4th Annual
IMMUNE IMAGING SYMPOSIUM

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

Saturday, November 3rd, 2018

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.

4th Annual Immune Imaging Symposium
Saturday, November 3rd, 2018. 8:30 a.m. – 5:30 p.m.
Saunders Research Building and Helen Wood Hall Auditorium

8:30 - 8:50 a.m.
REGISTRATION, Poster set-up, Continental Breakfast

8:50 - 9:00 a.m.,
Deborah Fowell - *WELCOME AND INTRODUCTION*

9:00 - 9:40 a.m.
MICHAEL DUSTIN, Oxford University
Dynamics and Structure of Immunological Synapses

9:40 - 9:55 a.m.
SHORT TALK: **Kibaek Choe**, Cornell University
Intravital Microscopy of T and B cell Migration in Abluminal Side of High Endothelial Venules in Mice Lymph Node

9:55 - 10:35 a.m.
MATTHEW KRUMMEL, University of California, San Francisco
Imaging the Patterns of Immune Detection and Control

10:35 - 10:50 a.m.
SHORT TALK: **Dillon Schrock**, University of Rochester
T cell Interactions with the Extracellular Matrix Regulate Localization to the Germinal Center

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

11:20 - 12:00 p.m.
SUSAN SCHWAB, New York University
Exit Strategies: S1P Gradients and T cell Migration

12:00 – 12:15 p.m.
SHORT TALK: **Alissa Trzeciak**, University of Rochester
Chronic Brain Dysfunction Driven by Acute Systemic Inflammation

12:15 – 12:55 p.m.
PHILIPPE BOUSSO, Pasteur Institute
Dissecting Mechanisms of Tumor Immunotherapies at the Single Cell Level

1:00 – 2:30 pm
LUNCH
POSTER VIEWING & IMAGE CONTEST VOTING
Odd numbered posters 1:30-2:00
Even numbered posters 2:00-2:30
Last votes for Images 2:30

2:30 – 2:45 p.m.
SHORT TALK: **Judy Cannon**, University of New Mexico
Quantitative Analysis of T cell Motion in Influenza-Infected Lung

2:45 – 3:25 p.m.
MORGAN HUSE, Memorial Sloan Kettering Cancer Center
Mechanical Control of Cytotoxic T cell Function

3:25 – 3:40 p.m.
SHORT TALK: **Jonathan Pinney**, University of Rochester
Novel Method for Quantification of Phagocytosis Using Live Cell Imaging in Real-Time

3:40 – 4:20 p.m.
PATRICK OAKES, University of Rochester
Mechanics of CD4 T cell Migration

4:20 – 4:30 p.m.
POSTER AWARDS

IMAGE AWARD

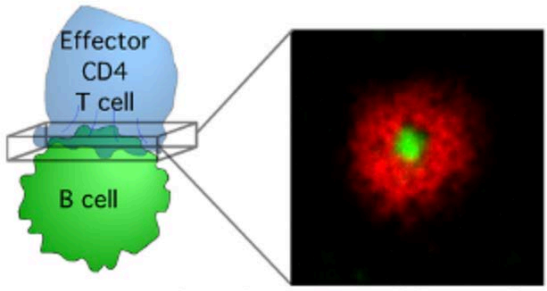
4:30 – 5:30 p.m.
WINE AND CHEESE RECEPTION

9:00 - 9:40 a.m.

MIKE DUSTIN, PhD
Director of Research, The Kennedy Institute of Rheumatology
Professor, Immunology
University of Oxford

Dynamics and Structure of Immunological Synapses

RESEARCH INTERESTS



Research in Dr Dustin's lab has been focused on the immunological synapse and integrating this into the 3D tissue context. His lab has modeled this interaction by replacing the antigen presenting cell with a supported planar bilayer. Bilayers presenting MHC-peptide complexes and ICAM-1 to trigger a minimal immunological synapse enabled the first visualization of supramolecular activation clusters. He has also applied this system to the investigation of regulatory T cell defects in rheumatoid arthritis. He continues to study how the immunological synapse contributes to tolerance and immunity using tools of systems biology and super-resolution imaging. His work includes the recent observation that small vesicles enriched in T cell receptor, synaptic ectosomes, are directly budded into the immunological synapse, handing off T cell receptor and other cargo to the antigen presenting cell. The advantage of this mode of communication is that it may operate on a time frame longer than the relatively short-lived immunological synapse. Thus, the synaptic ectosomes may deliver more durable instructions that last from days to weeks and continue to influence the responding cells long after the synaptic interaction has dissociated. A major focus will be the targeting of therapies to the immunological synapse to cure chronic inflammatory diseases like rheumatoid arthritis.

9:40 - 9:55 a.m.
SHORT TALK: **Kibaek Choe**, Postdoc, Cornell University

Intravital Microscopy of T and B cell Migration in Abluminal Side of High Endothelial Venules in Mice Lymph Node

Kibaek Choe[1,3], Soo Yun Lee[1], Jieun Moon[1], Eunjoo Song[1], Joo-Hye Song[4], Young-Min Hyun[5], Kenji Uchimura[6] and Pilhan Kim[1,2]
[1]Graduate School of Nanoscience and Technology, [2] Graduate School of Medical Science and Engineering, Korea Advanced Institute of Science and Technology (KAIST), Daejeon, Republic of Korea [3] School of Applied and Engineering Physics, Cornell University, Ithaca, New York, USA [4] Center for Vascular Research Institute for Basic Science Daejeon Republic of Korea [5] Department of Anatomy and Brain Korea 21 PLUS Project for Medical Science, Yonsei University College of Medicine, Seoul 03722, Republic of Korea [6] Unité de Glycobiologie Structurale et Fonctionnelle, UMR 8576 CNRS, Université de Lille 59655 Villeneuve d'Ascq, France

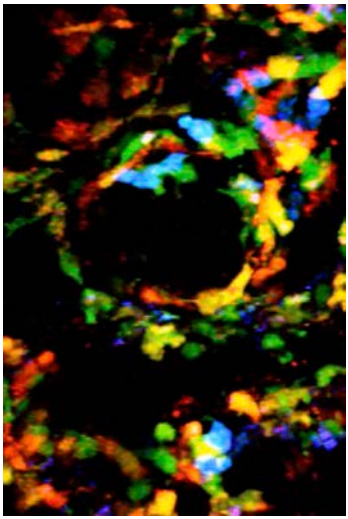
High endothelial venules (HEVs) are specialized blood vessels in lymph node (LN) for lymphocyte recruitment from the blood. HEVs have distinctive cuboidal-shaped endothelial cells and prominent perivascular sheaths consisting of fibroblastic reticular cells (FRCs). There have been many studies to visualize the migration of lymphocyte along the HEV-endothelium in luminal side. However, abluminal side migration and trans-FRC migration have not been well studied. In this work, we utilized a custom-design laser scanning confocal microscope to visualize lymphocyte (T and B cells) migration in abluminal side of HEV of mice LN in vivo. GFP expressing T or B cells were adoptively transferred into a recipient mouse. In addition, HEV-endothelial cells and FRCs were labeled in vivo for simultaneous visualization. We successfully observed that T and B cells squeezed in between endothelial cells (ECs) and then migrated through the perivascular channel (PVC), the narrow space between ECs and FRCs, to an exit for trans-FRC migration to parenchyma of LN. Next, we investigated the role of L-selectin in the abluminal migration of T and B cells by using GlcNAc6ST-1 KO mice which have low expression of L-selectin ligands in the abluminal side of HEV-endothelial cells. Interestingly, in GlcNAc6ST-1 KO mice, T and B cell spent more time for trans-FRC migration in comparison with the control wildtype mice. In addition, we observed that there existed preferred exit sites (“hot spots”) from the PVC to parenchyma of LN for both of T and B cells. To investigate the potential role of perivascular resident dendritic cells in the hot spots for the T cells, we simultaneously visualized T cells and CD11c+ dendritic cells. We found that multiple T cells exited HEV through the same site where dendritic cells closely localized to FRCs.

9:55 - 10:35 a.m.

MAX KRUMMEL, PhD
Professor, Pathology
University of California, San Francisco

Imaging the Patterns of Immune Detection and Control

RESEARCH INTERESTS



Dr Krummel's work focusses on understanding patterns of immune cell-cell interactions and how these generate "the immune system". His studies of the immune synapse have shown how T cells regulate their motility, how they signal through synapses while moving, how they communicate with each other during arrest, and how they 'search' a new tissue. These are all fundamental findings and provide a lens through which to understand T cell function.

His lab has developed novel methods and computational platforms to understand immunological processes in space and in time within normal and diseased organs. They pioneered the use of intravital imaging to identify events in progressive tumors in which incoming tumor-specific T cells are captured by a population of myeloid cells. This imaging platform has 'guided' the development of next-generation protein immuno-therapeutics for tumor control. In addition, the lab has developed novel imaging technologies that allow observation of the immune system in the homeostatic, infected/injured, allergic or metastatic lung.

10:35 - 10:50 a.m.

SHORT TALK: **Dillon Schrock**, Postdoc, University of Rochester

T cell Interactions with the Extracellular Matrix Regulate Localization to the Germinal Center

Dillon Schrock[1], Scott Leddon[1], Angie Hughson[1], Jim Miller[1], Adam Lacy-Hulbert[2], and Deborah Fowell[1]

[1]Department of Microbiology and Immunology, University of Rochester, Rochester NY; Benaroya Institute, Seattle, WA.

For a successful response to infection or vaccination, the immune system requires precise regulation and coordination to allow efficient elimination of pathogens and generation of memory, while limiting inflammatory damage and avoiding autoimmunity. The immune response is regulated in multiple dimensions and on various levels. Chemokine signals and intercellular interactions are well-documented mediators of immune cell positioning, but more recently the extracellular matrix has become the subject of research into its role in governing lymphocyte localization.

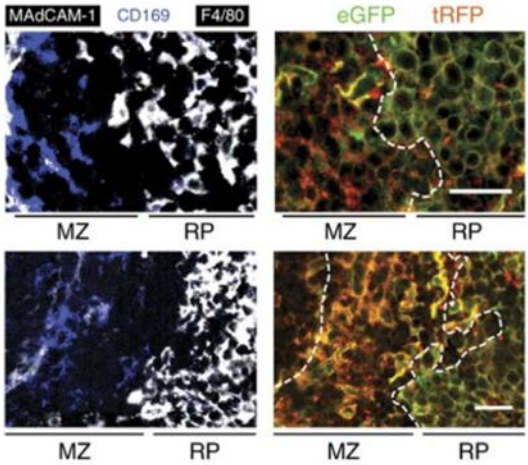
Previously, our lab has demonstrated the importance of the ECM-binding aV (alpha-V) integrins in CD4+ T cell motility in the inflamed dermis. We have also investigated the role of aV integrins in positioning CD4+ T cells within the lymph node. Our data suggest that CD4+ T cell expression of aV integrins is necessary for efficient localization to the germinal center (GC), a specialized region of the lymph node B cell follicle where affinity maturation and long-lived plasma cell differentiation take place. The GC light zone contains a specialized stromal cell subset known as follicular dendritic cells (FDC). During inflammation, GC FDCs become coated in a de novo ECM network composed of multiple ligands that contain the RGD binding sequence for aV integrins, including vitronectin, osteopontin, and MFG-E8. CD4+ T cells lacking aV integrins localize to the GC with reduced efficiency and the resulting GCs produce lower levels of affinity maturation and fail to generate long-lived plasma cells that seed the bone marrow. Strikingly, treatment with the clinical aVb3 (alpha-V beta-3) inhibitor cilengitide also significantly altered T cell localization within the GC and B cell follicle. Taken together, our data highlight the importance of appropriate localization of T cells within the B cell follicle for an efficient GC response.

We propose CD4+ Tfh cells are retained in the GC light zone via interactions between aV integrins and the RGD-rich FDC ECM network. This paradigm represents a novel mechanism regulating CD4+ T cell positioning within the lymph node. These data also suggest that therapeutic intervention at this axis may be capable of mitigating diseases associated with dysregulated GCs or of promoting the GC response to enhance vaccination.

11:20 - 12:00 p.m.
SUSAN SCHWAB, **PhD**
Associate Professor, Pathology
New York University

Exit Strategies: S1P Gradients and T cell Migration

RESEARCH INTERESTS



The Schwab lab studies lymphocyte migration, with an emphasis on three questions: (1) What determines how long a lymphocyte stays in a given location—surveying for antigens or fighting infection—before it moves on? (2) How are the gradients that direct immune cell migration established? (3) How do the trafficking requirements of normal and leukemic T cells differ, and can these differences be targeted therapeutically?

Much of their focus has been on how the residence time of T cells in lymphoid organs is determined. The lab have established that a gradient of the signaling lipid sphingosine 1-phosphate (S1P) is required to guide T cells out of lymphoid organs. They have also identified many of the key cells and enzymes that control this gradient, and have developed novel tools to map it. With this research, Dr Schwab hopes to provide fundamental insight into how lipid gradients are shaped. She also hopes that this work will translate to improved therapies for inflammatory disease. Drugs targeting S1P signaling are used clinically as immune suppressants. These drugs block the exit of activated T cells from lymphoid organs, preventing them from reaching organs that are subject to autoimmune attack. They may also have other anti-inflammatory effects. However, because S1P also regulates vascular stability and heart rate, side effects are a serious concern. By determining how S1P gradients are regulated, targets may be identified that enable spatially specific modulation of S1P signaling.

12:00 – 12:15 p.m.

SHORT TALK: **Alissa Trzeciak**, Graduate Student, University of Rochester

Chronic Brain Dysfunction Driven by Acute Systemic Inflammation

Alissa Trzeciak[1], Yelena Lerman[1], Tae-Hyoun Kim[1], Nguyen Mai[2], Marc Halterman[2], and Minsoo Kim[1]

[1]Department of Microbiology and Immunology, University of Rochester, Rochester, NY.
[2]Center for Neurodevelopment and Disease, University of Rochester, Rochester, NY.

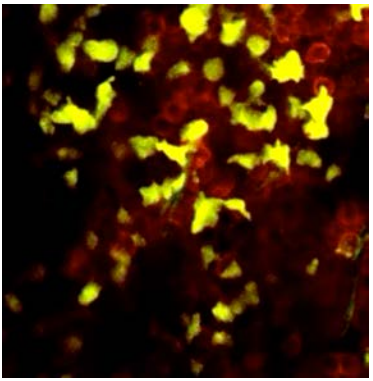
Sepsis is a condition defined by systemic inflammation due to infection, and accounts for 25-30% of deaths in intensive care units (ICUs). Even after full recovery, up to 70% of patients suffer neurological consequences because of sepsis-associated encephalopathy (SAE), which has been one of the most significant challenges for the long-term quality of life in sepsis survivors. To examine the mechanisms that mediate long-term brain damage induced by SAE, we are exploring an LPS-induced endotoxemia and cecal ligation and puncture sepsis models to establish a disease phenotype where septic mice with varying degrees of severity recover and develop chronic brain dysfunction. By observing cellular infiltrates during the early stages of disease, we detected a significant neutrophil recruitment to the lungs while monocytes preferentially homed to the brain. Utilizing the $CX_3CR1^{CreER\text{-}EYFP}$: $Rosa26D^{sRed}$ reporter mouse to discriminate microglia from infiltrating monocytes, we were able to distinguish cellular infiltrates from their resident counterparts. Strikingly, when surviving mice were fully recovered at day 50 post infection, the brain parenchyma was marked by a permanent increase in resident microglia, whose morphology resembled an activated amoeboid phenotype. However, using CCR2-knockout animals to inhibit circulating monocytes from bone marrow egress, the increase in activated microglia after 50 days-post infection was ablated. In contrast, numbers of tissue resident immune cells in other organs including the lungs, bone marrow, and circulation in the survived mice were comparable to sham mice. In addition, concentrations of pro-inflammatory cytokines such as IL-1β (beta), TNF–α (alpha), and IL-6 in both the serum and cerebral spinal fluid of the sepsis survivor mice were identical to those of naïve mice at day 50. To date, no direct mechanism has been elucidated to uncover monocyte-microglia interactions that may influence long-term changes in the central nervous system. Our data suggest that sepsis is associated with acute systemic inflammation and massive immune cell invasion, which leads to long-term changes in brain macrophage numbers and activation after recovery. We predict that CCR2-dependent monocytes drive microglia proliferation by increasing macrophage growth factors in the brain parenchyma during acute phases of sepsis. The consequences and kinetics of these innate immune cell changes is crucial for determining their effects on long-term cognitive dysfunction.

12:15 – 12:55 p.m.

PHILIPPE BOUSSO, PhD
Head, Dynamics of Immune Response Unit
Pasteur Institute, France

Dissecting Mechanisms of Tumor Immunotherapies at the Single Cell Level

RESEARCH INTERESTS



The immune system comprises a collection of specialized cells and organs that fight infections and can survey cancer cells. Immune responses to pathogens and tumors are highly coordinated events that take place in complex and specialized tissue microenvironments. An integrated view of innate and adaptive immune responses to infections and cancer requires a better understanding of how immune cells communicate and fulfill their task *in vivo*. In addition, it is essential to unravel how pathogens and tumor microenvironment subvert their functions. Recent progresses in intravital two-photon imaging offer a novel perspective to address some of these critical issues in physiological settings. By further developing functional *in vivo* imaging, the Bousso Lab aims at identifying critical aspects of T cell and NK cell activation and function during tumor growth, during infection by an intracellular parasite and during transplantation. Elucidating the fundamental mechanisms underlying the regulation of innate and adaptive immune responses *in vivo* will delineate basic principles to develop or improve immunotherapeutical strategies to treat cancer and infectious diseases.

2:30 – 2:45 p.m.

SHORT TALK: **Judy Cannon**, Associate Professor, University of New Mexico

Quantitative Analysis of T cell Motion in Influenza-Infected Lung

Judy L. Cannon[1,2,3], Melanie E. Moses[4,5], Janie R. Byrum[1], Paulus Mrass[1], G. Matthew Fricke[4], Humayra Tasnim[4]

[1]Department of Molecular Genetics and Microbiology, University of New Mexico School of Medicine, Albuquerque, NM. [2]Department of Pathology, University of New Mexico School of Medicine, Albuquerque, NM. [3]Autophagy, Inflammation, and Metabolism Center of Biomedical Research Excellence, University of New Mexico School of Medicine, Albuquerque, NM. [4]Computer Science Department, University of New Mexico, Albuquerque, NM. [5]Santa Fe Institute-External Faculty, Santa Fe, NM.

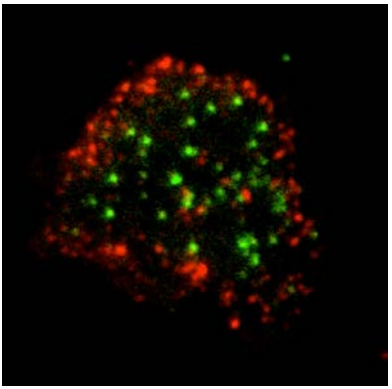
T cells are a key effector cell type in the immune response, migrating through tissues in order to clear infection including influenza infection in the lung. T cells must move through multiple tissues to mount an effective response: naïve T cells migrate in and out of lymph nodes searching for antigen on dendritic cells, while activated T cells migrate to peripheral tissue such as influenza infected lung to clear infection. We investigate how the lung environment affects T cell motion using two photon microscopy to visualize effector T cells moving in inflamed lung in two inflammatory settings: a model of acute lung injury and influenza infection. We perform quantitative analysis of in situ T cell movement to show that effector T cells in LPS treated lung move with intermittent motion, with periods of directional motion as well as periods of stopped motion. Computational modeling suggests that intermittent motion may be important to help T cells make longer lasting contacts with target cells as well as search tissue efficiently. Using these quantitative analyses, we find that effector T cells in the lung during influenza infection also move with intermittent motion. Intermittent T cell motion in lung requires signaling through ROCK, but surprisingly, inhibition of G-alpha-i signaling downstream of G-protein coupled receptors with pertussis toxin does not affect intermittent motion. Interestingly, we find that specific inhibition of CXCR3 and CXCR4 actually increased the overall speed of T cell motion in ALI inflamed lung, suggesting that specific chemokines may be important for T cell interactions with target cells. These results demonstrate that the type of T cell movement affects T cell interactions with target cells during immune responses in lung, leading to effective T cell clearance of infection.

2:45 – 3:25 p.m.

MORGAN HUSE, PhD
Professor, Immunology
Memorial Sloan Kettering Cancer Center

Mechanical Control of Cytotoxic T cell Function

RESEARCH INTERESTS



Effective immune responses against infectious agents and cancer require that immune cells traffic to the correct locations and then identify and specifically respond to threats by physically interacting with other cells. Leukocytes are particularly well suited for these tasks because they can completely reorganize their structure in a matter of minutes in response to surface receptor stimulation. This remarkable plasticity enables them to function both rapidly and effectively in a wide variety of physiological contexts. The Huse lab studies the signaling mechanisms that control leukocyte architecture and also how specific cellular structures contribute to immune function. A better understanding of these issues could aid in the development of strategies to better modulate immune responses in vivo. Interests include: (1) Killer lymphocytes destruction of infected or transformed target cells, focused on how the cytolytic synapse is assembled and how specific aspects of synaptic architecture promote target cell killing; (2) Strategies for modulating CTL-dependent immune responses during cellular immunotherapy by engineering lymphocytes to target tumors with enhanced potency and selectivity; (3) Cell-to-cell interactions contribution to intercellular communication to understand how the unique physical environment imposed by the synapse affects the communicative chemical interactions that occur within it. The group has developed novel photochemical reagents for micron-scale activation by ultraviolet light and continues to apply bioengineering and materials science to create synthetic microenvironments for the observation of localized secretory and biophysical responses.

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

Novel Method for Quantification of Phagocytosis Using Live Cell Imaging in Real-Time

Jonathan Pinney, Charles Chu, Clive Zent, Joshua Zent, Karl VanDerMeid and Michael R. Elliott
Department of Microbiology and Immunology, University of Rochester, Rochester NY

Antibody-dependent cellular phagocytosis (ADCP) by macrophages is an important mechanism of cellular cytotoxicity induced by many therapeutic monoclonal antibodies (mAbs) and an important pathogenic mechanism in numerous diseases, including immune thrombocytopenia and autoimmune hemolytic anemia. While much is known about the receptors and signaling that regulates the engulfment of mAb-opsonized targets, ADCP involves very complex intercellular spatiotemporal events that are poorly understood. To address this need, we have developed a novel method to measure ADCP in real time using fluorescent microscopy to quantify phagocytosis by primary mouse and human macrophages. The method involves capturing images of phagocytic cells every 2 minutes for up to 24 hours over a range of experimental conditions. Each image is then analyzed separately and the data is combined to quantify the timing and relative amount of phagocytosis that occurs over the course of a given experiment. Phagocytic quantification is made possible by fluorescently staining phagocytic cells and counting the voids, circular spots that arise within the dye labeled phagocytes due to fluorescent dye exclusion, which form as target cells are internalized. The number of voids and phagocytes within each image for every microscopic field are then counted using various NIS-Elements functions. Once the number of voids and phagocytic cells are known for each image we are able to calculate a phagocytic index that we developed, which allows the amount of phagocytosis to be comparable across experiments. Our method has proven to be highly sensitive and reproducible, and has been validated through comparisons with other established methods for phagocytic quantification. Using this method, we have discovered that ADCP-mediated cell clearance by macrophages is carried out in multiple phases. The first phase, called “engorgement,” lasts for approximately 30 minutes and is characterized by the rapid engulfment of up to 30 targets per macrophage. The second phase, termed “acute attenuation,” follows immediately and is characterized by a sharp reduction in the uptake of mAb-opsonized targets. The third phase, “persistent attenuation,” also features reduced levels of engulfment and lasts for many hours. We believe our strategy is an improvement upon some of the previously used methods due to its ability to quantify phagocytickinetics in real time over a long period at a very high sensitivity.

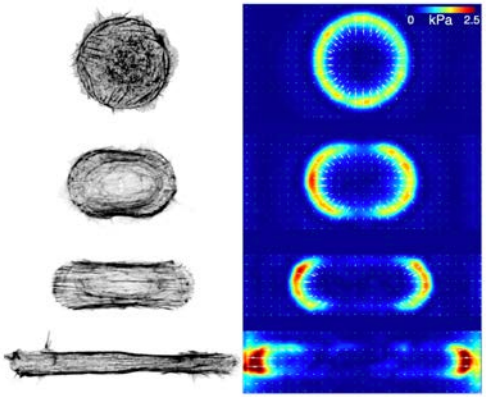
3:40 – 4:20 p.m.

PATRICK OAKES, PhD

Assistant Professor, Dept. Physics & Astronomy, University of Rochester

Mechanics of CD4 T cell Migration

RESEARCH INTERESTS



The Oakes lab is interested in the balance between biochemical and mechanical signaling that regulates cell shape, motility and function. A wide variety of fundamental physiological processes (division, migration, development, etc…) rely on the cell being able to either maintain its shape, or alter it in a tightly regulated manner. There is a lack an understanding of how the cell coordinates these interactions at the cellular and tissue length scales. Put more simply: how does the cell know where and how hard to pull? And how does the cell know to resist deformation or undergo a shape change? To tackle these questions the Oakes group use a combination of techniques, including Traction Force Microscopy (TFM), micropatterning, computational modeling, and genetic, optogenetic and pharmacological perturbations.

Non-biochemical signals from the surrounding external environment, such as ligand density and distribution, fluid shear stress and environmental stiffness, play an equally important role in regulating cell shape and cytoskeletal architecture. In particular, the stiffness of the Extracellular Matrix (ECM) has been shown to directly influence signaling, differentiation, metastasis and even cell spreading. Despite this behavior appearing to affect a wide variety of cell types and processes, there is a current lack of understanding of how cells translate physical signals into biochemical ones. The lab is focused on trying to understand how adhesions and the cytoskeleton might play a role in cells interpreting mechanical signals from the extracellular environment. The use of polyacrylamide substrates, whose Young's moduli can be controlled easily, enables the Oakes lab to explore the role of stiffness in processes like cell spreading and migration of immune cells.

POSTERS

Presenter(s) listed BOLD

1.	*Regulation of CD8+ T cell Metabolism in the Tumor Microenvironment* **Andrea M. Amitrano**[1,2], Brandon J. Berry[3], Andrew P. Wojtovich[3,4], Minsoo Kim[1,2] [1] Department of Pathology, University of Rochester, Rochester, NY; [2] Center for Vaccine Biology and Immunology, Dept. of Microbiology and Immunology, University of Rochester, Rochester, NY; [3]Department of Pharmacology and Physiology, University of Rochester, Rochester, NY; [4] Department of Anesthesiology and Perioperative Medicine, University of Rochester, Rochester, NY
2.	*Response to Fractionated and Ablative Radiotherapy Does Not Depend on RIPK1 Mediated Necroptosis* **Nicholas G. Battaglia**, Scott A. Gerber, and Edith M. Lord Department of Microbiology and Immunology, University of Rochester, Rochester, NY
3.	*Increased Activation of Canonical NF-κ(Kappa)B Signaling in Tendon Healing Results in Increased Macrophage Presence and Accelerated Extracellular Matrix Deposition* **Katherine T. Best**[1,2], Alayna E. Loiselle[2] [1]Department of Pathology and Laboratory Medicine, University of Rochester, Rochester NY; [2]Center for Musculoskeletal Research, University of Rochester, Rochester NY
4.	*Targeting the Tumor Draining Lymph Node to Enhance Anti-tumor Responses in Pancreatic Ductal Adenocarcinoma* **Booyeon J.** Han, Bradley N. Mills, Brian A. Belt, David C. Linehan, Scott A. Gerber Surgery Research, University of Rochester, Rochester, NY
5.	*Quantitative Analysis of T cell Motion in Influenza-Infected Lung* **Judy L. Cannon**[1,2,3], Melanie E. Moses[4,5], Janie R. Byrum1, Paulus Mrass1, G. Matthew Fricke[4], Humayra Tasnim[4] [1] Department of Molecular Genetics and Microbiology, University of New Mexico School of Medicine, Albuquerque, NM; [2] Department of Pathology, University of New Mexico School of Medicine, Albuquerque, NM; [3]Autophagy, Inflammation, and Metabolism Center of Biomedical Research Excellence, University of New Mexico, Albuquerque, NM; [4]Computer Science Department, University of New Mexico, Albuquerque, NM; [5]Santa Fe Institute-External Faculty, Santa Fe, NM.

6.	*Intravital Microscopy of T and B cell Migration in Abluminal Side of High Endothelial Venules in Mice Lymph Node* **Kibaek Choe**[1,3], Soo Yun Lee[1], Jieun Moon[1], Eunjoo Song[1], Joo-Hye Song[4], Young-Min Hyun[5], Kenji Uchimura[6] and Pilhan Kim[1,2] [1] Graduate School of Nanoscience and Technology; [2] Graduate School of Medical Science and Engineering, Korea Advanced Institute of Science and Technology (KAIST), Daejeon, Republic of Korea; [3]School of Applied and Engineering Physics, Cornell University, Ithaca, New York; [4] Center for Vascular Research Institute for Basic Science Daejeon Republic of Korea; [5] Department of Anatomy and Brain Korea 21 PLUS Project for Medical Science, Yonsei University College of Medicine, Seoul 03722, Republic of Korea; [6] Unité de Glycobiologie Structurale et Fonctionnelle, UMR 8576 CNRS, Université de Lille 59655 Villeneuve d'Ascq, France
7.	*Th1 cell Motility and Function Modulated by Fibronectin Blockade at Sites of Inflammation* **Ninoshka R.J. Fernandes**[1,4], Dillon Schrock[4], Chris Barilla[4], Denise Hocking[1,3], Jane Sottile[2], Deborah J. Fowell[4] [1]Department of Biomedical Engineering, University of Rochester, Rochester, NY; [2]AabCardiovascular Research Institute, University of Rochester, Rochester, NY; [3]Department of Pharmacology & Physiology, University of Rochester, Rochester, NY; [4]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY.
8.	*Spontaneous Skin Inflammation and Dysbiosis in Mice Deficient in 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]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [2]Department of Dermatology, University of Rochester, Rochester, NY; [3]Department of Microbiology and Immunology, University of Rochester, Rochester, NY.
9.	*Long-live Plasma Cell Responses are Optimized by Integrin αv Expression on CD4 cells.* **Scott A. Leddon[1]**, Dillon C. Schrock[1], Angela Hughson[1], Jim Miller[1], Adam Lacy-Hulbert[2], and Deborah J Fowell[1]. [1]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [2]Benaroya Research Institute at Virginia Mason, Seattle, WA.

10.	*T-bet-dependent ILC1-derived IFN-γ is Required for Sustaining DCs During T.Gondii Infection* **Américo H. López-Yglesias**, Ellie Camanzo, Elise Burger, Alessandra Araujo, Andrew T. Martin, Felix Yarovinsky Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY.
11.	*Propagation of S. aureus through Canalicular Sized Nanopores is Independent of the Accessory Gene Regulator (Agr)* **Elysia Masters[2]**, Alec Salminen[2], Karen de Mesy Bentley[4], James McGrath[2], Steven Gill[3], Hani Awad[2] and Edward Schwarz[1] [1]Center for Musculoskeletal Research, University of Rochester, Rochester, NY; [2]Department of Biomedical Engineering, University of Rochester, Rochester, NY; [3]Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [4]Department of Pathology and Laboratory Medicine, University of Rochester, Rochester, NY; Department of Orthopaedics, University of Rochester, Rochester, NY
12.	*Lung Megakaryocytes are Professional Antigen Presenting Cells* **Daphne N. Pariser[1]**, Zachary T. Hilt[2], Sara K. Ture[3], and Craig N. Morrell[3] [1]Department of Microbiology and Immunology, University of Rochester , [2]Translational Biomedical Sciences, University of Rochester, [3]Aab Cardiovascular Research Institute, University of Rochester, Rochester, NY
13.	*Novel Method for Quantification of Phagocytosis Using Live Cell Imaging in Real-Time* **Jonathan Pinney[1]**, Charles Chu[2], Clive Zent[2], Joshua Zent, Karl VanDerMeid[3] and Michael R. Elliott[1] [1]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [2]Hematology/Oncology, University of Rochester, Rochester, NY; [3]Cancer Center, University of Rochester, Rochester, NY

14.	*Focal Perivascular Regions of CXCL10 Production Define Hubs for Antigen Presentation to CD4+ Th1 cells* **Hen Prizant**, Deborah Fowell Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY.
15.	*Comparison of CD4 T cell Responses Elicited by Influenza A and B Viruses: Effect of Microenvironment on the Fate of CD4 T cell Effector Function(s).* **Rattan A**[1], Elena A. Govorkova[2], Richard J. Webby[2] and Andrea J. Sant[1] [1]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [2]Department of Infectious Diseases, St. Jude Children's Research Hospital, Memphis, TN.
16.	*CD49a and CD103 Define Distinct Populations of T_{RM} cells that Possess Different Capacities for Motility and Functionality* **Emma C Reilly[1]**, Kris Lambert Emo[1], Francisco A Chaves[1], Nicholas S Reilly[3], Patrick W Oakes[2,3], and David J Topham[1] [1]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY ;[2]Department of Biology, University of Rochester, Rochester, NY; [3]Department of Physics and Astronomy, University of Rochester, Rochester, NY
17.	*$\alpha V\beta 3$* (alphaVbeta3) Integrin Expression Regulates CD4+ T cell Migration Phenotypes **Nicholas S. Reilly[1]**, Ninoshka R. J. Fernandes [3,4], Malavika Satheesh[4], Jim Miller[4], Deborah J. Fowell[4], Patrick W. Oakes[1,2] [1]Department of Physics and Astronomy, University of Rochester, Rochester, NY; [2]Department of Biology, University of Rochester, Rochester, NY; [3]Department of Biomedical Engineering, University of Rochester, Rochester, NY; [4] Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY.
18.	*Dual-Scale Porous Ultrathin Membranes for Vasculature Barrier Modeling* **Alec Salminen[1]**, Jingkai Zhang[2], Greg Madejski[1], Tejas Khire[1], Richard Waugh[1], James McGrath[1], Thomas Gaborski[3] [1]Department of Biomedical Engineering, University of Rochester, Rochester NY; [2]Institue of Optics, University of Rochester, Rochester NY; [3]Department of Biomedical Engineering, Rochester Institute of Technology, Rochester NY

19.	*Imaging Immune Dynamics in a Mouse Model of Perioperative Neurocognitive Disorders* **Priyanka Saminathan[1],** Patrick Miller-Rhodes[2], Herman Li[2] and Harris A. Gelbard[1,2] [1]Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [2]Department of Neuroscience, Center for Neurotherapeutics Discover, University of Rochester, Rochester, NY
20.	*T cell Interactions with the Extracellular Matrix Regulate Localization to the Germinal Center* **Dillon Schrock[1],** Scott Leddon[1], Angie Hughson[1], Jim Miller[1], Adam Lacy-Hulbert[2], and Deborah Fowell[1] [1]Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [2]Benaroya Research Institute at Virginia Mason, Seattle, WA.
21.	*Pretreatment Peripheral Blood Monocyte Subset Signature is Predictive of Patient Response to Dendritic Cell Vaccination* **Anand P. Sharda[1]**, Nicholas C. Hoffend[1,2],, Alexander A. Wald[2],, Katja Koeppen[3], Jan L. Fisher[3], Lionel D. Lewis[4], Thomas H. Hampton[3], Camilo E. Fadul[5], Marc S. Ernstoff[6], Thomas Schwaab[1,2], and Jason B. Muhitch[1,2] [1]Department of Immunology, Roswell Park Comprehensive Cancer Center, Buffalo, NY; [2]Department of Urology, Roswell Park Comprehensive Cancer Center, Buffalo, NY; [3]Department of Microbiology and Immunology, The Geisel School of Medicine at Dartmouth, Lebanon, NH; [4]Section of Clinical Pharmacology & Hematology Oncology, Department of Medicine, The Geisel School of Medicine at Dartmouth, Lebanon, NH; [5]Department of Neurology, University of Virginia, Charlottesville, VA; [6]Department of Medicine, Roswell Park Comprehensive Cancer Center, Buffalo, NY
22.	*Chronic Brain Dysfunction Driven by Acute Systemic Inflammation* **Alissa Trzeciak[1]**, Yelena Lerman[1], Tae-Hyoun Kim[1], Nguyen Mai[2], Marc Halterman[2], and Minsoo Kim[1] [1] Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY; [2]Center for Neurodevelopment and Disease, University of Rochester, Rochester, NY.

23.	*Cross-Talk between the Immune and Nervous Systems: Effect of the Beta-Blocker, Propranolol, on the Immune Response Generated after Stereotactic Body Radiation Therapy (SBRT) in an Orthotopic Pancreatic Cancer Model* **Taylor P. Uccello**[1], Bradley N. Mills[1], Elizabeth A. Repasky[2], David C. Linehan[1], Scott A. Gerber[1]. [1]University of Rochester, Rochester NY; [2]University of Buffalo Roswell Park Comprehensive Cancer Center, Buffalo NY.

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