

**5th Annual
IMMUNE IMAGING SYMPOSIUM**



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

Saturday, November 2nd, 2019

**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 cytotoxicity, 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.

5th Annual Immune Imaging Symposium

Saturday, November 2nd, 2019. 8:30 a.m. – 5:30 p.m.

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.

KLAUS LEY, La Jolla Institute

SuperSTORM to Reveal Molecular Patterns of Activated Integrins on Cells

9:40 - 10:20 a.m.

XIAOLEI SU, Yale

Mechanism of Chimeric Antigen Receptor (CAR) Signaling

10:20 - 10:35 a.m.

SHORT TALK: Andrea Amitrano, University of Rochester

Regulation of CD8+ T cell Metabolism in the Tumor Microenvironment

10:35 - 11:10 a.m. Coffee Break

11:10 - 11:50 p.m.

ANNA HUTTENLOCHER, University of Wisconsin

Imaging Inflammation and its Resolution

11:50 - 12:05 p.m.

SHORT TALK: Oliver Bracko, Cornell

Selective Inhibition of NOX2 Decreases Non-flowing Brain Capillaries, Increases Flow & Improves Cognition in Alzheimer's Disease Models

12:05 - 12:55 p.m.

DANIEL HAMMER, University of Pennsylvania

Force Spectroscopy and Upstream Migration of Amoeboid Cells

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 pm

2:30 – 2:45 p.m.

SHORT TALK: Alessandra Araujo, University of Rochester
IFN- γ Directly Mediates Degranulation and Death of Metabolically Altered Paneth Cells During Toxoplasma gondii Infection

2:45 – 3:25 p.m.

MICHAEL GERNER, University of Washington
Organization of Innate-Adaptive Cell Crosstalk in Lymphoid Tissues

3:25 – 3:40 p.m.

SHORT TALK: Emma Reilly, University of Rochester
Tracheal Submucosal Glands Offer a Novel Habitat for Resident Memory CD8+ T cells

3:40 – 4:20 p.m.

MAIKEN NEDERGAARD, University of Rochester
Glymphatic/Lymphatic Connections

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.

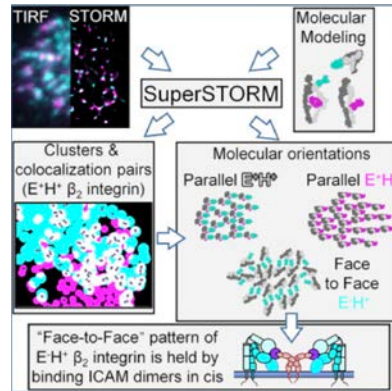
KLAUS LEY, M.D.

Professor and Head of the Division of Inflammation Biology
La Jolla Institute for Immunology, San Diego

SuperSTORM to reveal molecular patterns of activated integrins

RESEARCH INTERESTS

Dr. Ley and his team study inflammation as a defense reaction caused by tissue damage or injury, characterized by redness, heat, swelling, and pain. The primary objective of inflammation is to localize and eradicate the irritant and repair the surrounding tissue. For the survival of the host, inflammation is a necessary and beneficial process. The inflammatory response involves three major stages: first, dilation of capillaries to increase blood flow; second, microvascular structural changes and escape of plasma proteins from the bloodstream; and third, leukocyte transmigration through endothelium and accumulation at the site of injury.



The leukocyte adhesion cascade is a sequence of adhesion and activation events that ends with extravasation of the leukocyte, whereby the cell exerts its effects on the inflamed site. Dr. Ley has investigated the roles of adhesion molecules in acute and chronic inflammation with the ultimate goal to develop methods to control inflammation. One application is in atherosclerosis, the disease of the vessel wall that underlies heart attacks and strokes. Dr. Ley's team is working on developing a vaccine against atherosclerosis. They have developed a vaccine that is effective in mice and are now aiming at translating this to a vaccine for humans.

9:40 - 10:20 a.m.

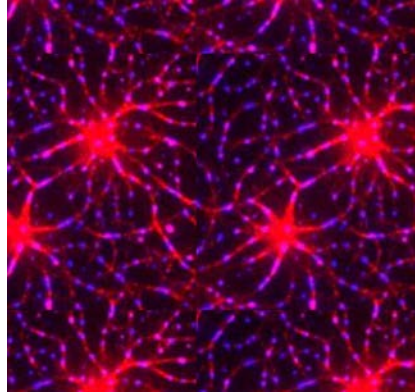
XIAOLEI SU, Ph.D.

Assistant Professor of Cell Biology
Yale School of Medicine

Mechanism of Chimeric Antigen Receptor (CAR) Signaling

RESEARCH INTERESTS

The Su Lab studies membrane remodeling and membrane-proximal signal transduction during immune responses. Using combined approaches of biochemical reconstitution, single molecule imaging, and cell engineering, the Su lab aims to understand how spatial and temporal organization of membrane proteins and lipids regulates immune cell activation. Their work may also lead to the development of new therapeutic targets and strategies for immune diseases and cancers.



The cell membrane not only separates the intracellular space from the external environment, but also provides a platform for the processing and transduction of inter- or intra-cellular signals. The two-dimensional geometry, the interaction between proteins and lipids, and the local membrane curvature all converge to generate emergent properties of membrane-proximal signaling whereas the underlying mechanisms are not well understood. The T cell receptor (TCR) pathway represents a good example of this scenario. Although major components in the pathway have been identified, it remains unclear how individual components assemble and coordinate to build up a pathway for effectively receiving and amplifying signals from pathogenic antigens. The goal of Dr Su's research program is to understand the general principals of membrane-proximal signaling under the biological context of T cell activation and other immune response processes.

10:20 - 10:35 a.m.

SHORT TALK: ANDREA AMITRANO, University of Rochester

Andrea M. Amitrano, Brandon J. Berry, Andrew P. Wojtovich & Minsoo Kim

Regulation of CD8+ T cell Metabolism in the Tumor Microenvironment

Despite recent advancements in the treatment of malignant tumors, cancer continues to be a widespread disease and a leading cause of death. CD8+ T cell-based immunotherapy has emerged as a potential treatment for several types of cancer. Chimeric antigen receptor transduced T cells (CAR-T cells) have been designed to augment CD8+ T cell persistence and anti-tumor activity. CAR-T cells have demonstrated tremendous success in eradicating hematological malignancies. However, CAR-T cells exhibit limited success in solid tumors. This is in part due to the tumor microenvironment (TME) which presents significant metabolic challenges to CD8+ T cells through the depletion of oxygen, glucose, and other key nutrients. Therefore, CD8+ T cells and tumor cells engage in fierce metabolic competition which often causes T cell exhaustion, resulting in reduced T cell functions. Our preliminary studies demonstrate that there is an increase in both mitochondrial mass and function during the activation of human T cells in vitro, demonstrating the importance of mitochondrial function in manufactured therapeutic T cells. In addition, our study shows increased oxygen consumption and glycolysis during activated CD8+ T cell migration, suggesting that T cell migration utilizes significant energy. Therefore, the development of a technique to specifically enhance the metabolism of T cells is an attractive target to improve CART cell function at the target tumor site.

To overcome the metabolic challenges in the TME and to boost both anti-tumor effector functions and migration of exhausted CD8+ T cells, we developed a genetically encoded photoactivatable proton pump (PA-OxPhos) that is expressed in the inner mitochondrial membrane. The outward proton pumping across the inner mitochondrial membrane by light stimulation of PA-OxPhos mimics the electron transport chain to induce increased mitochondrial ATP production. I hypothesize that increasing mitochondrial ATP production can overcome metabolic deficits effector CD8+ T cells face in the TME. This approach may provide CD8+ T cells a competitive energetic advantage in the TME and thus potentiate anti-tumor functions. Activation of PA-OxPhos in CD8+ T cells enhances migration on ICAM-1 compared to CD8+ T cells not expressing PA-OxPhos. Additionally, inhibition of glycolysis and ATP synthase decreases the velocity of activated CD8+ T cells on ICAM-1, further indicating the importance of ATP in T cell migration. Moving forward, I will investigate the impact of PA-OxPhos on effector functions of CD8+ T cells with the goal of increasing anti-tumor activity of CAR-T cells in solid tumors.

11:10 - 11:50 p.m.

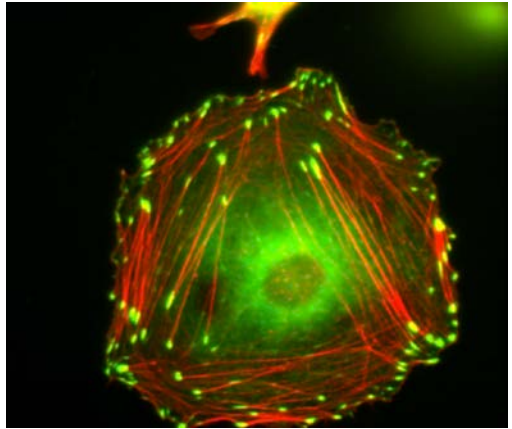
ANNA HUTTENLOCHER, M.D.

Professor of Medical Microbiology & Immunology and Pediatrics
University of Wisconsin

Imaging Inflammation and its Resolution

RESEARCH INTERESTS

The Huttenlocher Lab's research focuses on understanding the basic molecular mechanisms that regulate cell movement—in the context of wound healing, inflammation and cancer. Cell migration plays a central role in many different disease processes including cancer, heart disease and autoimmune disease. Insight into the mechanisms that regulate cell migration will



contribute to our understanding of basic cellular processes, but will also aid in the identification of new treatment strategies for a wide variety of medical conditions. Despite extensive interest in the receptors and mechanisms involved during cell migration, many fundamental questions remain unanswered. What are the mechanisms by which a cell initiates and then subsequently stops directional cell migration? How are intracellular signaling events coordinated both temporally and spatially to promote productive, directional cell movements? What are the mechanisms that regulate the migration of leukocytes into areas of inflammation? How do tumor cells invade and metastasize?

The lab's research is aimed at understanding the cellular and molecular mechanisms that regulate cell migration using biochemical genetic and imaging approaches. They use state of the art live imaging, biosensors and photomanipulation to examine and control polarity of cell signaling during cell migration in live animals. Dr Huttenlocher also examines host-pathogen interactions in zebrafish.

11:50 – 12:05 p.m.

SHORT TALK: OLIVER BRACKO, Cornell University

Oliver Bracko, Nancy E. Ruiz-Urbe, Jean C. Cruz-Hernández, Madison Swallow, Mohammad Haft-Javaherian, Muhammad Ali, Brendah Njiru, Kaja Falkenhain, Laibaik Park, Costantino Iadecola² Nozomi Nishimura, Chris B. Schaffer

Selective Inhibition of NOX2 Decreases Non-flowing Brain Capillaries, Increases Flow & Improves Cognition in Alzheimer's Disease Models

It has been known for decades that Alzheimer's Disease (AD) patients and AD mouse models display reduced cerebral blood flow (CBF). Using in vivo two-photon imaging, we recently identified the cellular mechanism that underlies this hypoperfusion. We found that neutrophils transiently adhere to the endothelial cell wall in about 2% of capillaries in the APP/PS1 mouse model of AD, plugging blood flow in those capillary segments (0.4% in wild-type mice). Blocking this adhesion using an antibody against a neutrophil surface protein increased CBF in APP/PS1 mice to near wild-type levels and led to a rapid improvement in cognitive function. The molecular drivers that link amyloid-beta pathology to this capillary plugging remain unknown. Here, we report that inhibiting NOX2-containing NADPH-oxidase, a reactive oxygen species producing enzyme shown to be activated in APP/PS1 mice, for two weeks with a small peptide, gp91 ds – tat, decreased the fraction of capillaries with stalled blood flow by 67%, increased CBF by 29%, and improved performance on object replacement and y-maze spatial memory tasks. A scrambled version of the peptide inhibitor did not lead to any of these changes. This study implicates the NOX2 pathway as a new molecular mechanism underlying capillary stalling and CBF reductions APP/PS1 mice and could represent a molecular pathway with potential therapeutic opportunities for AD.

12:05 – 12:55 p.m.

DANIEL HAMMER, Ph.D.

Alfred and Meta Ennis Professor of Bioengineering and Chemical & Biomolecular Engineering
University of Pennsylvania

Force Spectroscopy and Upstream Migration

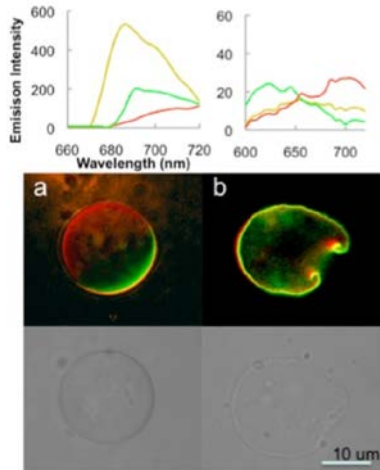
RESEARCH INTERESTS

The Hammer Laboratory has two main research areas: synthetic capsules and cell motility & adhesion.

Along with collaborators, the Hammer lab has developed polymersomes or biomimetic vesicles composed of diblock copolymers or recombinant proteins. These have the potential to serve as drug-delivery, imaging devices and synthetic cells (also known as protocells). These vesicles can be engineered to carry molecules in their interior and on their surface and have shown their potential to adhere to specific cell

adhesion molecules. Furthermore, by embedding specific molecules within the membrane on polymersome, the Hammer group can cause them to photo-destruct. The ability to assemble capsules from recombinant proteins allows facile incorporation of functional adhesion receptors and responsive motifs, such as protease cleavable domains.

Cell motility and adhesion is critical to many processes in the body including the proper functioning of the immune response. The lab uses compliant surfaces of different stiffness and micro post arrays to measure the forces exerted during motility for neutrophils, T- lymphocytes, macrophages and dendritic cells. Finally, the group studies the homing of T-cells using Adhesive Dynamics, in which we can simulate the rolling, tethering, and firm adhesion of cells in the vasculature.



Sensing membrane stress

2:30 – 2:45 p.m.

SHORT TALK: **Alessandra Araujo**, University of Rochester
Alessandra Araujo, Américo López-Yglesias, Elise Burger, Ellie T. Camanzo,
Andrew T. Martin, Alexandra Safronova, Felix Yarovinsky

IFN- γ Directly Mediates Degranulation and Death of Metabolically Altered Paneth Cells During Toxoplasma gondii Infection

Paneth cells secrete unique antimicrobial peptides and growth factors which allow for optimal intestinal homeostasis and replication of the intestinal stem cells. While it has become well appreciated that Paneth cells dysfunction results in dysbiosis and increased susceptibility to intestinal inflammation, how infection-induced acute inflammation interferes with Paneth cell functions is still unclear. Here we show that infection with the parasite *T. gondii* results in IFN- γ mediated Paneth cell death and loss of antimicrobial peptides which is dependent on IFN- γ receptor. In order to investigate the mechanism of IFN- γ mediated Paneth cell death we evaluated classical death pathways. We concluded that the loss of Paneth cells was not due to programmed apoptosis, pyroptosis, or autophagic cell death. Instead, metabolite analysis revealed that Paneth cell death was associated with mitochondrial dysfunction and abnormal energy mobilization. In agreement with altered metabolism, activity of mTORC1 complex, an important energy sensor and controller of protein synthesis was also altered in Paneth cells during *T. gondii* infection.

2:45 – 3:25 p.m.

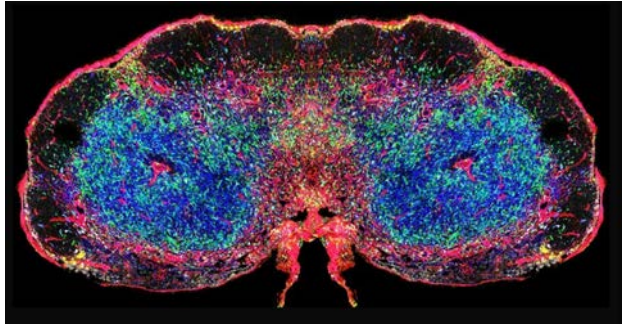
MICHAEL GERNER, Ph.D.

Assistant Professor of Immunology
University of Washington

Organization of Innate-Adaptive Cell Crosstalk in Lymphoid Tissues

RESEARCH INTERESTS

The immune system is composed of a highly heterogeneous network of innate and adaptive cell populations with unique phenotypic and functional properties. These diverse cells are



differentially distributed within tissues, creating specialized microenvironments with select roles and functions. Furthermore, these localized cells are in constant communication with one another and coordinate their activities to generate immune responses specifically tailored to distinct challenges. The Gerner lab investigates these processes directly in situ by studying micro-anatomical tissue organization on cellular and molecular levels, cell-cell communication events that generate immune responses, as well as localized effector responses in inflamed peripheral sites that allow for protective immunity. The lab utilizes cutting-edge microscopy tools, such as 2-photon intra-vital microscopy, multi-parameter whole-organ confocal microscopy and analytical Histo-Cytometry. Investigating such cell-cell crosstalk and coordinated activity, as well as the broader structure-function relationships for whole organs and inflamed sites is critical for understanding the underpinnings of immune processes. These studies will lead to improved design of vaccines and immuno-modulatory therapeutics and will allow for tight and regionalized regulation of select cells and functions.

3:25 – 3:40 p.m.

SHORT TALK: **Emma Reilly**, University of Rochester
Emma C Reilly, Kris Lambert Emo, Aitor Nogales, Cory J Poole, Gloria
Pryhuber, Luis Martínez-Sobrido, and David J Topham

*Tracheal Submucosal Glands Offer a Novel Habitat for Resident
Memory CD8+ T cells*

During acute influenza infection, CD8 T cells enter the lungs and the trachea and facilitate elimination of infected epithelial cells at the airway surface. A small population of CD8 T cells, termed resident memory T cells (TRM), remains within pulmonary tissue after viral clearance to provide local protection against future exposures. Prior reports suggest that TRM cells are exclusively poised within and underlying the airway epithelium, however our data show that CD8 T cells are most proximal to the airway at day 7 post-infection, and move toward the exterior by days 9 and 14. This led us to hypothesize that a feature of the trachea or the infection was drawing these cells away from the airway epithelium. Using E-cadherin reporter mice in an intravital tracheal imaging model, we discovered virus-specific CD8 T cells within submucosal glands of the trachea at days 14 and 90 post-infection. We show that these glands can be productively infected by human influenza A virus and that labeled ovalbumin given intranasally can be taken up directly into these glands. Similar to airway epithelial cells, glands express E-cadherin and collagen, the ligands for TRM integrins CD103 and CD49a, respectively. CD8 T cells within glands at day 14 are more motile compared with the few cells remaining in the airway at this time point, however at 90 days post-infection T cells in both compartments display more active “flossing”, likely as a means of surveillance, compared with cells not associated with E-cadherin expressing cells. Overall, these data suggest that submucosal glands in the trachea provide a novel microenvironment for TRM, allowing them to patrol for future infections.

3:40 – 4:20 p.m.

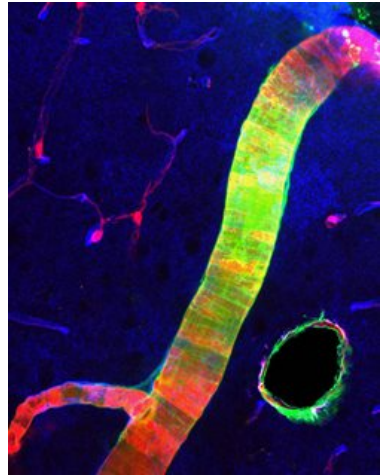
MAIKEN NEDERGAARD, M.D., D.M.Sc.

Professor of Neurology and Neuroscience and
Co-Director of the Center for Translational Neuromedicine
University of Rochester

Glymphatic/Lymphatic Connections

RESEARCH INTERESTS

Research in the Nedergaard laboratory has been dedicated to deciphering the role of neuroglia. Astrocytes in particular (the most prevalent type of neuroglia in the central nervous system) have been considered as simply the scaffolding for neurons, structurally supporting neural networks, providing metabolic substrates, and facilitating waste removal. Work over the past decade suggests a dramatically different and more active role for astrocytes in the brain, one in which they dynamically contribute to and regulate neuronal activity. Understanding the contribution of neuroglia to brain function remains an active and open pursuit in science, holding major implications for developing targeted therapies to neurological disease.



Throughout most of the body, a complex system of lymphatic vessels is responsible for cleansing the tissues of potentially harmful metabolic waste products, accumulations of soluble proteins and excess interstitial fluid. But the central nervous system lacks a lymphatic vasculature. The breakdown of the brain's innate clearance system may in fact underlie the pathogenesis of neurodegenerative disorders such as Alzheimer's, Parkinson's, and Huntington's disease, in addition to ALS and chronic traumatic encephalopathy. Recently, the Nedergaard lab found that small channels 'piggybacking' the blood vasculature allow the CSF to flow into the brain tissue along para-arterial spaces and exit via a para-venous route. This 'peri-vascular' route for CSF-ISF exchange constitutes a complete anatomical pathway, which Nedergaard dubbed the **glymphatic** system due to its dependence on glial cells performing a 'lymphatic' cleansing of the brain interstitial fluid.

POSTERS

Presenter(s) listed BOLD

1.	<p><i>Regulation of CD8+ T cell Metabolism in the Tumor Microenvironment</i> Andrea M. Amitrano^{1,2}, Brandon J. Berry³, Andrew P. Wojtovich^{3,4}, Ph.D., Minsoo Kim^{1,2}, Ph.D. ¹ Department of Pathology, University of Rochester, Rochester, NY ² Department 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</p>
2.	<p><i>IFN-γ directly mediates degranulation and death of metabolically altered Paneth Cells during Toxoplasma gondii infection.</i> Alessandra Araujo, Américo López-Yglesias, Elise Burger, Ellie T. Camanzo, Andrew T. Martin, Alexandra Safronova, Felix Yarovinsky Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, New York</p>
3.	<p><i>Selective inhibition of NOX2 decreases the incidence of non-flowing brain capillaries, leading to increased cerebral blood flow and improved cognition in mouse models of Alzheimer's disease</i> Oliver Bracko¹, Nancy E. Ruiz-Urbe¹, Jean C. Cruz-Hernández¹, Madison Swallow¹, Mohammad Haft-Javaherian¹, Muhammad Ali¹, Brendah Njiru, Kaja Falkenhain¹, Laibaik Park², Costantino Iadecola², Nozomi Nishimura¹, Chris B. Schaffer¹ ¹Nancy E. and Peter C. Meinig School of Biomedical Engineering, Cornell University, Ithaca, New York, USA ²Feil Family Brain and Mind Research Institute, Weill Cornell Medicine, New York, New York, USA</p>
4.	<p><i>Micelle-Encapsulated Quantum Dots as Structural Mimics of Spherical β-Amyloid Oligomers to Investigate the Cellular Mechanisms of Alzheimer's Associated Neurodegeneration</i> Wesley Chiang^{1,2}, Todd Krauss^{2,3}, Bradley Nilsson³, Harris Gelbard⁴ ¹Department of Biochemistry & Biophysics, ²Institute of Optics, ³Department of Chemistry, ⁴Department of Neurology – University of Rochester, Rochester NY</p>

5.	<p><i>Intravital three-photon microscopy of entire mouse lymph node</i> Kibaek Choe, Chris Xu School of Applied and Engineering Physics, Cornell University, Ithaca, New York, USA</p>
6.	<p><i>Imaging of CD4+ cells in the trachea in live mice</i> Sophia Eliseeva¹, Angie Hughson^{2,3}, Emma Rielly^{2,3}, Kris Lambert Emo^{2,3}, Dave Topham^{2,3}, Deborah Fowell^{2,3}, and Steve Georas¹ Pulmonary and Critical Care, University of Rochester Medical Center, Rochester NY¹, David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester NY², Department of Microbiology and Immunology, University of Rochester, Rochester NY³</p>
7.	<p><i>Genetically-Encoded Tracers of Interstitial Fluid Flow in Mouse Brain Accumulate at Arterioles</i> Chi-Yong Eom, David M. Small, Chris B. Schaffer, and Nozomi Nishimura Nancy E. and Peter C. Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY, USA.</p>
8.	<p><i>Targeting the Tumor-Draining Lymph Node to Enhance Anti-Tumor Responses in Pancreatic Ductal Adenocarcinoma</i> Booyeon J. Han^{1,2}, Bradley N. Mills^{1,2,3}, Shuyang Qin^{1,2}, Joseph Murphy^{1,2}, Taylor Uccello^{1,2}, Jian Ye^{2,3}, Brian A. Belt^{2,3}, Peter A. Prieto^{2,3,4}, David C. Linehan^{2,3,4}, Scott A. Gerber^{1,2,3,4} ¹Department of Microbiology and Immunology, University of Rochester, Rochester, NY. ²Center for Tumor Immunology, University of Rochester Medical Center, Rochester, NY. ³Department of Surgery, University of Rochester Medical Center, Rochester, NY. ⁴Wilmot Cancer Institute, University of Rochester Medical Center, Rochester, NY.</p>
9.	<p><i>In vivo Tracking: Using a Photoconvertible System to Probe Immune Cell Entry and Exit in the Context of Inflammation</i> Alicia Healey and Deborah Fowell, PhD Department of Microbiology and Immunology, University of Rochester, Rochester NY</p>

10.	<p><i>Imaging Classification and Analysis of CD8+ T-Cells During and After Influenza Virus Infection</i> Ian Smith¹, Rakshanda Jha¹, Emma C Reilly¹, Kris Lambert Emo¹, and David Topham^{1,2} David H. Smith Center for Vaccine Biology and Immunology¹, Department of Microbiology and Immunology², University of Rochester, Rochester, New York 14642.</p>
11.	<p><i>Lineage Tracing Studies of Lymphatic Muscle Cells in Mice Identify a Unique Population Distinct from Skeletal and Vascular Smooth Muscle Progenitors</i> H. Mark Kenney^{1,2}, Richard D. Bell^{1,2}, Elysia A. Masters^{2,3}, Edward M. Schwarz^{1,2,3,4} ¹Department of Pathology and Laboratory Medicine, ²Center for Musculoskeletal Research, ³Department of Biomedical Engineering, ⁴Department of Orthopaedics, University of Rochester, Rochester NY</p>
12.	<p><i>Development of Murine Lung and Tracheal Organoids</i> Kris Lambert Emo, and David J. Topham David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester School of Medicine and Dentistry, Rochester NY</p>
13.	<p><i>In situ neutrophil efferocytosis shapes T cell immunity to influenza infection</i> Kihong Lim, Tae-hyoun Kim, Alissa Trzeciak, Hen Prizant, Emma C. Reilly, Andrea Amitrano, Deborah J. Fowell, David J. Topham, and Minsoo Kim Department of Microbiology and Immunology, David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester, NY</p>
14.	<p><i>Transcriptional profiles of CD14+ cells in situ in melanoma reveal plasticity, localization dependent function and specific T cell interactions</i> Jan Martinek¹, Kyung In Kim¹, Te-Chia Wu¹, Hannah Boruchov¹, Ananya Gulati¹, Lili Sun¹, Victor Wang¹, Joshy George¹, Philipp Henrich¹, Florentina Marches¹, Anthony Rongvaux², Michael Chiorazzi³, Jeff Chuang¹, Paul Robson¹, Richard Flavell³, Jacques Banchereau¹, and Karolina Palucka¹ ¹The Jackson Laboratory for Genomic Medicine, Farmington, CT; ²Fred Hutchinson Cancer Research Center, Program in Immunology, Seattle, WA; ³Department of Immunobiology, School of Medicine, Yale University, New Haven, CT</p>

15.	<p><i>Lifting restrictions on fluorescence microscopy through machine learning based spectral unmixing and super resolution</i> Tristan D. McRae^{1,2}, Yurong Gao^{1,2} ¹Multiphoton Research Core Facility, University of Rochester Medical Center, Rochester NY ²Department of Neuroscience, University of Rochester Medical Center, Rochester NY</p>
16.	<p><i>Hyperspectral multiphoton microscopy for in vivo visualization of spectrally-overlapped fluorescent labels.</i> Menansili A. Mejooli¹, Amanda J. Bares¹, Scott Leddon², Steven Tilley, II¹, Jingyuan Dong¹, Minsoo Kim², Deborah J. Fowell², Nozomi Nishimura¹, and Chris B. Schaffer¹ ¹Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY 14853 ²Dept. of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY 14642</p>
17.	<p><i>Histologic Information Provided by Rapid Two-Photon Imaging of Lung Tissue</i> Jenna Montague¹, Vincent Ralph D. Ching-Roa¹, Dr. Michael Drage², Dr. Michael G. Giacomelli¹ ¹Department of Biomedical Engineering, University of Rochester, ²University of Rochester Medical Center</p>
18.	<p><i>Human influenza reactivity to conserved influenza hemagglutinin and its relation to age and repeat vaccination</i> Savannah Moritzky, Katherine Richards, and Andrea Sant Department of Microbiology and Immunology, University of Rochester, Rochester NY David H. Smith Center for Vaccine Biology and Immunology, University of Rochester, Rochester NY</p>
19.	<p><i>Potentiating protective local immunity to influenza virus through a novel self-assembling vaccine platform</i> Sean Nelson[*], Amy Rasley^{**}, Nicholas Fischer^{**}, and Andrea Sant[*] [*]David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY 14642, USA ^{**}Biosciences and Biotechnology Division, Lawrence Livermore National Laboratory, Livermore, CA 94551, USA</p>

20.	<p><i>Overcoming resistance to PD-L1 blockade by induction and activation of tumor-residing cross-presenting dendritic cells</i> Takaaki Oba¹, Tibor Keler², Henry C. Marsh², Hans Minderman³, Scott I. Abrams⁴, Fumito Ito^{1,5}) ¹Center for Immunotherapy, Roswellpark Comprehensive Cancer Center, Buffalo NY, ²Celldex Therapeutics, Inc., Needham MA, ³ Department of Immunology, Roswellpark Comprehensive Cancer Center, Buffalo NY, ⁴ Department of Flowcytometry core, Roswellpark Comprehensive Cancer Center, Buffalo NY, ⁵ Department of Surgical Oncology, Roswellpark Comprehensive Cancer Center, Buffalo NY</p>
21.	<p><i>The Effect of Aging on Innate Immune Responses to Influenza Virus Infection, and Processing of Influenza Virus Antigens</i> Raven M. Osborn^{1,3} ; David Topham, PhD³ ; Stephen Dewhurst, PhD^{1,3} ; Juilee Thakar, PhD^{2,3} ¹ Clinical and Translational Science Institute, University of Rochester, Rochester, NY; ² Department of Biostatistics and Computational Biology, University of Rochester, Rochester, NY; ³ Department of Microbiology and Immunology, University of Rochester, Rochester, NY</p>
22.	<p><i>Distinct mechanisms of FcγR- and complement-mediated hypophagia</i> Jonathan Pinney, Charles Chu, Fátima Rivera-Escalera, Hannah Whitehead, Karl VanDerMeid, Clive Zent, and Michael R. Elliott Department of Microbiology and Immunology, University of Rochester, Rochester NY</p>
23.	<p><i>CXCL10+ cell clusters define ‘hot spots’ for Th1 cell tissue entry and activation</i> Hen Prizant¹, Nilesh Patil¹, Seble Negatu¹, Alexandra Livingstone¹, Joanna Groom², Andrew Luster³, Deb Fowell¹ ¹Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY, ²Division of Immunology, Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia; Department of Medical Biology, University of Melbourne, Parkville, Victoria, Australia, ³Center for Immunology and Inflammatory Diseases, Division of Rheumatology, Allergy and Immunology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA</p>

24.	<p><i>Tracheal Submucosal Glands Offer a Novel Habitat for Resident Memory CD8 T cells</i></p> <p>Emma C Reilly¹, Kris Lambert Emo¹, Aitor Nogales⁵, Cory J Poole³, Gloria Pryhuber^{3,4}, Luis Martínez-Sobrido², and David J Topham^{1,2}</p> <p>David H Smith Center for Vaccine Biology and Immunology¹, Department of Microbiology and Immunology², Department of Pediatrics³, Department of Environmental Medicine⁴, University of Rochester, Rochester, NY, Centro de Investigación en Sanidad Animal (CISA)⁵, Valdeolmos, Madrid 28130, Spain</p>
25.	<p><i>Ligand Availability, and not $\alpha V\beta 3$ (alphaVbeta3) Integrin Expression Regulates Th1 Cell Migration</i></p> <p>Nick Reilly¹, Deborah Fowell², Patrick Oakes³</p> <p>¹Department of Physics and Astronomy, University of Rochester, Rochester, NY</p> <p>²David 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</p> <p>³Loyola University Chicago, Stritch School of Medicine, Department of Cell & Molecular Physiology, Maywood, IL</p>
26.	<p><i>T cell fate mapping: dysfunction or memory?</i></p> <p>Cooper Sailer¹, Minsoo Kim²</p> <p>¹Department of Pathology and Laboratory Medicine, University of Rochester Medical Center, Rochester, New York</p> <p>²Department of Microbiology and Immunology, Center for Vaccine Biology and Immunology, University of Rochester Medical Center, Rochester, New York</p>
27.	<p><i>Tropomyosin and Myosin 18A play important roles in the organization, dynamics and function of the actomyosin arcs that form at the T cell immunological synapse</i></p> <p>Dillon C. Schrock¹, Sricharan Murugesan², Jordan R. Beach³, and John A. Hammer¹</p> <p>¹Cell and Developmental Biology Center, National Heart, Lung, and Blood Institute; National Institutes of Health, Bethesda, MD</p> <p>²Zeiss Microscopy, Gaithersburg, MD</p> <p>³Loyola University, Department of Cell and Molecular Physiology, and Cardiovascular Research Institute, Chicago, IL</p>

28.	<p><i>Complement component C1q regulates sepsis progression by acting as an inflammation checkpoint</i></p> <p>Alissa Trzeciak¹, Yelena Lerman¹, Ma Rie Kim², Eric Harrower¹, Amy Rovitelli³, Casey Robinson³, Anthony Pietropaoli³, and Minsoo Kim¹.</p> <p>¹Department of Microbiology and Immunology, University of Rochester, Rochester, NY.</p> <p>²Department of Biomedical Engineering, University of Rochester, Rochester, NY.</p> <p>³Department of Medicine, Pulmonary Diseases and Critical Care, University of Rochester, Rochester, NY</p>
29.	<p><i>The role of PI3Kγ signaling in microglial dynamics and experience-dependent synaptic plasticity</i></p> <p>Brendan S. Whitelaw, Evelyn K. Matei, Ania K. Majewska Department of Neuroscience, University of Rochester, Rochester NY</p>
30.	<p><i>Microvessel occlusions alter amyloid-beta plaque morphology in a mouse model of Alzheimer's disease</i></p> <p>Silvia Zhang, Evan D. Bander, Yurim Lee, Celia Muoser, Chris B. Schaffer, Nozomi Nishimura Nancy E. and Peter C. Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY</p>

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Cornell University
Harvard University
Johns Hopkins University School of Medicine
Loyola University Chicago
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Nazarbayev University
Rochester Institute of Technology
Roswell Park Comprehensive Cancer Center
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