinct transcription factors controlling the expression of a distinct set of cytokine genes endowing the various ILC subsets with a specific range of effector functions. Group 1 ILCs (ILC1s) are a diverse group of ILCs comprised of natural killer (NK) cells and other, poorly defined subsets of ILCs. It is believed that the ILC1 fate decision is controlled by the T-box transcription factor T-bet. ILC2s express high levels of GATA-3, produce IL-5 and IL-13 and have been involved in immunity to helminth infections and in the pathogenesis of allergic diseases. Group 3 ILCs developmentally depend on the transcription factor RORgt and produce the cytokines IL-22, IL-17A and IL-17F. ILC3s are believed to be involved in the protection against intestinal bacterial infections and, if inappropriately stimulated, can be important drivers of inflammatory disorders. The transcriptional programs and effector cytokines of the various ILC subsets strikingly resemble those of the various T helper cell effector fates suggesting that such transcriptional circuitry already formed in the evolutionary older innate immune system. I will summarize new data how tissue-resident ILC3s regulate organ function and control epithelial integrity.

Research in my lab is supported by grants from the *European Research Council* (ERC) and *Deutsche Forschungsgemeinschaft*

Biography

Andreas Diefenbach is Professor and Chair of Medical Microbiology and the coordinator of the Research Centre of Immunology at the University of Mainz, Germany. His lab studies transcriptional and epigenetic control of cell fate decisions in the innate immune system. Current research interests include how the immune system coordinates adaptation of multicellular organisms to constantly changing environments (microbiota, nutrients).
As a Physician Scientist, Andreas won grant support by the *Howard Hughes Medical Institute*, by the *European Research Council,* and by the *Deutsche Forschungsgemeinschaft* (DFG). He is the coordinator of the DFG Priority Program ("Innate Lymphoid Cells"). He was awarded with the Main Scientific Prize of the *German Society of Hygiene and Microbiology*.

www.imb-mainz.de/students-postdocs/international-phd-programme/ipp-groups/andreas-diefenbach/

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Nadine Cerf-Bensussan

Laboratory of Intestinal Immunity. Institut Imagine and Université Paris-Descartes-Sorbonne Paris Cité

Wednesday, October 14 2015, 11:30 – 12:00

Host-microbiota interactions across the immune system: the role of segmented filamentous bacterium

To cope with the complex microbial community that settles in the distal part of our intestine after birth, hosts have evolved a spectrum of complementary innate and adaptive immune mechanisms that are programmed ante-natally but fully develop only after birth in response to signals from the microbiota. Studies in mice unexpectedly revealed that a restricted number of host-specific bacterial species, the prototype of which is Segmented Filamentous bacterium, are necessary for to stimulate the development of gut lymphoid tissues and the complete spectrum of homeostatic innate and T cell responses induced by a complex microbiota. Based on the observations that SFB has a reduced genome and a very unusual life cycle initiated by its attachment to the ileal mucosa, we have hypothesized that 1- SFB growth depends on nutrients derived from epithelial cells; 2- In turn, attachment of SFB to epithelial cells initiate innate signal(s) indispensable to instruct gut immune responses. Using I*n vitro* culture conditions that recapitulate the SFB niche in the intestine, we have succeeded in obtaining SFB growth in contact with epithelial cells. Studies in progress aim at dissecting the signals induced *in vitro* by SFB in epithelial cells and at testing their contribution *in vivo* to SFB-induced maturation of the gut immune system in gnotobiotic mice. Parallel studies in humans are in progress to interrogate the presence and distribution of a putative ”human specific” SFB that may be necessary to drive post-natal maturation of gut immune responses.

Biography

Nadine Cerf-Bensussan, MD PhD, is a Research Director at the French National Institute of Health and Medical Research (INSERM), and head of the Laboratory of Intestinal Immunity at Imagine Institute in Paris. Her laboratory studies the mechanisms underlying the interactions between the host immune system, the intestinal epithelium and intraluminal antigens in health and in disease. She was awarded an ERC grant in 2013 to support her work on host-microbiota interactions and the Research Prize of INSERM in 2014 for her work on celiac disease.

http://www.institutimagine.org/en/research/23-research-labs/119-laboratory-of-intestinal-immunity.html

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Fiona Powrie
2012 Louis-Jeantet Prize winner

Fiona Powrie FRS, Director, Kennedy Institute of Rheumatology, University of Oxford and Translational Gastroenterology Unit, Experimental Medicine Division

Wednesday, October 14 2015, 12:00 – 12:30

Keynote lecture: **Gut reactions: Immune pathways in the intestine in health and disease**

The gastrointestinal (GI) tract is home to a large number and vast array of bacteria that play an important role in nutrition, immune system development and host defense. In inflammatory bowel disease (IBD) there is a breakdown in this mutualistic relationship resulting in aberrant inflammatory responses to intestinal bacteria. Studies in model systems indicate that intestinal homeostasis is an active process involving a delicate balance between effector and immune suppressive pathways. The cytokine IL-23 plays a pivotal role in orchestrating intestinal inflammation and several genes in the IL-23/Th17 pathway confer risk to IBD. We have shown that IL-23 acts directly on T cells to promote pathological Th17 type responses at the expense of immune suppressive regulatory T cells. In this presentation I will discuss our new studies linking regulatory T cell function in the intestine to the tissue damage response through the alarmin IL-33. The results indicate that the balance between IL-23 and IL-33 is an important determinant of the intestinal immune response.

Biography

Fiona Powrie is the Director of the Kennedy Institute of Rheumatology and Principal Investigator in the Translational Gastroenterology Unit, University of Oxford. Her research interests include characterisation of the interaction between the intestinal microbiota and the host immune system and how this mutualistic relationship breaks down in inflammatory bowel disease. Her work has identified the functional role of regulatory T cells in intestinal homeostasis. She has received a number of awards including the Louis Jeantet Prize for Medicine 2012 and was elected a Fellow of the Royal Society in 2011.

http://www.kennedy.ox.ac.uk/research/group/fpowrie

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Facundo Batista

Francis Crick Institute, Cancer Research UK, London

Wednesday, October 14 2015, 13:45 – 14:15

Dynamic imaging of B cell activation – from single molecules to living tissue

B lymphocytes form an integral part of the immune system via the production of specific antibodies and by establishing immunological memory.
In the Lymphocyte Interaction Laboratory, we strive for a comprehensive understanding of the cellular and molecular events leading to B cell activation as well as elucidating how they differentiate into memory or antibody--producing cells. We address this by combining the power of genetics with biochemistry and advanced imaging technology. For instance, by tracking single particles of BCR we have previously shown that BCR diffusion is restricted by an ezrin--defined actin network, and that this restriction regulates receptor signalling. We have since explored this novel concept of signalling regulation in greater detail by implementing super—resolution microscopy methods.
As well as studying lymphocyte interactions at the micro-- and nano--scale, we actively pursue the understanding of how, where and when B cells are activated in vivo. We have recently focused special attention on the importance of lymph node architecture: specifically, investigating B cell activation in a model of double infection. We have seen that the changes that occur in the structure of the inflammatory lymph node have a dramatic effect on the ability of B cells to respond to pathogens and suggest a potential mechanism for increased susceptibility to secondary infections. Up-to-the-minute unpublished data from the group will be presented, adding to a body of work that shows how a clear understanding of lymphocyte interactions and signalling has wide--ranging implications for the study of cancer and infectious disease.

Biography

Facundo D Batista obtained his biology degree at the University of Buenos Aires, Argentina, and his MSc and PhD in Trieste, Italy, before pursuing his work at the University of Cambridge, UK. In 2002, Facundo established his own independent laboratory at the London Research Institute, Cancer Research UK. Throughout Facundo's scientific career, his research has consistently been communicated in high-impact journals. He has been invited both to author reviews in prestigious journals, and to present at key international conferences, such as Keystone, AAI and ICI. Furthermore, Facundo is member of the Editorial Board of four journals including Science. Facundo was elected to the EMBO Young Investigator Programme in 2004 and has since been awarded EMBO membership and the Royal Society Wolfson Research Merit Award and was nominated Fellow of the Academy of Medical Science in the UK.

http://www.crick.ac.uk/research/a-z-researchers/researchers-a-c/facundo-d-batista

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Yasmine Belkaid

Mucosal Immunology section, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda

Wednesday, October 14 2015, 14:15 – 14:45

Long term consequences of acute infections

Our laboratory aims to understand the mechanisms controlling infection at barrier sites such as the skin and the gut. These two sites represent the first portal of pathogen exposure and are major anatomical sites for development of inflammatory disorders. The skin and the gut also represent highly specialized environments with distinct structures, cell types, and innate defense mechanisms tailored to support their individual challenges. These include their exposure to factors from the outside environment, to dietary antigens, and to antigens derived from resident commensals. In particular, all barrier surfaces are covered by a diverse and abundant microbiota that play a dominant role in host physiology and immunity. However, this symbiotic relationship also poses a constant threat to the host, and aberrant reactivity against commensals can lead to life-threatening tissue damage. These conflicting pressures present the host system that defends the skin or the gut with unique challenges: tolerating constant exposure to innocuous antigens while simultaneously maintaining the capacity to rapidly respond to encounters with pathogens.

The Major Areas of Research are: Role of the microbiota in immunity to infection; role of dietary metabolites in promoting immune regulation and immune responses to pathogens; tissue specific regulatory responses to infection.

Biography

Dr. Yasmine Belkaid obtained her Ph.D. in 1996 from the Pasteur Institute in France on innate responses to *Leishmania* infection. Following a postdoctoral fellowship at NIAID on immune regulation during *Leishmania* infection, she joined the Children's Hospital Research Foundation in Cincinnati as an assistant professor in 2002. In 2005, she joined the Laboratory of Parasitic Diseases as a tenure-track investigator. Since 2008, she has worked as an adjunct professor at the University of Pennsylvania.

http://www.niaid.nih.gov/labsandresources/labs/aboutlabs/lpd/mucosalimmunology/Pages/belkaid.aspx

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Bart N. Lambrecht

Department Director IRC, Head Laboratory of immunoregulation and mucosal immunology, Professor of Medicine, Ghent University

Wednesday, October 14 2015, 14:45 – 15:15

Dendritic cell and epithelial cell crosstalk at the heart of the allergy epidemic

Allergic asthma is characterized by accumulation of eosinophils, mast cells, Th2 cells and IL-21 producing effector cells that lead to goblet cell hyperplasia, bronchial hyperreactivity and airway wall remodeling. Dendritic cells are crucial not only in the initiation of T cell responses, but also for their maintenance. Exogenous danger signals like LPS are commonly found in allergens like house dust mite. Airway DCs get activated in response to LPS in HDM, but do so indirectly, via GM-CSF, TSLP, IL-1, IL-25 and IL-33 released by bronchial epithelial cells. Different DC subsets seem to perform different tasks in the process of allergic sensitization. Inflammatory type ($CD64^+$, $MAR\text{-}1^+$) DCs and CD11b+ cDCs present allergens best, whereas $CD103^+$ cDCs, pDCs and non-professional APCs like B cells and macrophages are poor APCs that can even tolerize lung immune responses to allergens. When small amounts of allergens are given, mainly CD11b cDCs perform the task of Th2 priming *in vivo*. The main function of $CD64^+$ inflammatory DCs is presentation of allergens to already primed Th2 cells in the lung, and the production of proinflammatory chemokines.
Sensitization to inhaled allergens can thus be caused by direct or indirect activaton of cDCs. Some environmental pollutants stimulate DC maturation and cause allergic sensitization while others like farm dust protect from allergic sensitization. We have recent evidence that LPS and farm dust dampen the epithelial response to allergens, via induction of TNFAIP3, thus preventing proper DC activation. Polymorphisms in the TNFAIP3 genetic locus are associated with allergy and asthma risk in children living on farms. Thus, a complex interaction between genes and environment controls the precise epithelial threshold for innate immune activation to inhaled allergens, and subsequent development of allergic diseases.

Biography

Bart N. Lambrecht obtained an MD (1993) and PhD (1999) in Medicine at Ugent and specialized in Pulmonary Medicine (2002) at Erasmus University Medical Center in Rotterdam, The Netherlands. In 2005 he became Professor of Medicine at ErasmusMC, and in 2007 returned to Belgium on an Odysseus grant of the Flemish government, and became professor of Pulmonary Medicine at UGent and UZ Gent. In 2012 he became director of the VIB Inflammation Research Centre (IRC) in Gent. He is an ERC grant awardee and serves as associate editor of Mucosal Immunology, Trends in Immunology and advisory editor of Journal of Experimental Medicine. He has (co)authored more then 200 papers in the field of asthma and allergy. The thematic area of his group is centered around unraveling the role of antigen presenting cells in the lungs. In 2014, he won the famous Scientific Francqui award.

http://www.dmbr.ugent.be/

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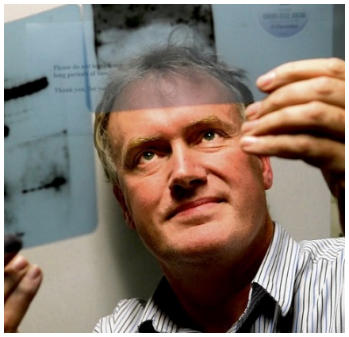

Luke O'Neill

School of Biochemistry and Immunology, Trinity Biomedical Sciences Institute, Trinity College Dublin

Wednesday, October 14 2015, 15:45 – 16:15

Metabolic reprogramming in innate immunity

Metabolic changes triggered by innate immune receptors have become a recent focus for researchers interested in immunity and inflammation. LPS-activated macrophages undergo metabolic reprogramming with a major increase in glycolysis, which is required for ATP production and also the generation of biosynthetic intermediates. Changes in the TCA cycle also occur such that intermediates such as citrate are withdrawn for lipid biosynthesis. We have found a role for the Kreb's cycle intermediate succinate in activated macrophages. Succinate has 4 potential roles here – 1. induction of HIF-1alpha and it's target genes, which include that encoding IL-1beta; 2. Histone and DNA modification via effects on demethylases; 3. Activation of the succinate receptor SUCRN1 on cells (which can synergise with TLRs) and 4. Reverse electron transport (RET) via Complex I in the mitochondria. Succinate might therefore act as important signal for inflammation. We have also found that interference of RET leads to IL-10 production, indicating that the direction of electron flow in the inner mitochondrial membrane may be an arbiter of cytokine production. The LPS-activated macrophage therefore has a Krebs cycle that is broken (and in essence is not a cycle at all) and has chemiosmosis possibly acting in reverse. These insights are providing a new view of metabolism in immunity and inflammation and might indicate new therapeutic approaches.

Biography

Professor Luke O'Neill was appointed to the Chair of Biochemistry at Trinity College Dublin in 2008, where he leads the Inflammation Research Group. He has a PhD in Pharmacology from the University of London and carried out Post-Doctoral research at Cambridge U.K. on the pro-inflammatory cytokine IL-1 and innate immune signaling. His research is in the area of the molecular basis to inflammatory diseases, with a particular interest in Toll-like receptors, inflammasomes and metabolic control of inflammatory cell signaling. He has won numerous awards for his research, notably the Royal Irish Academy Medal for Biochemistry, the Royal Dublin Society/ Irish Times Boyle medal for Scientific Excellence in 2014 the European Federation of Immunology Societies Medal. He was elected a member of EMBO in 2005. In 2014 he was named by Thompson Reuters as one of the world's most influential scientists, being in the top 1% in both Immunology and Pharmacology/Toxicology. He is a European Research Council Advanced Grant Holder and is co-founder and director of Opsona Therapeutics, a drug development company working in the area of Toll-like receptors.

https://www.tcd.ie/Biochemistry/research/l_o_neill.php

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Federica Sallusto

Cellular Immunology Laboratory and Center of Medical Immunology, Institute for Research in Biomedicine, University of Italian Switzerland, Bellinzona

Wednesday, October 14 2015, 16:15 – 16:45

The human T cell response in health and disease

We study the human system to address fundamental questions in the context of the immune response to different classes of antigens, such as microbial pathogens, allergens or self-antigens, to gain insights into mechanisms that induce host protection or immune-mediated pathology. Our efforts towards human immunology have resulted in the development of high-throughput cell-based screening methods to analyze the human immune response and, in particular, the functional diversity and repertoire of human effector and memory T cells. The T cell library method allows the efficient and high-throughput interrogation of the human T cell repertoire without being limited by the complexity of the antigen, the HLA haplotype (as it is the case of the HLA-tetramer technology) and the sample size (that is often small when obtained from patients or from donors in clinical trials). The method can be used to predict antigenicity and identify T cell epitopes to guide design of T cell vaccines or optimization of biologicals. When applied to the study of T cells in cancer and autoimmunity, it can provide tools for immunotherapy and insights as to the mechanisms of immune-mediated pathology. The T cell library method, together with high throughput TCR Vβ sequencing, which can analyze million of T cell clonotypes in blood or tissues, mass spectrometry, and single-cell gene expression analysis, can provide a wealth of information that is expected to contribute to the progress in medical immunology research.

Biography

Federica Sallusto is expert in the field of human cellular immunology. Her main contributions deal with mechanisms that regulate migration and effector function of T lymphocytes. These studies allowed to define Th1, Th2, Th17 and Th22 cells on the basis of chemokine receptor expression and to distinguish central and effector memory T cells. Recently, she clarified mechanisms that generate Th17 lymphocytes with different inflammatory capacity and demonstrated that, within a single clone, T cells can acquire different functional properties. Her studies in experimental models clarified mechanisms controlling lymphocyte migration in lymph nodes and brain and generation of follicular helper T cells.

http://www.irb.usi.ch/cellular-immunology

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Alain Fischer
2001 Louis-Jeantet Prize winner

1. Paris Descartes – Sorbonne Paris Cité University, Imagine Institute, Paris
2. Immunology and Pediatric Hematology Department, Assistance Publique-Hôpitaux de Paris, Paris
3. INSERM UMR 1163, Paris
4. Collège de France, Paris

Wednesday, October 14 2015, 16:45 – 17:15

Keynote lecture: **Primary immunodeficiency diseases: from molecular mechanisms to therapy**

A flurry of monogenic defects of immunity has been characterized over the recent years. They are instrumental in providing key information on the human immune system in a reductionist perspective. They also contribute to develop new therapies. This was the case more than 50 years ago with allogeneic hematopoietic stem cell transplantation. It seems that it is today the case with gene therapy. Understanding pathophysiology of severe combined immunodeficiencies led to conceptualize that any transduced lymphoid progenitor cell will benefit from a tremendous repopulating selective advantage. This was proven over the last 16 years to be effective. Few progenitors (~ 1000) can generate in a sustainable manner a fully diversified T cell repertoire. Gene transfer technology has evolved to improve both safety and efficacy, today making feasible an extension of its usage to treat many more primary immunodeficiencies and other hematopoietic genetic defects as well.

Biography

Dr Alain Fischer studied medicine, with a specialization in pediatrics and immunology at the Université of Paris. After completing a postdoctoral fellowship at the University College London, he started independent research in an INSERM unit at the Necker Hospital in Paris. Since 2009, he is the director of the Institute for Genetic Diseases (Imagine) at Necker University Hospital. Dr Fischer also served as a professor of pediatric immunology at the Université Paris Descartes. From 1996 to 2012, he has served as the director of the pediatric immunology department at the Necker Hospital. Dr Fischer is presently Professor at College de France. Dr Fischer's main research interests are gene therapy, primary immunodeficiency diseases, and the development of the lymphoid system. Dr Fischer received the Louis Jeantet Prize for Medicine in 2001 and the Japan Prize 2015.

http://www.institutimagine.org