6th Annual
IMMUNE IMAGING SYMPOSIUM

Hosted by:
THE PROGRAM FOR ADVANCED IMMUNE BIOIMAGING, UNIVERSITY OF ROCHESTER

THE CORNELL CENTER FOR IMMUNOLOGY, CORNELL UNIVERSITY

Saturday, November 12th, 2022

College of Veterinary Medicine
Cornell University

About our program:

PROGRAM FOR ADVANCED IMMUNE BIOIMAGING

Deborah Fowell[1], Minsoo Kim[2], David Topham[2], Patrick Oakes[3], Jim Miller[2], Nozomi Nishimura[4]

[1]Department of Microbiology and Immunology, Cornell University, Ithaca NY; [2]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester NY; [3]Department of Cell & Molecular Physiology, Loyola University,Chicago IL; Meinig School of Biomedical Engineering, Cornell Univeristy, Ithaca NY.

Pathogen control ultimately requires the recruitment and activation of innate and adaptive immune effectors to specific infected tissue microenvironments. While we have gained much insight into effector T cell generation in lymphoid tissues there exists a significant knowledge gap on the fate of effector T cells once they leave the lymph node. The ability of T cells to sense and interpret different inflammatory environments in infected or damaged tissues is poorly understood. Yet it is within the inflamed tissue milieu that T cells must mediate their effector functions, including cytokine secretion and cytolysis, to clear infection. The central premise of this program is that the specific tissue and the local inflammatory milieu will shape T cell recruitment and effector function. Such tissue-control is likely to impact the magnitude and functional diversity of the immune response. Optimizing T cell function in tissues is critical for pathogen clearance and the avoidance of collateral damage. The goal of this program is to define the checkpoints and identify molecular interactions that guide successful immunity at sites of inflammation. The objective is to bring together scientific expertise in migration, effector function and tissue structure to address fundamental effector T cell processes in infected tissues using cutting-edge intra-vital imaging approaches.

6th Annual Immune Imaging Symposium
Saturday, November 12th, 2022 8:15 a.m. – 5:15 p.m.

8:15 - 8:50 a.m. REGISTRATION, Poster set-up, Continental Breakfast

Session 1: Visualizing Immunity
(Chair: Deborah Fowell)

8:50 - 9:00 a.m.,
DEBORAH FOWELL - *WELCOME AND INTRODUCTION*

9:00 - 9:40 a.m.
RONALD GERMAIN, NIH
Visualizing Immunity – Moving From Dynamics to High Content Methods in 2D and 3D and Back

9:40 – 9:55 a.m.
SHORT TALK: **Menansili Mejooli**, Cornell University
Hyperspectral multiphoton microscopy for in vivo visualization of complex multi-cellular interactions

9:55 - 10:35 a.m.
CHRIS XU, Cornell University
Long wavelength three-photon imaging of mouse lymph node and spleen

10:35 – 10:50 a.m.
SHORT TALK: **Alexia Caillier**, Loyola University
T cells switch between integrin-dependent and integrin-independent migration modes to migrate in complex environments

10:50 - 11:20 a.m. Coffee Break

Session 2: Innate Cell Dynamics
(Chair: Minsoo Kim)

11:20 a.m. - 12:00 p.m.
CAROLE PARENT, University of Michigan
Exosomes as key regulators of neutrophil chemotaxis

12:00 – 12:15 p.m.
SHORT TALK: **Sangwoo (Steven) Park**, Cornell University
Nanoscale physical barrier by cellular mucin protects cancer cell from immune cell attack

12:15 – 12:55 p.m.
MILKA SARRIS, University of Cambridge, UK
Signaling dynamics of neutrophil migration at sites of tissue damage

1:00 – 2:15 pm
LUNCH
POSTER VIEWING & IMAGE CONTEST VOTING
Odd numbered posters 1:15-1:45
Even numbered posters 1:45-2:15

Session 3: Immune Regularion in Tissues
(Chair: David Topham)

2:15 – 2:30 p.m.
SHORT TALK: **Noor Bala**, Cornell University
Perivascular Chemokine-Rich Niches Drive Spatially Restricted T-cell Activation in Inflamed Tissues

2:30 – 3:10 p.m.
JENS STEIN, University of Fribourg, Switzerland
Analyzing CD8+ T cell biology in the tissue context

3:10 – 3:25 p.m.
SHORT TALK: **Kun He**, University of Pittsburgh
Autocrine paracrine pro-inflammatory IL-10 initiates lung-specific Th2 responses to inhaled allergen

3:25 – 4:05 p.m.
ULRICH VON ANDRIAN, Harvard University
Neuro-immune interactions in barrier tissues

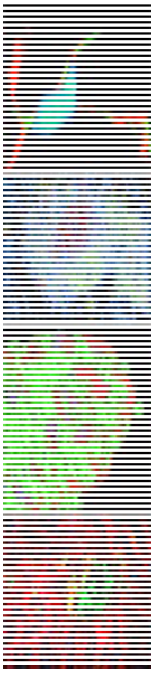
4:05 – 4:15 p.m.
POSTER AWARDS

4:15 – 5:15 p.m.
WINE AND CHEESE RECEPTION

9:00 - 9:40 a.m.

RONALD GERMAIN, M.D., Ph.D.
Chief, Laboratory of Immune System Biology & Lymphocyte Biology Section
National Institute of Allergy & Infectious Diseases, Bethesda

Visualizing Immunity – Moving From Dynamics to High Content Methods in 2D and 3D and Back



RESEARCH INTERESTS

Ronald N. Germain received his M.D. and Ph.D. from Harvard University. Since then he has investigated basic immunobiology, first on the faculty of Harvard Medical School, then in the Laboratory of Immunology, NIAID, NIH, and most recently at NIAID, NIH as Chief of the Laboratory of Immune System Biology. He has made key contributions to understanding MHC class II molecule structure–function relationships, the cell biology of antigen processing, the molecular basis of T cell recognition, and the application of systems biology to understanding immune function. More recently, his laboratory has explored the immune system using dynamic and static *in situ* microscopic methods that his laboratory helped pioneer. He has published more than 400 scholarly research papers and reviews. Among numerous honors, he was elected Associate member of EMBO (2008), elected to the National Academy of Medicine (2013), received the Meritorious Career Award from the American Association of Immunologists (2015), chosen as NIAID Outstanding Mentor (2016), elected to the National Academy of Sciences (2016), designated an NIH Distinguished Investigator and named a Distinguished Fellow of the AAI. He has trained more than 70 postdoctoral fellows, many of whom hold senior academic and administrative positions at leading universities and medical schools.

9:40 - 9:55 a.m.
SHORT TALK: **MENANSILI MEJOOLI**, Cornell University

Menansili A. Mejooli, Michael L. Buttolph, Yishai Eisenberg, Chi-Yong Eom, Frank W. Wise, Nozomi Nishimura, Chris B. Schaffer.

Hyperspectral multiphoton microscopy for in vivo visualization of complex multi-cellular interactions

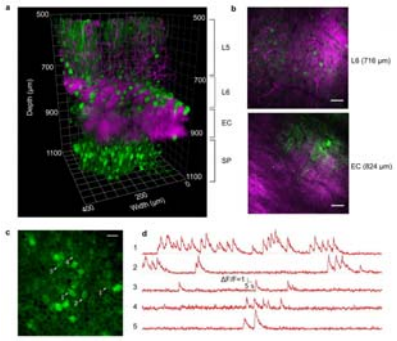
Normal and disease-state physiology studies would benefit from the capability to visualize a broad variety of cell types simultaneously, *in vivo*. Two-photon excited fluorescence (2PEF) microscopy has become the technique of choice for visualization of fluorescently labeled features deep into scattering samples (~1 mm in mouse cortex), at subcellular resolution. However, poor spectral resolution hinders the use of current microscopes for *in vivo* imaging of more than a few fluorescent markers, making the simultaneous study of multiple cell types difficult. To date, increasing spectral resolution in 2PEF imaging has largely relied on using diffraction gratings and prisms to spectrally disperse emitted florescence, which is only suitable for collimated fluorescence emission exiting the objective. For deep *in vivo* imaging, the light exiting the objective is highly uncollimated due to tissue associated scattering, and 2PEF systems relying on spectral dispersion are unsuitable. In previous work *(Bares et al., 2020,)* we built a hyperspectral multiphoton microscope (HMM) to address the challenge of achieving clear separation of multiple fluorescent species while maintaining the deep imaging capability of 2PEF. We imaged a model of inflammation in the mouse ear to demonstrate the application of the HMM in imaging the cellular interactions governing immune response. The HMM relies, in part, on sequentially taking images with several different excitation wavelengths to distinguish different fluorophores. Changing excitation wavelength frame-to-frame limits HMM imaging speed, limiting the ability to study fast biological and cellular dynamics. Slow imaging speed may also cause motion related artifacts to impact accurate fluorescent species identification - live sample breathing or movement between frame acquisition during laser switching causes pixel misregistration across excitation colors that confounds the linear unmixing used to assign pixels to specific fluorophores. We are now constructing HMM 2.0 that will excite fluorophores in five different spectral bands - three spectrally distinct femtosecond pulses that are cycled through from pulse to pulse (degenerate excitation), and simultaneous pairs of different wavelength pulses providing the other two spectral bands (non-degenerate excitation). With pixel level switching of laser excitation wavelength, the detected fluorescence is tagged to the corresponding excitation color. With this change, the HMM 2.0 will increase hyperspectral imaging speed by ~10X and will completely avoid misalignment between different excitation colors due to sample motion. We envision HMM 2.0 to have several applications, including in studies of central nervous system and immune response where multiple cell types and tissue components need to be visualized simultaneously to understand biological mechanisms.

9:55 - 10:35 a.m.

CHRIS XU, Ph.D.
Director, School of Applied & Engineering Physics
Cornell University, Ithaca

Long wavelength three-photon imaging of mouse lymph node and spleen

RESEARCH INTERESTS



Our research has two main thrusts: biomedical imaging and fiber optics. We are exploring new concepts and techniques for in vivo imaging deep into scattering biological specimens, such as mouse brain. We are developing new medical endoscopes for non-invasive real-time diagnostics of tissues without any exogenous contrast agent. We are developing novel optical fibers and fiber-based devices for biomedical imaging and optical communications.

Laser scanning multiphoton microscopy (MPM) has greatly improved the penetration depth of optical imaging and is proven to be well suited for a variety of imaging applications deep within intact or semi-intact tissues. Nonetheless, MPM has so far been restricted to ~ 1 mm in depth in brain tissues, which is only about a quarter of the ~ 4 mm cortex thickness of human. We are currently developing new concepts and techniques for imaging deep into scattering biological specimens.

Optical endoscopes have played a major role in medical diagnostic and minimally invasive surgery by making it possible to visualize tissue at remote internal sites. In our current research, we are exploring new concepts and devices that would significantly improve the performance of existing medical endoscopes, both rigid and flexible. Our goal is to create multiphoton endoscopes for non-invasive real-time diagnostics via 'optical biopsies' without any exogenous contrast, providing guidance for biopsy devices for more accurate sampling, and assessing surgical margins following tumor resection.

We are developing novel optical fibers to generate energetic pulses for multiphoton deep tissue imaging and coherent Raman scattering imaging. We are also developing novel optical fiber based devices to support our medical multiphoton endoscope effort so that high performance imaging can be achieved in a miniature endoscope.

10:35 – 10:50 a.m.
SHORT TALK: **ALEXIA CAILLIER**, Loyola University

Alexia Caillier, David Oleksyn, Jim Miller, Patrick Oakes

T cells switch between integrin-dependent and integrin-independent migration modes to migrate in complex environments

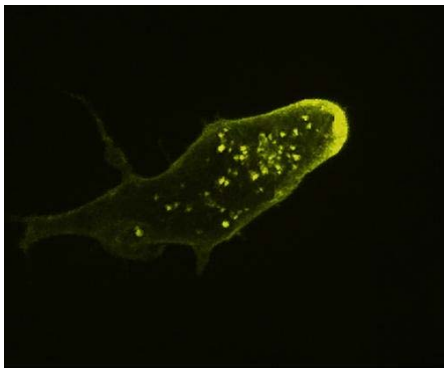
Lymphocyte migration is essential to ensure a quick and efficient immune response. As they screen the body for antigen presenting cells, they are exposed to different complex environments
composed of various extracellular matrix (ECM) components, architectures, and density. While previous work in vitro has shown that immune cells can migrate without integrin-dependent
adhesion, other studies in vivo have shown that integrin-dependent adhesion is required to induce the migration of lymphocytes. This suggests that lymphocytes have multiple mechanisms of migration and can adapt to their environment, fluctuating between integrin-independent to integrin-dependent migration as needs of the environment demand. Using primary t cells, we find that confinement is necessary to induce rapid and robust migration, independent of ECM composition. The presence of ECM proteins, however, does lead to a subtle but significant difference in migration speed and persistence. We also find in the presence of ECM proteins; T cells form focal adhesions that include clusters of integrins and adaptor proteins such as vinculin. When T cells are confined between two deformable surfaces, we can see that focal adhesions coincide with regions of traction stress as measured using Traction Force Microscopy. We find that traction stresses are exerted on both surfaces, though on occasion cells will pull only on one side, while pushing the other surface out of the way. Finally, using micropatterned surfaces we find that cells can switch between integrin-independent to integrindependent migration modes depending on whether the ECM coating is present. Together these data illustrate that t cells are able to effortlessly switch between integrindependent and integrin-independent migration to maintain efficient migration, navigate a wide range of complex environments, and facilitate effective an immune response.

11:20 a.m. - 12:00 p.m.

CAROLE PARENT, Ph.D.
Professor, Life Sciences Institute & Medical School
University of Michigan, Ann Arbor

Exosomes as key regulators of neutrophil chemotaxis

RESEARCH INTERESTS



Carole A. Parent studied at the Université de Montréal, Canada, the University of Illinois at Chicago, USA, and the Johns Hopkins University, Baltimore, Maryland, USA, before joining the National Cancer Institute, Rockville, Maryland, USA, as a principal investigator in 2000. She was tenured in 2006, appointed Adjunct Professor in the Institute for Physical Science and Technology at the University of Maryland College Park, USA, in 2011 and served as the Deputy Director of the Center for Cancer Research until 2017, when she moved to the University of Michigan, Ann Arbor, USA, as the Raymond Ruddon Collegiate Professor of Cancer Biology and Pharmacology. In 2021, Dr. Parent was appointed Research Professor in the Life Sciences Institute at the same institution. Using a plurality of model systems, along with a transdisciplinary approach, her research aims to understand how cells detect and respond to external chemotactic signals and, in particular, how the spatial and temporal relay of chemotactic signals between cells impacts single and group cell migration in the context of inflammation and cancer invasion.

12:00 – 12:15 p.m.

SHORT TALK: **Sangwoo (Steven) Park**, Cornell University

Sangwoo Park, Marshall Colville, Carolyn Shurer, Ling-Ting Huang, Joe Kuo, Justin Paek, Marc Goudge, Jin Su, Matthew DeLisa, Jan Lammerding, Warren Zipfel, Claudia Fischbach, Heidi Reesink, Matthew Paszek

Nanoscale physical barrier by cellular mucin protects cancer cell from immune cell attack

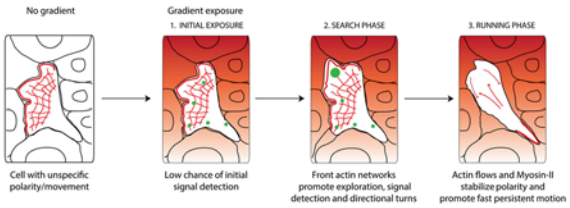
Cancer cells construct a glycocalyx with biochemical and physical attributes that protectagainst immune surveillance. Whether the structural properties of the glycocalyx also physicallyshield cancer cells from immune recognition has not been fully resolved. Here, we havedeveloped interference-based imaging tools called Scanning Angle Interference Microscopy(SAIM) to image the nanoscale physical dimensions and structural organization of the cellularglycocalyx. To improve the precision of SAIM for glycocalyx research, we utilized a pair of high-speed, galvanometer-controlled mirrors to generate a revolving circle or "ring", of excitation light at defined sample incidence angles (Ring-SAIM). By combining genetic approaches and the imaging tools, we reveal how the surface density, glycosylation, and crosslinking of cancer-associated mucins contribute to the nanoscale material thickness of the glycocalyx, and further analyze the effect of the glycocalyx thickness on resistance to effector cell attack. We uncovered a strong reciprocal relationship between the thickness of the glycocalyx and immune cell killing. Natural Killer (NK) cell-mediated cytotoxicity exhibits a nearly perfect inverse correlation with the glycocalyx thickness of target cells regardless of the specific glycan structures present, suggesting that the physical properties of glycocalyx may be key determinants of cancer immune evasion. Similar relationships were found for chimeric antigen receptor (CAR) NK cells against target cells with engineered glycocalyces. We further suggest strategies for improved penetration of mucin-rich glycocalyx barrier through glycocalyxediting enzymes on engineered NK cell surface.

12:15 – 12:55 p.m.

MILKA SARRIS, Ph.D.
Principal Investigator, Department of Physiology, Development & Neuroscience
University of Cambridge, United Kingdom

Signaling dynamics of neutrophil migration at sites of tissue damage

RESEARCH INTERESTS



Milka Sarris did her PhD studies with Alex Betz at the LMB, where she studied the formation of immunological synapses between immune cells in mouse models. She was awarded her PhD in 2008 and then joined the group of Philippe Herbomel in Institut Pasteur in Paris for her post-doctoral studies, where started working with zebrafish on the fundamentals of leukocyte movement mechanisms in vivo. In 2014 she was awarded an MRC Career Development award to set up her independent group in the Department of Physiology, Development and Neuroscience in Cambridge (PDN). She was then appointed as Assistant Professor in PDN and Fellow in Trinity College in 2016. Her group exploits high-end imaging, genetic and optogenetic manipulations to dissect leukocyte guidance and signalling dynamics in vivo.

2:15 – 2:30 p.m.
SHORT TALK: **Noor Bala**, Cornell University

Noor Bala, Hen Prizant, Deborah Fowell

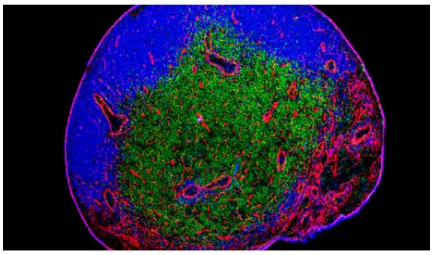
Perivascular Chemokine-Rich Niches Drive Spatially Restricted T-cell Activation in Inflamed Tissues

Pathogen control requires T-cells to locate and make direct contact with antigenpresenting cells (APCs) for peripheral reactivation and delivery of effector molecules. The signals that orchestrate T-cell-APC interactions are poorly understood in inflamed peripheral tissues. Using intravital multiphoton microscopy (IV-MPM), we have found that Th1 cell entry into the inflamed dermis is directed by perivascular CXCL10+ cellular clusters enriched for APCs. Th1 cells form more stable interactions with APCs within these clusters compared to outer regions. CXCL10+ clusters are amplified in a IFNγ-dependent manner, where CD4+T-cell IFNγ release increases APC recruitment and CXCL10 expression. We hypothesize that these chemokine clusters serve as CXCL10+ Peripheral Activation (PAC-10) niches, where Th1 cells achieve early peripheral activation away from the pathogen modulated milieu. We seek to determine the relationship between these spatially restricted PAC-10 niches, infection foci, and pathogen clearance. Using a combination of IV-MPM, flow cytometry, and single cell transcriptomics we characterized the cellular composition of the niche and the specific transcriptional program induced by activation in the chemokine-rich niche. Initial studies reveal that niche specific Th1 cells have increased CD69 and IFNγ expression compared to non-niche counterparts. PAC-10 niches appear to have a spatially distinct innate cell immune population that display increased expression of chemokines responsible for APC recruitment, likely responsible for the nucleation of the Th1-activating niche environment. Future studies will characterize the functional status of niche specific Th1 cells and evaluate the necessity of PAC-10 niches for pathogen control. These studies will inform therapeutic strategies to boost T-cell activation in infection or hinder activation in autoimmunity.

2:30 – 3:10 p.m.

JENS STEIN, Ph.D.
Professor, Department of Medicine
University of Fribourg, Switzerland

Analyzing CD8+ T cell biology in the tissue context



RESEARCH INTERESTS

The adaptive immune system protects us from harmful microbial infections and cancer, while providing life-long immunity after vaccination. To accomplish this extraordinary feat, cellular components of the immune system, T and B cells, continuously interact with antigen-presenting cells (APCs) in lymphoid organs. A well-studied example are naïve $CD8^+$ T cells interactions with dendritic cells (DCs), the most powerful APCs for this subset. This leads to $CD8^+$ T cell activation, differentiation to cytotoxic effector cells and invasion of infected organs. This process contributes decisively to elimination of intracellular pathogens such as viruses, as well as tumor cells. After clearing of a pathogen, memory $CD8^+$ T cells patrol the body to protect from reinfection. While the general principle of such adaptive immune responses is well established, little is known on how this dynamic process unfolds on a single cell level in the context of tissue-derived environmental cues.Our laboratory is combining multiple platforms including multicolor flow cytometry, functional in vitro assays and high-end microscopy to "shed light" on the molecular and cellular processes that govern adaptive immune responses mediated by cytotoxic $CD8^+$ T cells. We follow three lines of investigation:

- We are examining the role of key regulators of T cell activation by using genetically modified $CD8^+$ T cells. Our technical platforms include flow cytometry, RNA sequencing, viral infection models, immunofluorescent analysis and twophoton microscopy (2PM) of lymphoid tissue. Using software-based analysis of key parameters, we determine the critical decision-making steps at the onset of immune responses.
- We follow $CD8^+$ T cells at their effector sites, for example in exocrine glands, skin and other non-lymphoid organs and observe how these cells contribute to host protection. A special focus is on tissue-resident memory T cells that provide a first line of defense against reinfection.
- We are applying large-scale imaging techniques, Optical Projection Tomography (OPT) and Selective Plane Illumination Microscopy (SPIM) for a quantitative analysis of adaptive immune responses by visualizing the entire 3D structure of lymph nodes and other organs during inflammation.

The combination of these approaches permits to obtain unprecedented insight into the dynamic nature of the adaptive immune system on a single cell level.

3:10 – 3:25 p.m.
SHORT TALK: **Kun He**, University of Pittsburgh

Kun He, Zhongli Xu, William A. MacDonald, Anuradha Ray, Wei Chen, Bart Lambrecht and Amanda C. Poholek

Autocrine paracrine pro-inflammatory IL-10 initiates lung-specific Th2 responses to inhaled allergen

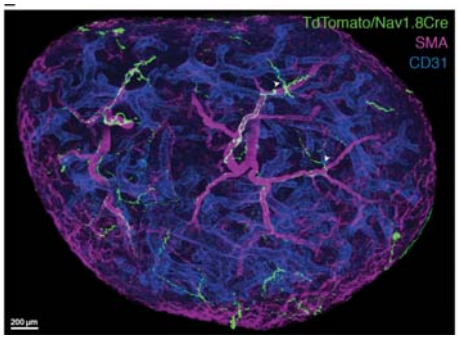
Allergic asthma remains a significant health burden for both children and adults in developed and developing nations. However, it remains unclear how inhaled allergens initiate the Th2 cell response that is critical for mediating allergic asthma. We demonstrated a distinct requirement for the transcriptional repressor Blimp-1 to promote Th2 cell development in the lung to inhaled but not systemically or subcutaneously delivered allergens. Applying flow cytometry, RNAScope, multiplexed imaging, spatial and single cell transcriptomic tracking of house dust mite (HDM) specific T cell responses with Blimp-1 YFP reporter, we found Blimp-1 YFP-producing cells initiated very early in the mediastinal lymph node (mLN) bore characteristics of T effector cells but not T follicular helper cells (Tfh). Early expression of Blimp-1 was required for Th2 cell development, and in HDM-specific CD4+ T cells drove GATA3 expression that coincides with IL-2Rα in the mLN during the first 3 days of priming. Spatial transcriptomics combined with single cell transcriptomes inferred distinct microniches of immune cell types in the mLN that changed between 3 and 5 days after HDM priming. To understand factors that may drive early expression of Blimp-1 in T cells, we explored sources of IL-10, known to induce Blimp-1 in Th2 cells. Using genetic deletion strategies and confirmed via imaging, we found IL-10 from T cells, but not other relevant IL-10 sources, acts directly on HDM-specific T cells driving the Blimp-1-dependent Th2 cell response. Furthermore, HDM-specific CD4+ T cell derived IL-10 was sufficient to promote Blimp-1 expression an autocrine/paracrine manner. These data highlight lung-specific factors that initiate the Th2 cell response to inhaled environmental allergens.

3:25 – 4:05 p.m.

ULRICH VON ANDRIAN, M.D.
Professor of Immunopathology, Microbiology & Immunology
Harvard University, Boston

Neuro-immune interactions in barrier tissues

RESEARCH INTERESTS



My research seeks answers to the question how circulating blood cells find their way in the body. Directed migration of blood-borne cells to distinct target tissues can be observed in embryos as soon as the circulatory system is established and plays a critical role throughout life in numerous physiologic and pathologic conditions. Despite considerable progress in this field, it is still beyond the reach of even the most sophisticated in vitro methodology to simulate the complex interplay of physical, cellular, biochemical, and other factors that determine blood cell behavior in microvessels. Therefore, we employ intravital microscopy to study the molecular mechanisms of interactions between blood cells and the vascular wall by direct observation in anesthetized mice. Using this approach, we have demonstrated that blood cell homing to most target tissues requires an initial tethering step that leads to rolling in postcapillary venules and is followed by an activation step which, in turn, triggers stationary adhesion and emigration. Each of these three steps (i.e. 1. rolling; 2. chemotactic stimulation; and 3. firm arrest) involves distinct molecular pathways whose unique combination is the reason why certain blood cells migrate to a particular organ, whereas others don't. We are now dissecting the site-specific adhesion cascades that direct myeloid and lymphoid cells, hematopoietic stem cells and platelets to normal and diseased tissues. We have established models in mice that allow quantitative observations in Peyer's patches; gut; bladder; striated muscle; skin; pancreas; liver; knee joint; bone marrow; bone; and peripheral lymph nodes. The techniques for observing the latter three tissues were developed in my laboratory. Understanding how lymphocytes, in particular T cells, home to and migrate within peripheral lymph nodes is a major focus of my group. To this end, we are using a panel of mice that are genetically deficient in specific adhesion pathways. We have also generated transgenic mouse strains that express fluorescent proteins in distinct T cell subsets. We are using these mice to study how T cells differentiate into effector and memory subsets; how this differentiation affects their migratory properties; and how antigen-presenting dendritic cells influence these processes. T cells and dendritic cells can be visualized in the intra- and extravascular space by intravital microscopy using both single- and multi-photon fluorescence techniques. This allows us to dissect the trafficking behavior of these immune cells at a resolution and specificity that could not be achieved with other methods. Besides supervising and instructing the students and fellows in my laboratory (15-20 members), my current teaching activities include the co-direction and organization of the Immunology 201 and Immunology 202 semester courses for HMS graduate students. In addition, we have numerous collaborators in the Harvard community and elsewhere who perform intravital microscopy studies in our facility under my supervision. I also lecture regularly to HMS graduate students, postdoctoral fellows and staff about leukocyte adhesion, migration and homing.

POSTERS

Presenter(s) listed BOLD

1.	*Perivascular Chemokine-Rich Niches Drive Spatially Restricted T-cell Activation in Inflamed Tissues* **Noor Bala**[1], Hen Prizant[2], & Deborah J. Fowell[1] [1]Department of Microbiology & Immunology, Cornell University, Ithaca, NY, USA [2]David H. Smith Center for Vaccine Biology and Immunology, Aab Institute of Biomedical Sciences, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY, USA
2.	*Cooperativity of Oral Microbes in Evading Neutrophil Mediated Killing* **Kiana Bynum**, Michelle Panasiewicz and Jason Kay Department of Oral Biology, University at Buffalo, Buffalo NY
3.	*T cells switch between integrin-dependent and integrin-independent migration modes to migrate in complex environments* **Alexia Caillier**[1], David Oleksyn[2], Jim F. Miller[2], Patrick W. Oakes[1]; [1]Cell & Molecular Physiology, Loyola University Chicago, Maywood, IL, [2]Program for Advanced Immune Bioimaging, University of Rochester Medical Center, Rochester, NY.
4.	*Th1 effector function in inflamed tissue is mediated by spatiotemporal downregulation of CXCR3* Anastasia N. Rup[1], **Evan M. Carter**[1,2], Noah Salama[3], Noor Bala[1], Scott A. Leddon[1], and Deborah J. Fowell[1] [1]Department of Microbiology and Immunology, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853, USA [2]Harvard Extension School, Harvard University, Cambridge, MA 01238, USA [3]Division of Endocrinology and Metabolism, Department of Medicine, School of Medicine and Dentistry, University of Rochester, Rochester, NY 14642, USA
5.	*Use of tetrameric hemagglutinin probes for analysis of antigen-specific B cells in mouse and human lymphoid tissues* **Francisco Chaves**, Molly Niska, Preshetha Kanagaiah, Theresa Fitzgerald, Aizan Embong, Emma Reilly, Phuong Nguyen, Mark Sangster and David Topham Department of Microbiology and Immunology, University of Rochester, Rochester NY

6.	*Intravital Three Photon Imaging of Dynamic Lymphocytes in Lymph Node and Spleen* **Kibaek Choe** and Chris Xu Applied and Engineering Physics, Cornell University, Ithaca NY
7.	*The molecular mechanism of PMN-MDSC differentiation in the TME of Pancreatic Cancer Ductal Adenocarcinoma (PDAC)* **Ankit Dahal**, Raj Kumar Mongre, Yeonsun Hong, Cooper Sailer, Minsoo Kim David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester, New York 14642.
8.	*Autocrine paracrine pro-inflammatory IL-10 initiates lung-specific Th2 responses to inhaled allergen* **Kun He**[1,2], Zhongli Xu[1], William A. MacDonald[1], Anuradha Ray[2], Wei Chen[1], Bart Lambrecht[3] and Amanda C. Poholek[1,2] [1]Department of Pediatrics, University of Pittsburgh School of Medicine, Pittsburgh, PA 15261, USA. [2]Department of Immunology, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213, USA. [3]Laboratory of Mucosal Immunology and Immunoregulation, VIB-UGent Center for Inflammation Research, Ghent, Belgium.
9.	*In vivo CRISPR screen reveals how to drive therapeutic T cell function to solid tumors* **Yeonsun Hong** Department of Microbiology and Immunology, University of Rochester, Rochester NY
10.	*PDE10A is a key mediator of ferroptosis in macrophages* **Chia George Hsu**, Mark Sowden, Chen Yan, Bradford C Berk Department of Medicine, Aab Cardiovascular Research Institute, University of Rochester School of Medicine and Dentistry, Rochester, NY, USA.
11.	*Should I stay or should I go: Understanding how CD49a impacts lung resident memory CD8 T cell localization and function* **Taylor Jones**, Michael Sportiello, Adam Geber, and David Topham Department of Microbiology and Immunology, University of Rochester, Rochester NY.
12.	*HSV1 drives VEGFA production in macrophages via a ROS-HIF1a axis* **J. Connor Mahaney**, Matthew Evans, Anthony St. Leger David H. Smith Center for Vaccine Biology and Immunology, Department of Ophthalmology, University of Pittsburgh, Pittsburgh PA. Department of Immunology, University of Pittsburgh, Pittsburgh, PA.

13.	*moDC and Th1 Cooperation in the Formation of Peripheral Activation Niches* **Alexander McGurk**[1], Hen Prizant[2], Noor Bala[1], Noah Salama[2], and Deborah Fowell[1] [1] Department of Microbiology and Immunology, Cornell University, Ithaca, NY, USA [2] David H. Smith Center for Vaccine Biology and Immunology, Aab Institute of Biomedical Sciences, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.
14.	*Hyperspectral multiphoton microscopy for in vivo visualization of complex multi-cellular interactions* **Menansili A. Mejooli**, Michael L. Buttolph, Yishai Eisenberg, Chi-Yong Eom, Frank W. Wise, Nozomi Nishimura, Chris B. Schaffer. Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY. School of Applied and Engineering Physics, Cornell University, Ithaca, NY.
15.	*Lysophosphatidylserine Nanoparticles Induce LC3-Associated Phagocytosis* **Edwin Ovalle**[1], Sathy Balu-lyer[2], Jason G. Kay[1] [1]Department of Oral Biology, School of Dental Medicine, University at Buffalo, Buffalo, NY, USA [2]Department of Pharmaceutical Sciences, School of Pharmacy and Pharmaceutical Sciences, University at Buffalo, Buffalo, NY, USA
16.	*Cancer-associated mucins provide sustained physical protection against Natural Killer cell attack* **Justin H. Paek**[1], Sangwoo Park[2], Marshall J. Colville[3], Matthew J. Paszek[1,2,3] [1]Nancy E. and Peter C. Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY; [2]Field of Biophysics, Cornell University, Ithaca, NY; [3]Robert Frederick Smith School of Chemical and Biomolecular Engineering, Cornell University, Ithaca, NY
17.	*Nanoscale physical barrier by cellular mucin protects cancer cell from immune cell attack* **Sangwoo Park**[1], Marshall Colville[1,2,] Carolyn Shurer[2], Ling-Ting Huang[2], Joe Kuo[2], Justin Paek[3], Marc Goudge[3], Jin Su[4], Matthew DeLisa[2], Jan Lammerding[3,4,] Warren Zipfel[1,3,] Claudia Fischbach[3], Heidi Reesink[5], Matthew Paszek[1,2,3*] [1]Field of Biophysics, Cornell University, Ithaca, NY 14853, USA [2]Robert Frederick Smith School of Chemical and Biomolecular Engineering, Cornell University, Ithaca, NY 14853, USA [3]Nancy E. and Peter C. Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY 14853, USA

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18.	*CAR T Cell Migration to Solid Tumors* **Allison Ryan**, Cooper Sailer, Ankit Dahal, TashJaé Scales, Emily Whitt, Minsoo Kim Department of Microbiology and Immunology, University of Rochester, Rochester NY David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester NY
19.	*PD-1High CAR T cells exhibit superior effector functions in solid tumors* **Cooper Sailer**[1]**,** Yeonsun Hong[2], Ankit Dahal[2], Allison Ryan[2], Margaret Youngman[3], Minsoo Kim[1,2] [1] Pathology and Laboratory Medicine, URMC, Rochester, NY [2] Center for Vaccine Biology and Immunology, URMC, Rochester, NY [3]Department of Otolaryngology, URMC, Rochester, NY
20.	*Nfil3 plays a role in CD8 T cell function and differentiation* **Michael Sportiello** David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester NY
21.	*Collagen I Regulates Macrophage Polarization with Possible Effects on Tumor Malignancy in Obesity* **Regan Stephenson**[1], Benjamin Hopkins[2,3,] Yajas Shah[4], Akanksha Verma[4], Olivier Elemento[4], Cynthia Leifer[5], Claudia Fischbach[1,6,7] [1]Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY, [2]Genetics and Genomic Sciences, [3]Oncological Sciences, Mount Sinai, New York, NY, [4]Englander Institute for Precision Medicine, Weill Cornell Medicine, New York, NY, [5]Department of Microbiology and Immunology, Cornell University, Ithaca, NY, [6]Cornell Center on the Physics of Cancer Metabolism, Ithaca, NY, [7]Cornell NanoScale Science and Technology Facility (CNF), Ithaca, NY.
22.	*In Vivo Imaging of Cortical Microglia During Voluntary Wheel Running* **Alexandra Strohm, M.S.** [3], Thomas O'Connor M.S.[2], Robert Dirksen, PhD, [2] and Ania Majewska, PhD [1,4,5] [1]Department of Neuroscience, [2]Department of Pharmacology and Physiology, [3]Department of Environmental Medicine, [4]Center for Visual Science, [5]Del Monte Neuroscience Institute, University of Rochester, Rochester, NY

23.	*Cell metabolism regulates contractility* **Lee D Troughton**, Alexia Caillier, Jordan R Beach, and Patrick W Oakes. Cell and Molecular Physiology, Loyola University Chicago, Stritch School of Medicine, Chicago, Maywood, IL 60153, USA.
24.	*Effect of neutrophils on mesenchymal stromal cell motility.* **Adam Tyrlik**, Emily R. Quarato, Noah A. Salama, Jane Zhang, Yuko Kawano, Hiroki Kawano, Mary Chen, Laura M. Calvi Wilmot Cancer Institute; Department of Environmental Medicine; Department of Medicine; Center for Musculoskeletal Research; Department of Microbiology and Immunology; Department of Pathology and Laboratory Medicine University of Rochester Medical Center, Rochester, NY

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