3rd Annual
IMMUNE IMAGING SYMPOSIUM

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

Saturday, November 4th, 2017

Saunders Research Building
University of Rochester

About our program:

PROGRAM FOR ADVANCED IMMUNE BIOIMAGING

Deborah Fowell, Minsoo Kim, Jim Miller and David Topham
Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, 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.

3rd Annual Immune Imaging Symposium
Saturday, November 4th, 2017. 8:30 a.m. – 5:30 p.m.
Saunders Research Building and Helen Wood Hall Auditorium

8:30 - 8:55 a.m.
REGISTRATION, Poster set-up, Continental Breakfast

8:55 - 9:00 a.m.,
Deborah Fowell - *WELCOME AND INTRODUCTION*

9:00 - 9:40 a.m.
ANDREW LUSTER, Harvard University
Imaging Chemoattractant Function in Disease: New Roles for Old Mediators

9:40 - 9:55 a.m.
SHORT TALK: **Hen Prizant**, postdoc, University of Rochester
Local CXCL10 Expression Shapes Micro-Anatomical Positioning of Th1 Cells in the Inflamed Skin

9:55 - 10:35 a.m.
GILLIAN GRIFFITHS, University of Cambridge
Membrane Changes at the Immunological Synapse

10:35 - 10:50 a.m.
SHORT TALK: **Jan Martinek**, student, Jackson Labs, U. Conn
Quantitative Cellular and Molecular Imaging of the Intact Tumor Microenvironment

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

11:20 - 12:00 p.m.
GABRIEL VICTORA, The Rockefeller University
Monitoring Cell-Cell Interactions in vivo

12:00 – 12:15 p.m.
SHORT TALK: **Aliyah Weinstein**, student, University of Pittsburgh
Imaging Cytokine Biology in the Tumor Microenvironment: the Expression and Function of IL-36g in Colorectal Cancer

12:15 – 12:55 p.m.
DAVID MASOPUST, University of Minnesota
Immunosurveillance by Resident Memory T Cells

1:00 – 2:30 pm
LUNCH & POSTER VIEWING and 'People's Choice Award'

2:30 – 2:45 p.m.
SHORT TALK: **Rianne Stowell**, student, University of Rochester
Noradrenergic Modulation of Microglial Dynamics and Synaptic Plasticity

2:45 – 3:25 p.m.
CHRIS XU, Cornell University
Pushing the Imaging Depth of Multiphoton Microscopy

3:25 – 3:40 p.m.
SHORT TALK: **Brandon Walling**, student, University of Rochester
Chemokine Independent Effector CD8+ T cell Migration

3:40 – 4:20 p.m.
DAVID TOPHAM, University of Rochester
Establishment, Positioning & Maintenance of Tissue Resident Memory in the Influenza Infected Airway

4:20 – 4:30 p.m.
BioLegend POSTER AWARDS

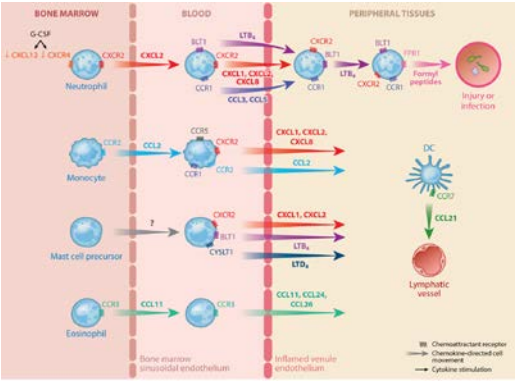
Leica IMAGING AWARD

4:30 – 5:30 p.m.
WINE AND CHEESE RECEPTION

9:00 - 9:40 a.m.

ANDREW LUSTER, MD, PhD
Professor, Center for Immunology and Inflammatory Diseases, Mass. General Hospital, Harvard University

Imaging Chemoattractant Function in Disease: New Roles for Old Mediators



RESEARCH INTERESTS

The Luster Laboratory focuses on understanding the role of chemokines and lipid chemoattractants and their receptors in controlling the trafficking of leukocytes in vivo. Gene-targeted and transgenic mouse strains have been developed to study the role of chemokines and chemoattractant receptors in organ-specific innate and adaptive immune and inflammatory responses in mouse models of inflammatory, autoimmune, allergic and infectious diseases. System biology approaches are being utilized to understand how multiple chemoattractant pathways are integrated in vivo for the fine control of leukocyte trafficking. In vivo multiphoton microscopy is being utilized to interrogate the function of chemokines in controlling the migratory and interactive behavior of T cells and dendritic cells in lymph nodes and peripheral tissue. We also study the regulation of chemokine production in vivo since this is a critical determinant of their role in a given biological response. We have a particular interest in the role of innate immune receptors, such as toll-like receptors, in regulating chemokine production in vivo. Finally, chemokines and chemoattractant receptors are interrogated in human diseases to determine chemokine systems relevant for disease pathogenesis.

9:40 - 9:55 a.m.
SHORT TALK: **Hen Prizant**, postdoc, University of Rochester

Local CXCL10 Expression Shapes Micro-Anatomical Positioning of Th1 Cells in the Inflamed Skin

Hen Prizant[1], Milan Popovic[1], Christopher Barilla[2], Deborah J. Fowell[1]
[1]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, U. of Rochester, [2]Bioinformatics, RIT, Rochester, NY

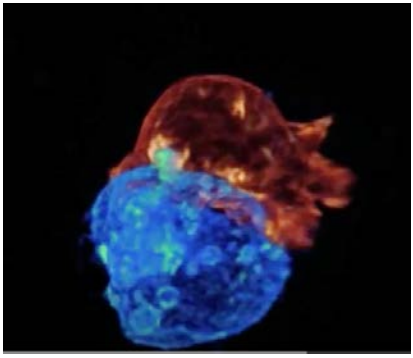
While T cell migration in infected tissues appears non-directional, efficient recruitment and positioning of T cells within infected tissues is crucial for productive antigen presentation and pathogen clearance. CXCR3 chemokines (CXCL9 and CXCL10) have been shown to influence the positioning of Th1 cells in the lymph node (Groom et. al. 2012), however it is not known whether Th1 cells are dependent on local CXCR3 chemokine cues for interstitial motility, localization and activation once in the inflamed tissue. Using real-time PCR and flow cytometry, we found that upon intradermal injection of CFA adjuvant and OVA protein to the ear pinnae, CXCL9 and CXCL10 are locally induced in the inflamed ear. Using a reporter mouse (REX3) where transcriptional expression of CXCL9 and CXCL10 are linked to fluorescent proteins and intra-vital multiphoton microscopy (IV-MPM) we identified the formation of CXCL10 expressing cell clusters located perivascularly around the site of inflammation in the ear dermis. Strikingly, Th1 cells preferentially localized to the CXCL10 rich clusters and computational analysis revealed the majority of Th1 cells (~72%) were positioned within 10μm from a CXCL10 expressing cell. Motility analysis of Th1 cells within the inflamed dermis revealed that Th1 cells migrated slower, displayed shorter displacements and paused more often in CXCL10 rich regions compared to CXCL10 poor regions. These data suggest a possible role for chemokines in controlling immune cell micro-anatomical accumulation by providing deceleration and/or arrest signals in areas of high chemokine production. One possible physiological consequence of this mechanism would be optimized T cell location to areas of antigen presentation. Indeed, using flow cytometry, we found that approximately 45% of CD11c+MHC-II+ expressing cells in the inflamed ear also express CXCL10. By IV-MPM, cell-cell contact analysis in 3D showed that Th1 cells were capable of forming stable interactions with CXCL10 expressing cells. These findings shed light on the function of CXCR3 chemokines in shaping Th1 positioning for activation and effector function. Understanding the fundamental micro-environmental cues for Th1 positioning and activation may be critical for the development of therapeutic approaches that optimize responses to vaccination and chronic infections.

9:55 - 10:35 a.m.

GILLIAN GRIFFITHS, **PhD**

Professor, Cambridge Institute for Medical Research, University of Cambridge

Membrane Changes at the Immunological Synapse



RESEARCH INTERESTS

Control of secretion at the immunological synapse. Cells of the immune system protect the body against pathogens. If cells in our bodies are infected by viruses, or become cancerous, then killer cells of the immune system identify and destroy the affected cells. Cytotoxic T cells (CTLs) are very precise and efficient killers. They are able to destroy infected or cancerous cells, without destroying healthy cells surrounding them. We aim to uncover the mechanisms controlling secretion from CTL and natural killer (NK) cells. By understanding how this works, we can develop ways to control the 'killer' cells of the immune system. This will allow us to find ways to improve cancer therapies, and ameliorate autoimmune diseases caused when killer cells run amok and attack healthy cells in our bodies.

Our laboratory is interested in understanding the mechanisms that control polarized secretion from cytotoxic T lymphocytes and NK cells. We use cutting-edge imaging, molecular, genetic and biochemical techniques to identify the proteins required for polarized secretion, and to understand the way in which they work.

Clinical collaborations have a very important role in identifying proteins required for secretion and understanding how these proteins work. Our studies have focused on Familial Hemophagocytic Lymphohistiocytosis (FHL), a disease in which secretion of lytic proteins from CTLs and NK cells is disrupted, and the cells fail to kill. Loss of perforin, Rab27a, Munc13-4, 18-2 and syntaxin 11 all lead to loss of CTL secretion and we wish to determine the interactions that allow these proteins to control polarized secretion from these cells.

10:35 - 10:50 a.m.
SHORT TALK: **Jan Martinek**, student, Jackson Labs, U. Conn

Quantitative cellular and molecular imaging of the intact tumor microenvironment.

Jan Martinek[1], Hannah Brookes[1], Te-Chia Wu[1], Kyung In Kim[1], Joshy George[1], Paul Robson[1,2] Jacques Banchereau[1,2,] and Karolina Palucka[1,2]
[1]The Jackson Laboratory for Genomic Medicine, Farmington, CT, [2]Dept. of Genetics & Genome Sciences, U. of Connecticut Health Center, Farmington, CT

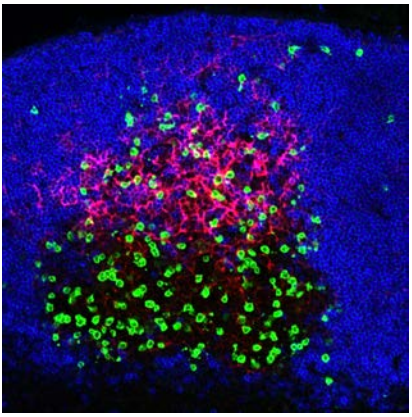
The causes underlying the extent and character of tumor-associated immune responses in cancer are not well defined and are likely multifactorial including cancer cell heterogeneity, host genotype, and the immune status of individual patients. Tumors are organized tissues with numerous reciprocal local and systemic connections with immune cell populations of both the myeloid and lymphoid lineages. Understanding the cellular and molecular composition of tumors could help uncover the variation in immune response to cancer in patients and to delineate interpatient patterns that could help elucidate causes of disease progression. Multi-parameter flow cytometry and single cell transcriptomics technologies have provided an unprecedented level of cell typing and annotation of cells present in tumors. However, tissue dissociation and single cell analysis do not enable the determination of cellular context in which the cells find themselves at the time of analysis. Given this, we have established a comprehensive approach to cellular and molecular analysis of intact non-dissociated tissues. To this end, we have developed polychromatic (10+) immunofluorescence staining protocols for frozen tissue sections that are analyzed by confocal microscopy and whole tissue scanning. Post-acquisition quantitation is based on adapted and improved histo-cytometry method based on Gerner et al (Immunity. 2012 Aug 24;37(2):364-76) with cell segmentation using Imaris software and a flow-cytometry like analysis conducted with Flowjo, allowing us to gate on different immune populations and subpopulations based on their phenotype while preserving each cell's original location within the tissue. In the next step, specific cells of interest are captured with laser capture microdissection and profiled with RNAseq. Transcripts derived by this computational pipeline are further analyzed using in-situ hybridization with ViewRNA to confirm cell expression and establish the cellular context. We are also implementing light-sheet microscopy for 3D assessment of tissue architecture and organization by a given molecule. Thus, we are generating extensive quantitative and qualitative "maps" of cancer, stromal and immune infiltrate allowing us to study its dynamic within the tissue.

11:20 - 12:00 p.m.

GABRIEL VICTORA, PhD
Assistant Professor, The Rockefeller University

Monitoring Cell-Cell Interactions in vivo

RESEARCH INTERESTS



The immune system has the peculiar ability to respond to foreign substances (or antigens) by producing antibody molecules that bind to these antigens with extremely high affinity and a remarkable degree of specificity. In order to achieve this high level of affinity, B cells – the cells that produce antibodies – must undergo a series of steps that culminate in the generation of an anatomical structure known as the germinal center (GC). Within this structure, B cells that have acquired affinity-enhancing mutations proliferate, and are eventually directed to differentiate into antibody-producing plasma cells or memory cells that can re-expand upon future contact with the same antigen. It is this process that allows vaccines to work. On the flip side, failures in the GC reaction can result in the production of high- affinity antibodies in allergy and autoimmunity.

In the Victora lab, we combine a number of cutting-edge techniques – from the development of novel mouse models to intravital multiphoton microscopy – to shed light on the intricacies of the GC reaction and its regulation. For example, using multiphoton-based geotagging of GC cells in a newly developed photoactivatable mouse, we have been able to define the cellular and molecular characteristics of different subpopulations of GC B cells, as well as their dynamic behavior and its relationship to selection. The characteristics we defined in mice are now being used in human studies to better understand the events leading to B cell lymphoma. We believe that unveiling the molecular mechanisms of the GC reaction will be essential if we wish to design better vaccines, develop treatments for allergies and autoimmune diseases, and dissect the molecular basis of lymphomagenesis.

12:00 – 12:15 p.m.
SHORT TALK: **Aliyah Weinstein**, student, University of Pittsburgh

Imaging Cytokine Biology in the Tumor Microenvironment: the Expression and Function of IL-36g in Colorectal Cancer

Aliyah M. Weinstein[1,3], Nicolas A Giraldo[1], Catherine Julie[2], Laetitia Lacroix[1], Florent Petitprez[1], Jean-François Emile[2], Wolf H Fridman[1], Walter Storkus[3,4], Catherine Sautès-Fridman[1]
[1]Centre de Recherche des Cordeliers, Paris, [2]Laboratoire d'anatomie pathologique, Hopital Ambroise Paré, AP-HP, Boulogne, France, [3]U. of Pittsburgh Sch. of Med., Depts. of Dermatology & Immunology, Pittsburgh, PA, [4]U. of Pittsburgh Sch. of Med., Depts. of Pathology & Bioengineering, and U. of Pittsburgh Cancer Institute, Pittsburgh, PA

IL-36g/IL-1F9 is a member of the IL-1 superfamily of cytokines and a mediator of Type 1 immunity and a necessary mediator of tertiary lymphoid organ (TLO) formation in a murine model of colon carcinoma. When IL-36g was introduced therapeutically into the tumor microenvironment of MC38-bearing mice, tumors exhibited delayed progression in correlation with TLO formation; this was not observed in control-treated mice. Co-treatment with the IL-36g antagonist, IL-1F5, blocked the efficacy of the therapy. CD11c+ dendritic cells (DC) were observed to be a major source of IL-36g in the tumor microenvironment (TME), and signaling through IL-36R+ host cells was necessary for the efficacy of IL-36g-based treatment. In the present study, we sought to identify a role for IL-36g in the de novo formation of TLO, using a cohort of 33 patients with primary colorectal cancer. We analyzed the histologic localization and magnitude of expression of IL-36g, IL-1F5, CD20, CD138, CD8, CD31, PNAd, DC-LAMP, CD68, and Tbet using immunohistochemical and immunofluorescent imaging. Images were analyzed using Visiopharm and CaloPix software to compute the localization of IL-36g and IL-1F5 in various cell populations, and statistical analyses performed using R. Expression of IL-36g by the tumor vasculature positively correlates with CD20+ B cell density in TLO, but not the number of TLO observed in the tumor, indicating that IL-36g may play a role in the maintenance, not formation, of these structures. Neither observation holds for IL-1F5. Some IL-36g+ vessels appear to be HEV. While B cells are the primary cellular component of TLO, they do not produce IL-36g. Within the tumor, CD68+ macrophages are the major immune source of IL-36g. These results support that the pro-inflammatory conditions of colorectal cancer can promote a Type 1-polarized immune microenvironment; that IL-36g is secreted by diverse yet distinct cell populations in the TME; and that IL-36g may play a role in the maintenance of TLO and in lymphocyte recruitment in colorectal cancer.

This material is based upon research supported by the Chateaubriand Fellowship of the Office for Science & Technology of the Embassy of France in the United States.

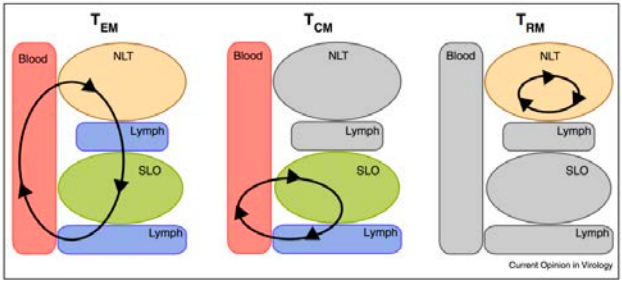
12:15 – 12:55 p.m.

DAVID MASOPUST, PhD

Professor, Dept. of Microbiology and Immunology, U. of Minnesota

Immunosurvelliance by Resident Memory T Cells

RESEARCH INTERESTS



My laboratory studies CD8 and CD4 T cell responses to a variety of viral and bacterial infections to help understand the development of immunological protection from re-infection. We observe and manipulate pathogen specific T cell responses over time by using MHC tetramers, adoptive transfer of transgenic T cells, fluorescence flow cytometry and sorting, and gene microarry analysis. Upon activation in lymphoid tissue, rare pathogen-specific naïve T cells proliferate, acquire effector functions, disseminate throughout the organism, and contribute to the eradication of pathogens. In situations where antigen is successfully eliminated, most effector T cells die by apoptosis. However, a fraction of effector T cells escape death and differentiate into long-lived memory T cells that contribute to protective immunity. We are currently dedicated to elucidating the developmental cues that govern T cell migration to different anatomical locations, commitment to the memory lineage, and the contribution of memory T cell differentiation state and location to protection from re-infection. Memory T cells that reside at common portals of pathogen entry or infection, especially the intestinal mucosa, are of particular interest. By understanding these issues, we hope to contribute to the development of better vaccination strategies, and are currently focused on informing development of a protective HIV vaccine.

2:30 – 2:45 p.m.
SHORT TALK: **Rianne Stowell**, student, University of Rochester

Noradrenergic Modulation of Microgial Dynamics and Synaptic Plasticity

Rianne Stowell, Ania Majewska
Department of Neuroscience, U. of Rochester Medical School, Rochester, NY.

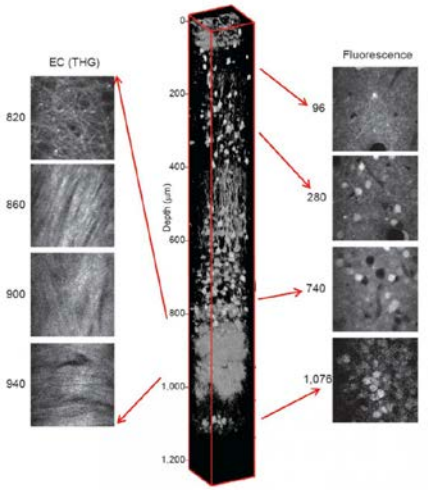
Microglia, the innate immune cells of the central nervous system (CNS), respond rapidly and dynamically to homeostatic perturbations of the CNS milieu. In the healthy unperturbed brain, microglial processes make frequent contacts with neurons at synapses, impacting synaptic remodeling and turnover of dendritic spines. However, it remains unclear what receptors and signaling pathways govern microglial surveillance and synapse monitoring. Noradrenaline is a powerful signal that can affect many aspects of synaptic function and plasticity. Because microglia express high levels of β2 adrenergic receptors (AR) compared to other cell types in the brain, we asked whether noradrenergic tone could alter microglial behavior with respect to synapses through β2 AR signaling. To test this hypothesis we have manipulated β2 AR signaling pharmacologically using the following agents: Nadolol (blood brain barrier (BBB) impermeant β AR antagonist), Clenbuterol (BBB permeant β2 AR agonist), and ICI 118-551 (BBB permeant β2 AR antagonist). We paired nadolol with clenbuterol to stimulate β2 ARs centrally without concomitant peripheral stimulation. We then evaluated changes in basic microglial physiology through a combination of in vivo two-photon microscopy and immunohistochemical staining for Iba-1, a microglia-specific protein. We have found that stimulation of β2 AR signaling in vivo reduces microglial motility and pseudopodia formation and causes microglia to assume a less ramified morphology. We also found that stimulation of β2 ARs leads to impaired microglial responsiveness to focal tissue injury. These experiments show that β2 AR signaling can affect microglial physiology and immune responses. Our next question was if these changes in basic microglial function could impact microglial interactions with neurons and functional experience dependent plasticity. Using intrinsic optical signal imaging we have shown that pharmacological manipulation of β2 AR signaling impairs ocular dominance plasticity in the visual cortex during the visual critical period in mice. We have shown that microglia are directly involved in this impairment through cre-mediated excision of β2 AR specifically in microglia. Based on our results we believe that β2 AR signaling serves important roles in modulating microglial physiology and our future experiments will begin to address how the endogenous ligand, norepinephrine, is involved in mediating the effects observed. These results and future findings will improve our understanding of the signaling mechanisms that govern microglial interactions with synapses and how they impact activity-dependent synaptic modifications.

2:45 – 3:25 p.m.

CHRIS XU, PhD
Professor, Dept. Applied & Engineering Physics, Cornell University

Pushing the Imaging Depth of Multiphoton Microscopy

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.



Two-photon fluorescence microscopy enables scientists in various fields including neuroscience, embryology and oncology to visualize *in vivo* and *ex vivo* tissue morphology and physiology at a cellular level deep within scattering tissue. However, tissue scattering limits the maximum imaging depth of two-photon fluorescence microscopy to the cortical layer within mouse brain, and imaging subcortical structures currently requires the removal of overlying brain tissue or the insertion of optical probes. We have demonstrated non-invasive, high-resolution, *in vivo* imaging of subcortical structures within an intact mouse brain using three-photon fluorescence microscopy at a spectral excitation window of 1,700 nm. Vascular structures as well as red fluorescent protein-labelled neurons within the mouse hippocampus can be imaged. The combination of the long excitation wavelength and the higher-order nonlinear excitation overcomes the limitations of two-photon fluorescence microscopy, enabling biological investigations to take place at a greater depth within tissue.

3:25 – 3:40 p.m.
SHORT TALK: **Brandon Walling**, student, University of Rochester

Chemokine Independent Effector CD8+ T cell Migration

Brandon Walling[1], Tara Capece[2], Nick Reilly[3], Patrick W. Oakes[3,4], Minsoo Kim[1]
[1]Department of Microbiology and Immunology, University of Rochester, Rochester NY,
[2]National Institute of Allergy and Infectious Diseases (NIAID), Bethesda, MD,
[3]Department of Physics and Astronomy, University of Rochester, Rochester NY,
[4]Department of Biology, University of Rochester, Rochester NY

Our current understanding of T cell migration is focused on chemokine-mediated integrin activation, which dictates that chemokines provide critical directional signals to guide T cell migration to target tissues within the body. Emerging evidence, however, supports that activated T cells can migrate to certain vascularized tissues, despite chemokine receptor deficiency or with the inhibition of chemokine receptors. This suggests the presence of a chemokine-independent mechanism through which T cells can activate integrins, induce adhesion and subsequent cell migration. We found with OT-I T cell receptor (TCR) transgenic mice that integrin LFA-1 mediated migration of naïve T cells was strictly dependent on chemokine stimulation. In contrast, activated CD8+ T cells migrated in the absence of chemokines both in vitro and in vivo. Additionally, activated CD8+ T cells migrate in the presence of the chemokine inhibitors pertussis toxin (PTx) and Gallein (a Gβγ inhibitor), further supporting chemokine-independent T cell migration. Using mass spectrometry, we discovered three cytoskeletal molecules, non-muscle myosin heavy chain IIA (MyH9), αII-spectrin, and βII-spectrin, associate with LFA-1 in T cells. Western blot analysis of mouse T cells revealed expression levels of MyH9 and αII-spectrin remained stable after T cell activation, but surprisingly, βII-spectrin expression was dramatically down-regulated during CD8+ T cell activation. Over-expression of βII-spectrin in activated CD8+ T cells induced chemokine-dependent migration on ICAM-1. Therefore, we hypothesize that βII-spectrin provides a mechanical link between LFA-1 and the cytoskeleton to regulate T cell migration without chemokine stimulation.

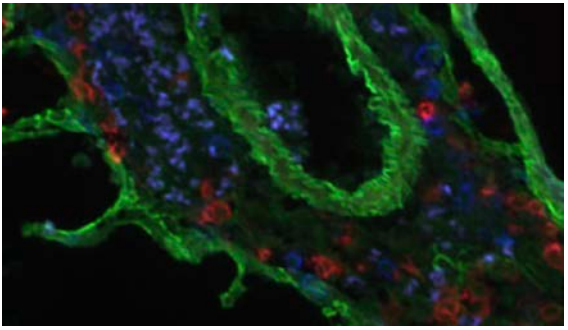
3:40 – 4:20 p.m.

DAVID TOPHAM, PhD

Professor, Dept. Microbiology and Immunology, University of Rochester

Establishment, Positioning & Maintenance of Tissue Resident Memory in the Influenza Infected Airway

RESEARCH INTERESTS



Influenza virus targets the epithelial lining of the respiratory tract. Cytotoxic CD8 T cells that leave the circulation and infiltrate the airway epithelium play a key role in controlling infection. However, little is known about mechanisms of T cell migration in the airway environment and engagement with infected epithelial cells. Our lab has developed a mouse model of influenza tracheitis and used live imaging tools to follow the tissue trafficking of infiltrating, virus-specific T cells. Ongoing studies aim to understand molecular interactions that regulate this process.

Optimal T cell mediated immunity to virus infection of peripheral tissues depends on the ability of the T cells to home to, function, and be retained in these sites. Because of the diversity in the structure and function of peripheral organs, a mechanism to regulate T cells in different organs must involve components shared by many tissues. A feature common to many tissues is the presence of extracellular matrix. Collagen IV is uniquely found in the basement membranes that form the foundation for all epithelial and endothelial surfaces. The receptor for collagen IV is the alpha-1 beta-1 integrin heterodimer, Very Late Antigen-1 (VLA-1). VLA-1 is expressed by subsets of activated and memory T cells of both the CD4 and CD8 lineages. It is important for the maintenance of memory T cells in peripheral non-lymphoid tissues, especially the lung and other mucosal sites. More recently, VLA-1 expression was associated with localization of T cells in proximity to epithelial surfaces and in promoting their survival during the acute phase of infections. Thus not only can it function to maintain memory T cells, it also is important for protection of effector T cells during the acute response.

POSTERS

Presenter(s) listed BOLD

1.	OPTOGENETIC REGULATION OF T CELL METABOLISM IN THE TUMOR MICROENVIRONMENT **Andrea Amitrano[1,2]**, Brandon Walling[2], Brandon Berry[3], Adam Trewin[3], Ph.D., Andrew Wojtovich[3], Ph.D., Minsoo Kim[2], Ph.D. *[1] Department of Cell Biology of Disease, University of Rochester, Rochester, N.Y. [2] Department of Microbiology and Immunology, David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester, N.Y. [3]Department of Pharmacology and Physiology, University of Rochester, Rochester, N.Y.*
2.	SYSTEMATIC STUDY OF INFLUENZA A AND B VIRUS CO-INFECTIONS **Pilar Blanco-Lobo**, Laura Rodriguez, Aitor Nogales, Luis Martinez-Sobrido *Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, N.Y.*
3.	**THE ROLE OF INTEGRIN $\alpha_1\beta_1$ (VLA-1) IN CD8$^+$ T CELL MOTILITY** **Patrick Buckley**, Emma Reilly, Ph.D., David Topham, Ph.D. *David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester, NY.*
4.	MEASUREMENT OF ENDOTHELIAL GLYCOCALYX BARRIER PROPERTIES IN SEPSIS **Luis F. Delgadillo[1]**, Julie M. Kuebel[1], Richard E. Waugh[1] *[1]Department of Biomedical Engineering, University of Rochester, Rochester N.Y.*
5.	TISSUE LOCALIZATION OF PULMONARY CD4 T CELLS FOLLOWING INFLUENZA INFECTION MODIFIES EXPRESSION OF MARKERS ASSOCIATED WITH TRAFFICKING BUT DOES NOT REMODEL CYTOKINE PROFILE **Anthony DiPiazza[1]**, Nathan Laniewski[1], Andrea J. Sant[1] *[1]Department of Microbiology and Immunology, DH Smith Center for Vaccine Biology and Immunology, University of Rochester Medical Center, Rochester, N.Y.*

6.	INHIBITION OF THE LATE STAGES OF AUTOPHAGY OVERCOMES HYPOXIA INDUCED CHEMORESISTANCE IN ACUTE MYELOID LEUKEMIA **Kaitlyn M. Dykstra**, Matthew Johnson, Jenna Whitmore, Houman Nourkeyhani, Megan Johnson, Dirkje Hanekamp and Eunice S. Wang *Department of Medicine, Roswell Park Cancer Institute, Buffalo, N.Y.*
7.	TARGETING FIBRONECTIN TO MODULATE CD4 EFFECTOR T CELL INTERSTITIAL MIGRATION AT INFLAMED SITES **Ninoshka R.J. Fernandes[1,3]**, Jane Sottile[2], Deborah J. Fowell[3] *[1]Department of Biomedical Engineering, University of Rochester, [2]Aab Cardiovascular Research Institute, and [3]David H Smith Center for Vaccine Biology & Immunology, University of Rochester Medical Center, Rochester, NY.*
8.	NK CELLS MAINTAIN INTESTINAL VASCULATURE INTEGRITY DURING HELMINTH INFECTION **Marilena Gentile[1]**, Yue Li[1], Amicha Robertson[1], Ghislaine Fontes[1], Nargis Khan[1,2], Eva Kaufmann[2], Maziar Divangahi[2] and Irah King[1] *[1]Department of Microbiology and Immunology, McGill University, Montreal, Quebec, Canada, [2]Department of Medicine, McGill University, Montreal, Quebec, Canada.*
9.	SPONTANEOUS SKIN INFLAMMATION AND DYSBIOSIS IN MICE DEFICIENT IN THE WISKOTT-ALDRICH SYNDROME PROTEIN **Katherine E. Herman[1]**, Takeshi Yoshida[2], Angela Hughson[1], Lisa Beck[2], Alex Grier[3], Steve Gill[3], and Deborah J Fowell[1] *[1]David H. Smith Center for Vaccine Biology and Immunology, Aab Institute of Biomedical Sciences, University of Rochester Medical Center; [2]Department of Dermatology, University of Rochester Medical Center and [3]Department of Microbiology and Immunology, University of Rochester Medical Center Rochester, N.Y.*
10.	FUNCTIONAL ROLES OF INNATE-LIKE T CELLS AND MACROPHAGES DURING *MYCOBACTERIUM MARINUM* INFECTION IN A TRANSGENIC *XENOPUS* TADPOLE MODEL **Kun Hyoe (Jules) Rhoo**, Eva-Stina Edholm and Jacques Robert *Department of Microbiology and Immunology, University of Rochester, Rochester, N.Y.*

11.	EVALUATION OF IMMUNE-MEDIATED MYOGENESIS DURING CHRONIC INFECTION WITH *T. GONDII* **Richard M. Jin**, Jordan Warunek and Elizabeth A. Wohlfert *Department of Microbiology and Immunology, Jacobs School of Medicine and Biomedical Sciences, University at Buffalo, Buffalo, N.Y.*
12.	TIME-LAPSE IMAGING OF RETINAL MICROGLIA *IN VIVO* SHOWS DYNAMIC PROCESS MOTILITY AT REST **Aby Joseph**, Jesse Schallek *Institute of Optics, University of Rochester, Flaum Eye Institute, University of Rochester, and Center for Visual Science, University of Rochester, Rochester N.Y.*
13.	PERTUSSIS TOXIN TREATMENT SUGGESTS CD8+ T CELLS NEED CHEMOKINE SIGNALS TO LOCATE ANTIGEN-BEARING CELLS DURING INFLUENZA INFECTION **Kris Lambert** and David J. Topham *David H. Smith Center for Vaccine Biology & Immunology, Department of Microbiology & Immunology, University of Rochester School of Medicine and Dentistry, Rochester, N.Y.*
14.	EPIDERMAL GROWTH FACTOR RELEASED FROM APOPTOTIC NEUTROPHILS **Kihong Lim**, David Topham and Minsoo Kim *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*
15.	QUANTITATIVE CELLULAR AND MOLECULAR IMAGING OF THE INTACT TUMOR MICROENVIRONMENT **Jan Martinek**[1], Hannah Brookes[1], Te-Chia Wu[1], Kyung In Kim[1], Joshy George[1], Paul Robson[1,2] Jacques Banchereau[1,2], and Karolina Palucka[1,2] *[1]The Jackson Laboratory for Genomic Medicine, Farmington, CT* *[2]Department of Genetics & Genome Sciences, University of Connecticut Health Center, Farmington, C.T.*

16.	BCL6 INHIBITS THE DEVELOPMENT OF IL-10 PRODUCING TH2 CELLS DURING INTESTINAL HELMINTH INFECTION **Alexandre P. Meli**, Ghislaine Fontés and Irah L. King *Department of Microbiology and Immunology, Microbiome and Disease Tolerance Centre, McGill University, Montreal, Quebec, Canada*
17.	IMAGING PHAGOSOMAL SURVIVAL OF COMMENSAL ORAL MICROBES Andrew Croft, **Sarah Metcalfe**, and Jason G. Kay *Department of Oral Biology, University at Buffalo, Buffalo, N.Y.*
18.	TNFα INHIBITS THE FORMATION OF GERMINAL CENTER-PHENOTYPE CD11C^{+} T-BET^{+}IGM^{+} MEMORY B-CELLS DURING INTRACELLULAR BACTERIAL INFECTION **Maria Popescu** and Gary Winslow *Department of Microbiology and Immunology, SUNY Upstate Medical University, Syracuse N.Y.*
19.	LOCAL CXCL10 EXPRESSION SHAPES MICRO-ANATOMICAL POSITIONING OF TH1 CELLS IN THE INFLAMED SKIN Hen Prizant[1], Milan Popovic[1], Christopher Barilla[2], Deborah J. Fowell[1] *[1]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, N.Y., [2]Bioinformatics, Rochester Institute of Technology, Rochester, N.Y.*
20.	QUANTIFYING LFA-1 DRIVEN SURFACE INTERACTION DYNAMICS IN SPREADING CD8^{+} T CELLS **Nick Reilly[1]**, Brandon L. Walling[2], Austin V. Skeeters[1], Minsoo Kim[2], and Patrick W. Oakes[1,3] *[1]Department of Physics and Astronomy, University of Rochester, [2]Department of Microbiology and Immunology, University of Rochester, [3]Department of Biology, University of Rochester, Rochester N.Y.*

21.	A NOVEL *IN VITRO* PLATFORM TO STUDY VASCULAR DISORDERS: ADVANCING THE KNOWLEDGE OF BLOOD-ENDOTHELIAL INTERACTIONS WITH OPTICAL AND ELECTRON MICROSCOPY **Alec Salminen**, Kilean Lucas, Tejas Khire, Richard Waugh, Ph.D., James McGrath, Ph.D. *Department of Biomedical Engineering, University of Rochester, Rochester, NY*
22.	α_V INTEGRIN IS CRITICAL FOR TFH POSITIONING AND GERMINAL CENTER OUTPUT **Dillon Schrock**, Scott Leddon, Deborah Fowell *Department of Immunology and Microbiology, University of Rochester, Rochester, N.Y.*
23.	NORADRENERGIC MODULATION OF MICROGLIAL DYNAMICS AND SYNAPTIC PLASTICITY **Rianne Stowell** and Ania Majewska *Department of Neuroscience, University of Rochester Medical School, Rochester, N.Y.*
24.	CHRONIC BRAIN DYSFUNCTION DRIVEN BY ACUTE SYSTEMIC INFLAMMATION **Alissa Trzeciak**[1], Yelena Lerman[1], Nguyen Mai[2], Marc Halterman[2], and Minsoo Kim[1] *[1]Department of Microbiology and Immunology, University of Rochester, [2]Center for Neurodevelopment and Disease, University of Rochester, Rochester, N.Y.*
25.	CHEMOKINE INDEPENDENT EFFECTOR $CD8^+$ T CELL MIGRATION **Brandon Walling**[1], Tara Capece[2], Nick Reilly[3], Patrick W. Oakes[3,4], Minsoo Kim[1] *[1]Department of Microbiology and Immunology, University of Rochester, Rochester N.Y., [2]National Institute of Allergy and Infectious Diseases (NIAID), Bethesda, M.D., [3]Department of Physics and Astronomy, University of Rochester, Rochester N.Y., [4]Department of Biology, University of Rochester, Rochester N.Y.*

26.	IMAGING CYTOKINE BIOLOGY IN THE TUMOR MICROENVIRONMENT: THE EXPRESSION AND FUNCTION OF IL-36G IN COLORECTAL CANCER **Aliyah M. Weinstein[1,3]**, Nicolas A Giraldo[1], Catherine Julie[2], Laetitia Lacroix[1], Florent Petitprez[1], Jean-François Emile[2], Wolf H Fridman1, Walter Storkus[3,4], Catherine Sautès-Fridman[1] *[1]UMR_S1138, Centre de Recherche des Cordeliers, 15 rue de l'Ecole de Médecine, 75006, Paris, [2]Laboratoire d'anatomie pathologique, Hopital Ambroise Paré, AP-HP, Boulogne, France, [3]University of Pittsburgh School of Medicine, Departments of Dermatology and Immunology, Pittsburgh, PA, USA, [4]University of Pittsburgh School of Medicine, Departments of Pathology and Bioengineering, and University of Pittsburgh Cancer Institute, Pittsburgh, PA,USA*
27.	THE ROLE OF CD109 IN REGULATING THE CUTANEOUS IL-23/IL-17 IMMUNE AXIS **Hualin Zhang**, Giustino Carnivale, Ghislaine Fontes, Irah King *Department of Microbiology and Immunology, McGill University, Montreal, Quebec, Canada.*

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