2nd Annual
IMMUNE IMAGING SYMPOSIUM

Hosted by:

THE PROGRAM FOR ADVANCED IMMUNE BIOIMAGING

&

UNIVERSITY OF ROCHESTER

Saturday, November 5th, 2016

Saunders Research Building
University of Rochester

8:00 a.m. – 6:30 p.m.

About our program:

PROGRAM FOR ADVANCE 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.

8:00 - 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.
PAUL KUBES, University of Calgary
Tracking different immune cells in sterile inflammation

9:40 - 9:55 a.m.
SHORT TALK: **Sung Ji Ahn,** Cornell University
Inflammatory responses after a laser-induced cortical microhemorrahge

9:55 - 10:35 a.m.
ELLEN ROBEY, UC Berkeley
T cell behavior in vivo

10:35 - 10:50 a.m.
SHORT TALK: **Kyun-Do Kim,** University of Rochester
Targeted calcium influx boosts cytotoxic T lymphocyte function in the tumor microenvironment

11:20 - 12:00 p.m.
DANIEL MUCIDA, The Rockefeller University
Tissue Adaptation: consequences of immunity and tolerance

12:00 – 12:15 p.m.
SHORT TALK: **Amanda Bares,** Cornell University
Hyperspectral Multiphoton Microscopy for simultaneous in vivo visualization of multiple cell types

12:15 – 12:55 p.m.
CHRIS HUNTER, University of Pennsylvania
Imaging immunity to toxoplasma in the CNS

3:00 – 3:15 p.m.
SHORT TALK: **Emma Reilly,** University of Rochester
Understanding the Roles of Tissue Binding Integrins in the Development and Maintenance of Resident Memory CD8 T cells

3:15 – 3:55 p.m.
THORSTEN MEMPEL, Massachusetts General Hospital, Harvard
T cell migration and function during the anti-tumor response

3:55 – 4:10 p.m.
SHORT TALK: **Hanna Vinitsky,** University of Rochester
Reciprocal Effects of Glymphatic Function and the Experimental Autoimmune Encephalomyelitis (EAE) Model of Multiple Sclerosis

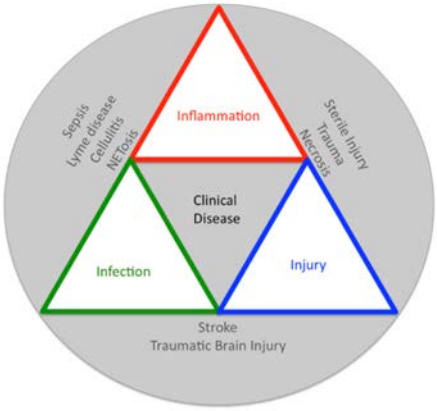
4:10 – 4:50 p.m.
DEBORAH FOWELL, University of Rochester
Programming effector T cells for interstitial motility

4:50 – 5:00 p.m.
Meeting Wrap-up and Awards

9:00 - 9:40 a.m.

PAUL KUBES, Ph.D.
Professor, University of Calgary, Calgary, Canada
Tracking different immune cells in sterile inflammation

RESEARCH INTERESTS



The Kubes lab is committed to understanding complex immune responses in the context of human clinical disease. The primary focus is to directly visualize the roles of immune cells during inflammation, infection and tissue Injury. The lab is leading the way in directly imaging the immune system using cutting edge technology, including spinning-disk confocal and multi-photon microscopy. By imaging complex cellular behaviors in real time, both in vitro and in vivo, the group can begin to understand how immune cells, such as neutrophils, monocytes, NKT cells and Kupffer cells function under physiological and pathological disease states.

9:40 - 9:55 a.m.

SHORT TALK: **Sung Ji Ahn,** Cornell University
Inflammatory responses after a laser-induced cortical microhemorrahge

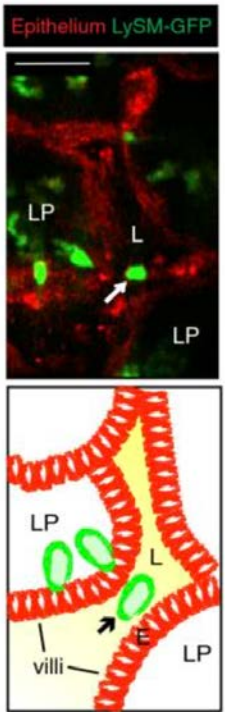
Sung Ji Ahn[1], Josef Anrather[2], Nozomi Nishimura[1], and Chris B Schaffer[1]
[1]Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY. [2] Feil Family Brain and Mind Institute, Weill Cornell Medical College, New York, NY.

Clinical studies have linked microhemorrhages with cognitive decline, although how these small bleeds impact cognition is not understood at the cellular level. We used optical approaches to create microhemorrhages and image the cellular responses in the brain of mice. Irradiation with tightly-focused femtosecond laser pulses ruptured targeted penetrating arterioles, producing a hematoma of ~150-μm diameter in the cortex. Such lesions did not cause neural death, but did drive an increase in the number of nearby activated inflammatory cells. We aim to understand the relative role of brain-resident and blood-derived inflammatory cells as this triggered inflammation could have a profound impact on neural health and function. We used chimeric transgenic animals with different inflammatory cell populations labeled with fluorescent proteins, and mapped the spatial and temporal profile of the cellular response after a microhemorrhage. We observed brain-resident microglia activate soon after the microhemorrhage and the doubling of their density adjacent to the lesion is the dominant aspect of the inflammatory response. A small number of blood-derived inflammatory cells were also found, with proinflammatory monocytes (CCR2+) entering the brain during the first two days after the lesion, and patrolling monocytes (CX3CR1+) entering after two days. The doubling of microglia density one day after the lesion was due to migration and proliferation of microglia. Using time-lapse imaging, we observed microglia immediately send processes to, and then migrate over one day toward, the ruptured arteriole. For microglia within 150 μm of the targeted vessel, the migration speed was 0.9 μm/hr over the first 16 hours and then slowed. We assessed proliferation by injecting EDU (25mg/kg) every 8 hours for two days following a cortical microhemorrhage. Microglia expressing EDU were found primarily in a donut-shaped band surrounding the lesion, and not in close proximity to the hemorrhage. In summary, small brain bleeds trigger migration of nearby microglia toward the lesion and proliferation of slightly more distant microglia causing a doubling the microglia density near the lesion, and drive the infiltration of a small number of inflammatory and patrolling monocytes. In ongoing work, we are exploring how this inflammation could affect the health and function of nearby brain cells and thus drive cognitive decline.

9:55 - 10:35 a.m.

ELLEN ROBEY, Ph.D.
Professor, UC Berkeley, Berkeley, CA
T cell behavior in vivo

RESEARCH INTERESTS



The Robey lab is interested in how signaling pathways control cell fate decisions. By using T cell development and immune responses in the mouse as model systems, they take advantage of the powerful genetic approaches available in the mouse, while learning about a mammalian immune system that is very close to our own. The lab uses mouse infection by *Toxoplasma gondii* to understand the mammalian immune response to an intracellular pathogen, with particular relevance for the design of vaccines for CD8-mediated protection and oral pathogens. Current investigations focus on the mechanisms that make certain T cell responses effective and the impact of innate immune responses on CD8 T cell responses. The group makes extensive use of 2-photon imaging approaches to observe and analyze T cell behavior in real-time in situ during development and in infectious disease.

10:35 - 10:50 a.m.

SHORT TALK: **Kyun-Do Kim,** University of Rochester
Targeted calcium influx boosts cytotoxic T lymphocyte function in the tumor microenvironment

Kyun-Do Kim[1], Seyeon Bae[1], Tara Capece[1], Hristina Nedelkovska[1], Rafael G. de Rubio[2], Alan V. Smrcka[2], Woojin Jung[3], Byeonghak Park[3], Tae-il Kim[3], and Minsoo Kim. *[1]Department of Microbiology and Immunology, David H. Smith Center for Vaccine Biology and Immunology, [2]Department of Pharmacology & Physiology, University of Rochester, Rochester, NY. [3]School of Chemical Engineering, Sungkyunkwan University, Suwon, Republic of Korea, Center for Neuroscience Imaging Research, Institute of Basic Science, Suwon, Republic of Korea.*

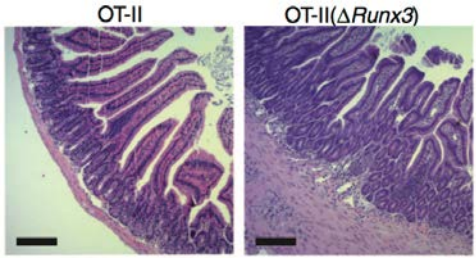
Adoptive cell transfer utilizing tumor-targeting cytotoxic T lymphocytes (CTLs) is one of the most effective immunotherapies against hematological malignancies, but significant clinical success has not yet been achieved in solid tumors due in part to the strong immunosuppressive tumor microenvironment. Systemic or intratumoral delivery of an immune boosting molecule to overcome local suppression has been proposed, but the full potential is limited by non-specific stimulation of tumor growth, metastasis, and angiogenesis.

Here, we show that suppression of CTL killing by CD4+CD25+Foxp3+ regulatory T cell (Treg) is mainly mediated by TGFβ-induced inhibition of inositol trisphosphate (IP3) production, leading to a decrease in T cell receptor (TCR)-dependent intracellular Ca2+ response. Both in vitro and in vivo assays revealed that highly selective optical control of Ca2+ signaling in adoptively transferred CTLs was sufficient to overcome immunosuppression at the tumor site by enhancing T cell activation, IFN-γ production and antitumor cytotoxicity, leading to a significant reduction in tumor growth in mice. Together, our findings indicate that the targeted optogenetic stimulation of intracellular Ca2+ signal allows for the remote control of cytotoxic effector functions of adoptively transferred T cells with outstanding spatial resolution by boosting T cell immune responses only at the targeted tumor sites.

11:20 - 12:00 p.m.

DANIEL MUCIDA, Ph.D.
Associate Professor, The Rockefeller University, New York, NY
Tissue Adaptation: consequences of immunity and tolerance

RESEARCH INTERESTS



At the surface of the intestinal lining, immune responses must be carefully balanced: invasive pathogens must be eliminated or excluded, while nutrients and trillions of commensal microbes that contribute to homeostasis must be tolerated. The Mucida lab studies how the immune system associated with intestinal mucosae walks this fine line by generating efficient protective responses without jeopardizing its tolerance to innocuous antigens. The goal is to characterize the cellular and molecular mechanisms that lead to the development of pro-inflammatory and regulatory cells at the mucosal surfaces, both from the innate and the adaptive immune system. The laboratory uses imaging and genetic tools to investigate innate and adaptive immune cell dynamics in the intestinal tissue, and how they assimilate different environmental cues and signals from commensal flora to maintain the integrity of the epithelial barrier and intestinal homeostasis.

12:00 – 12:15 p.m.

SHORT TALK: **Amanda Bares,** Cornell University
Hyperspectral Multiphoton Microscopy for simultaneous in vivo visualization of multiple cell types

Amanda J. Bares, Mitchell A. Pender, Menansili A. Majooli, Steven Tilley, Kuang E. Chen, Jingyuan Dong, Nozomi N. Nishimura, and Chris B. Schaffer.
Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY.

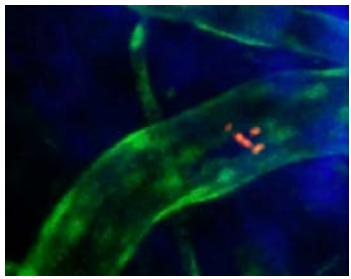
Studies of complex normal and disease state physiology require the visualization of multiple cells and cell types in live, optically scattering tissue. Multiphoton microscopy provides the necessary resolution and optical sectioning deep in tissue, but fails to provide the spectral resolution to differentiate between multiple, overlapping fluorescent labels. Current multi-color detection systems use a dispersive element that is sensitive to light scattered in the sample, losing spectral resolution at depth in tissue. We have designed and implemented a multiphoton microscope able to collect 48 channels of spectral data, while maintaining spectral resolution at depth.

Fluorescence from the sample is collected through the microscope objective and subdivided into four broad color channels with fixed dichroics. The light is further divided by angle-tuned bandpass filters placed in front of each detector. These filters, VersaChrome filters by Semrock, provide a wide tuning range (~80 nm), small passbands (15-20 nm) and high transmission across all angles (>90%). The detection system optics are optimized to collect scattered light and reduce divergence of light incident on the filters, minimizing the effect of imaging deep on spectral detection characteristics. Images are collected from all four detectors simultaneously across four angles of the band-pass filters, one after the other, producing a 16-channel image across the entire visible spectrum. Taking advantage of fluorophore emission intensity as a function of excitation laser wavelength, we route three femtosecond-pulsed lasers into the microscope (800, 900, 1035 nm) and acquire a 16-channel image for each laser consecutively, producing a 48-channel image of excitation and emission data for every frame. 48-channel images are reduced to composites color coding for each fluorophore using linear unmixing, fitting measured pixel spectra to most likely sum of spectral signatures of fluorophores in the sample.

We have demonstrated the capabilities of our microscope with a broad range of samples from 10 colors of fluorescent beads in a gel, to 6 fluorescent labels in live mouse cortex using transgenic and topical dye methods. This microscope will enable dramatic increases in the number of fluorescent labels used in an in vivo experiment. Rather than limiting hypotheses to two or three cell types, we can now visualize all cells of interest in the tissue micro-environment and expand hypotheses to include multiple cell types or leave room for discovery through observation.

12:15 – 12:55 pm

CHRIS HUNTER Ph.D.
Professor and Chair, University of Pennsylvania, Philadelphia, PA
Imaging immunity to toxoplasma in the CNS



RESEARCH INTERESTS

The Hunter lab's research centers around understanding how the immune system deals with *Toxoplamsa gondii*. The first focus is on host pathogen interactions at the cellular level and how the parasite interacts with intracellular signaling pathways (NF-kB/JAK-STAT). Genetic approaches are being used to identify parasite factors that are involved in these events to address fundamental questions about which cells are important in the initiation of adaptive immunity and the role of cross-presentation in the development of T cell responses. Additional studies have helped to define the cytokine networks that regulate the balance between protective and pathological immune responses and current studies focus on the IL-6 family of cytokines in these events. Imaging studies are aimed at a better understanding the neuro-pathogenesis of toxoplasmic encephalitis (TE) in the immunocompromised patients who develop this disease.

3:00 – 3:15 p.m.

SHORT TALK: **Emma Reilly,** University of Rochester
Understanding the Roles of Tissue Binding Integrins in the Development and Maintenance of Resident Memory CD8 T cells

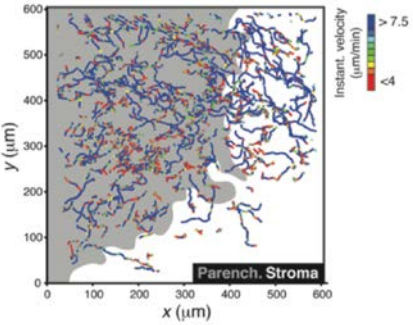
Emma C Reilly, Kris Lambert Emo, Aitor Nogales, Anthony DiPiazza, Luis Martínez-Sobrido, Andrea J Sant, and David J Topham. *Department of Microbiology and Immunology, University of Rochester, Rochester, N.Y.*

Lung tissue resident memory CD8 T cells (TRM) comprise a unique immune population that persists after influenza virus infection and are essential for protection from subsequent exposures. Integrins such as CD49a and CD103 that interact with the extracellular matrix or tissue ligands have been implicated in TRM formation and survival, however their exact role in establishing and maintaining the population is not well described. Therefore, we sought to interrogate the changes that occur in the absence of these integrins, with the hypothesis that inhibition of cell-tissue interactions alters motility and eventually long-term maintenance. In mice lacking collagen-binding integrin CD49a, we observe increases in the number of CD8 T cells expressing E-cadherin-binding CD103, suggesting that CD103 interactions may partiallycompensate for the absence of CD49a. However, absence of CD49a alone or in conjunction with blocking of CD103 results in changes in morphology, tissue localization, and motility. Together the data suggests these integrins are regulating CD8 T cell migration in the lung. To study maintenance, we can use the same model to evaluate TRM cells within the trachea at 3 months post-infection, and are currently developing a reporter virus to visualize CD8 T cells interacting with virally infected cells. Overall, we show that blocking CD8 T cell integrin–tissue interactions alters the cellular phenotype and motility at a time that is critical for the initiation of tissue memory. Additionally, we can utilize the tools that we are creating to visualize and understand TRM responses to heterosubtypic infections.

3:15 – 3:55 p.m.

THORSTEN MEMPEL, M.D., Ph.D.
Associate Professor, Massachusetts General Hospital, Harvard Medical School
T cell migration and function during the anti-tumor response

RESEARCH INTERESTS



The Mempel group studies how T lymphocytes traffic between lymphoid and non-lymphoid tissues and position themselves within these tissue environments to interact with various types of antigen-presenting cells (APC) to receive the instructive and selective cues that guide their development and function. To study these processes in vivo, the lab uses multiphoton intravital microscopy (MP-IVM) approaches which allow analysis, at single cell level, of the migration of immune cells in their physiological tissue context in living mice. In addition, the lab seeks to monitor the activities of various intracellular signaling pathways in real time and how these are controlled by intercellular contacts. Ultimately, integrating the analysis of dynamic gene activation processes and of the cellular interactions that drive these *in vivo* will provide us with a better understanding of the highly complex processes by which immune responses are regulated.

3:55 – 4:10 p.m.

SHORT TALK: **Hanna Vinitsky,** University of Rochester

Reciprocal Effects of Glymphatic Function and the Experimental Autoimmune Encephalomyelitis (EAE) Model of Multiple Sclerosis

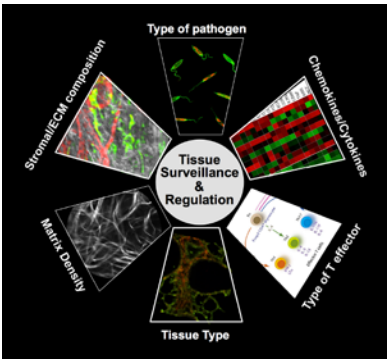
Hanna Vinitsky, Wei Wang, Shane O'Neil, Ben Reeves, Ezra Yang, Steven Goldman, Iben Lundgaard, Maiken Nedergaard

Department of Neurosurgery, University of Rochester, Rochester, NY.

Multiple Sclerosis (MS) is an autoimmune disease targeting myelin in the central nervous system. Lesions in MS patients and in the experimental autoimmune (EAE) model of MS are characterized by immune cell infiltration often forming peri-vascular cuffing around blood vessels. The glymphatic system is a brain-wide clearance system using peri-vascular pathways for transport. Here we used the EAE mouse model of MS and investigated glymphatic function dynamics using in vivo imaging of a fluorescent cerebrospinal fluid (CSF) tracer. We found that glymphatic influx to the brain was reduced and influx to the spinal cord was severely diminished. The distribution of CSF tracer was inversely correlated with the number of lesions, suggesting that EAE tissue pathology affects the glymphatic system in acute and chronic disease. Intriguingly, inhibition of the glymphatic function using acetazolamide and cisterna magna puncture (CMP) significantly ameliorated EAE clinical symptoms. This shows that glymphatic function is affected in EAE but that disease progression might be aided by the glymphatic system in the early phase. This suggests that targeting the glymphatic system in the early phase of MS might be a novel mechanism to curb disease.

4:10 – 4:50 p.m.

DEBORAH FOWELL, Ph.D.
Professor, University of Rochester, Rochester, NY
Programming effector T cells for interstitial motility



RESEARCH INTERESTS

The Fowell lab is interested in the regulation of immunity at tissues sites of inflammation due to infection or environmental damage. Specifically, the lab seeks to understand how tissue location changes function in the CD4 compartment; for Th1, Th2 and Th17 effector cell subsets. We have developed in vivo intravital imaging systems to visualize lymphocyte migration, T:APC interactions and T cell function in situ in the inflamed dermis using intravital multiphoton microscopy. Understanding the context-dependent environmental cues and the T cell intrinsic programming that modulates immune function at sites of inflammation has important therapeutic implications. Novel therapies that focus on manipulating the local infection/inflamed site to encourage appropriate recruitment or activation of effectors may be particularly beneficial.

POSTERS

Presenter(s) listed BOLD

1.	INFLAMMATORY RESPONSES AFTER A LASER-INDUCED CORTICAL MICROHEMORRHAGE **Sung Ji Ahn**[1] , Josef Anrather[2] , Nozomi Nishimura[1] , and Chris B Schaffer[1] *[1]Meinig School of Biomedical Engineering, Cornell University, Ithaca NY 14853 [2]Feil Family Brain and Mind Institute, Weill Cornell Medical College, New York, NY.*
2.	HYPERSPECTRAL MULTIPHOTON MICROSCOPY FOR SIMULTANEOUS IN VIVO VISUALIZATION OF MULTIPLE CELL TYPES **Amanda J. Bares,** Mitchell A. Pender, Menansili A. Majooli, Steven Tilley, Kuang E. Chen, Jingyuan Dong, Nozomi N. Nishimura, and Chris B. Schaffer *Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY.*
3.	STALLED BLOOD FLOW IN BRAIN CAPILLARIES IS RESPONSIBLE FOR REDUCED CORTICAL PERFUSION AND IMPACTS COGNITIVE FUNCTION IN MOUSE MODELS OF ALZHEIMER'S DISEASE [1]**Oliver Bracko***, [1]Jean C. Cruz Hernández*, [1]Calvin Kersbergen, [1]Victorine Muse, [1]Mohammad Haft-Javaherian, [2]Laibaik Park, [1]Iryna Ivasyk, [1]Lindsay Vinarcsik, [1]Yiming Kang, [1]Joan Zhou, [1]Gabriel Otte, [1]Jeffrey D Beverly, [1]Elizabeth Slack, [1]Thom P Santisakultarm, [2]Costantino Iadecola, [1]Nozomi Nishimura[#] and [1]Chris B. Schaffer[#] *[1]Meinig School of Biomedical Engineering, Cornell University, NY. [2]Brain and Mind Research Institute, Weill Cornell Medical College, NY.*
4.	DEVELOPMENT OF THE GLYMPHATIC SYSTEM Anne Sofie Munk[1, 2]*, **Anne Cheng**[1*], Wei Wang[1*], Benjamin Kress[1, 2], Abdellatif Benraiss[1], Maiken Nedergaard[1,2], and Iben Lundgaard[1] *[1]Center for Translational Neuromedicine, Department of Neurosurgery, University of Rochester, Rochester, NY. [2]Center for Basic and Translational Neuroscience, University of Copenhagen, DK. *Authors contributed equally*

5.	RATIOMETRIC IMAGING TO MEASURE PHAGOSOME ESCAPE OF ORAL MICROBES **Andrew Croft** and Jason G. Kay *Department of Oral Biology, University at Buffalo, Buffalo, NY.*
6.	DEVELOPMENT OF VENUS REPORTER VIRUS TO STUDY CELLULAR DYNAMICS BETWEEN ANTIGEN PRESENTING CELLS AND ADAPTIVE IMMUNE CELLS FOLLOWING INFLUENZA VIRUS INFECTION **Anthony DiPiazza**, Nicholas Poulton, Aitor Nogales, Luis Martinez-Sobrido, and Andrea Sant *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*
7.	FIBRONECTIN DEPOSITION MODULATES CD4+ TH1 CELL INTERSTITIAL MIGRATIONDURING INFLAMMATION **Ninoshka R.J. Fernandes**[1] and Deborah J. Fowell [2] *[1]Department of Biomedical Engineering, University of Rochester, Rochester, NY, [2]Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*
8.	LINEAGE-SPECIFIC PROGRAMMING OF EFFECTOR CD4+ T CELL INTERSTITIAL MOTILITY **Alison Gaylo[1]**, Chris Anderson[1], David Topham[1], Alan Smrcka[2], and Deborah Fowell[1] *[1]David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, [2]Department of Pharmacology and Physiology, University of Rochester Medical Center, Rochester, NY.*
9.	SPONTANEOUS SKIN IMMUNE DYSREGULATION IN THE ABSENCE OF WISKOTT-ALDRICH SYNDROME PROTEIN **Katherine Herman[1]**, Takeshi Yoshida[2], Deborah Fowell[1] *[1] David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, [2]Department of Dermatology M&D, University of Rochester Medical Center, Rochester, NY.*

10.	TARGETED CALCIUM INFLUX BOOSTS CYTOTOXIC T LYMPHOCYTE FUNCTION IN THE TUMOR MICROENVIRONMENT **Kyun-Do Kim**[1], Seyeon Bae[1], Tara Capece[1], Hristina Nedelkovska[1], Rafael G. de Rubio[2],Alan V. Smrcka[2], Woojin Jung[3], Byeonghak Park[3], Tae-il Kim[3], and Minsoo Kim[1] *[1]Department of Microbiology and Immunology, David H. Smith Center for Vaccine Biology and Immunology, [2]Department of Pharmacology & Physiology, University of Rochester School of Medicine and Dentistry, Rochester, NY. [3]School of Chemical Engineering, Sungkyunkwan University, Suwon, Center for Neuroscience Imaging Research, Institute of Basic Science, Suwon, Republic of Korea.*
11.	INFLUENZA SPECIFIC CD8 T CELL MOTILITY IS A DYNAMIC PROCESS THAT VARIES WITH THE TIME AFTER INFECTION, AND IS LINKED TO VIRUS REPLICATION KINETICS **Kris Lambert Emo**, Emma Reilly, David Topham *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*
12.	IMMUNOREGULATION OF CD28 COSTIMULATION AND LIGAND BINDING **Scott A. Leddon**[1,2] Kristin Abramo[1], Margaret Fettis[1] and Jim F. Miller[1] *[1] David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, [2]Center for Oral Biology, University of Rochester Medical Center, Rochester, NY.*
13.	POSSIBLE ROLE OF EPIDERMAL GROWTH FACTOR RELEASED FROM APOPTOTIC NEUTROPHILS IN MONOCYTE ACTIVATION **Kihong Lim**, David Topham, and Minsoo Kim *David H. Smith Center for Vaccine Biology and Immunology, Program for Advanced Immune Bioimaging, University of Rochester Medical Center, Rochester, NY.*

14.	VISUALIZING IFN-γ–MEDIATED EFFECTS ON CELLULAR IMMUNITY TO INTRACELLULAR PATHOGENS **Andrew Martin** and Felix Yarovinsky *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*
15.	LYMPH NODE STROMA: THE HIGHWAY TO AGE RELATED IMMUNE IMPAIRMENT **April R. Masters[1]**, Alexxus T. Hall[2], Laura Haynes[1] *[1]University of Connecticut Health Center, Department of Immunology and Center on Aging, Farmington, CT. [2]University of Saint Joseph, Department of Biology, West Hartford, CT.*
16.	CONDITIONAL DEPLETION OF MICROGLIAL CELLS DURING DEVELOPMENT USING THE Cx_3CR1^{CREER}: IDTR SYSTEM **Monique S. Mendes**, and Ania Majewska *Department of Neuroscience, University of Rochester, Rochester, NY.*
17.	SPLENIC MARGINAL ZONE CD169+ MACROPHAGES ORCHESTRATE INNATE IMMUNE RESPONSES TO BACTERIAL INFECTION **Oriana Perez[1]**, Stephen T. Yeung[1], Basak B. Cicek[1], Zhijuan Qiu[1], Pablo Romagnoli1, Alexandre P. Bénéchet[1], Leigh Maher[1], Masato Tanaka[3], Kamal M. Khanna[1,2] *[1] Department of Immunology, UCONN Health, Farmington, CT.* *[2] Department of Pediatrics, UCONN Health, Farmington, CT.* *[3] School of Life Science, Tokyo University of Pharmacy and Life Sciences, Hachioji, Japan.*
18.	DISTINCT T CELL-APC INTERACTION DYNAMICS FOR TH1 AND TH2 CELLS AT THE SITES OF INFLAMMATION **Milan Popovic** and Deborah Fowell *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*

19.	UNDERSTANDING THE ROLES OF TISSUE BINDING INTEGRINS IN THE DEVELOPMENT AND MAINTENANCE OF RESIDENT MEMORY CD8 T CELLS **Emma C Reilly**, Kris Lambert, Aitor Nogales, Anthony Dipiazza, Luis Martínez-Sobrido, Andrea J Sant, and David J Topham *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*
20.	ECM-BINDING INTEGRIN-DEPENDENT CD4+ T CELL POSITIONING TO HELP B CELLS **Dillon Schrock**, Scott Leddon, and Deborah J. Fowell *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester Medical Center, Rochester, NY.*
21.	IMAGING THE ROLE OF CXCR3 AND ITS LIGANDS IN THE INTERACTION OF T CELLS WITH THE BRAIN VASCULATURE IN CEREBRAL MALARIA **Elizabeth W. Sorensen,** Jeffery Lian, Yoshi Miyabe, Aleksandra J. Ozga, Sophia Ji, Akiba Sato, Shannon K. Bromley, Thorsten R. Mempel, and Andrew D. Luster *Center for Immunology & Inflammatory Disease, Massachusetts General Hospital, Harvard Medical School, Boston, MA.*
22.	NORADRENERGIC MODULATION OF MICROGLIAL DYNAMICS AND SYNAPTIC PLASTICITY **Stowell, R.S.**[1], Sipe, G.O.[2], and Majewska, A.K[1]. *[1]Department of Neuroscience, University of Rochester Medical Center, Rochester, NY. [2] MIT Department of Brain and Cognitive Sciences, Cambridge, MA.*
23.	THE TRANSCRIPTION FACTOR ETS1 COOPERATES WITH IL17 SIGNALING TO REGULATE ANTIBACTERIAL SKIN IMMUNE RESPONSES **Alex Sunshine**, Wei Luo, Satrajit Sinha, and Lee Ann Garrett-Sinha. *Department of Biochemistry, University at Buffalo, Buffalo, NY.*

24.	PROTEOLYTIC ENZYME EXPRESSION AND ACTIVITY IS INCREASED IN A Tsc2-NULL MOUSE MODEL FOR LAM – A METASTATIC LUNG DISEASE **Manisha Taya[1]**, Hen Prizant[2], Irina Lerman[2], Allison Light[2], Aritro Sen[2], Soumya Mitra[3], Thomas H. Foster[3] and Stephen R Hammes[2] *[1]Department of Pharmacology and Physiology, [2]Division of Endocrinology and Metabolism, Department of Medicine, [3]Department of Imaging Sciences, University of Rochester Medical Center, Rochester, NY.*
25.	LABEL-FREE DETECTION OF ATHEROSCLEROTIC PLAQUE FORMATION USING THIRD HARMONIC GENERATION MICROSCOPY **Irwin I. Tendler**, David M. Small, Jason S. Jones, Nozomi Nishimura Nancy E. and Peter C. *Meinig School of Biomedical Engineering, Cornell University, Ithaca, NY.*
26.	SEPSIS-INDUCED NEUROINFLAMMATION **Alissa Trzeciak**, Tae-Hyoun Kim, Eric Harrower, and Minsoo Kim *David H. Smith Center for Vaccine Biology and Immunology, Department of Microbiology and Immunology, University of Rochester, Rochester, NY.*
27.	RECIPROCAL EFFECTS OF GLYMPHATIC FUNCTION AND THE EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS (EAE) MODEL OF MULTIPLE SCLEROSIS **Hanna Vinitsky**, Wei Wang, Shane O'Neil, Ben Reeves, Ezra Yang, Steven Goldman, Iben Lundgaard, and Maiken Nedergaard *Department of Neurosurgery, University of Rochester School of Medicine and Dentistry, Rochester, NY.*

28.	SEXUAL DIMORPHISM OF POPLITEAL LYMPH NODE COLLAPSE AS A BIOMARKER OF DISEASE PROGRESSION IN THE TUMOR NECROSIS FACTOR TRANSGENIC MOUSE MODEL OF RHEUMATOID ARTHRITIS **Emily K. Wu[1,2]**, Richard D. Bell[2,3], Christopher A. Rudmann[2], Ronald W. Wood[2,4,5], Christopher T. Ritchlin[6], Homaira Rahimi[2,7], and Edward M. Schwarz[1,2,3,5,6,8] *[1]Department of Microbiology and Immunology, [2]Center for Musculoskeletal Research, [3]Department of Pathology and Laboratory Medicine, [4]Department of Obstetrics and Gynecology, [5]Department of Urology, [6]Department of Medicine, [7]Department of Pediatrics, [8]Department of Orthopedics, University of Rochester School of Medicine and Dentistry, Rochester, NY.*
29.	REFLECTANCE CONFOCAL MICROSCOPY FOR IMMUNE CELL IMAGING IN UNPREPARED AND IN-VIVO TISSUES **James M. Zavislan** *The Institute of Optics, University of Rochester School of Medicine and Dentistry, Rochester, NY.*

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Participating Institutions

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Harvard Medical School, Boston, Massachusetts
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Massachusetts General Hospital, Boston, Massachusetts
Rochester Institute of Technology, Rochester, New York
Rockefeller University, New York, New York
Roswell Park Cancer Institute, Buffalo, New York
State University of New York Upstate Medical University, Syracuse, New York
University at Buffalo, Buffalo, New York
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University of California at Berkeley, Berkeley, California
University of Connecticut Health Center, Mansfield, Connecticut
University of Minnesota, Minneapolis, Minnesota
University of Pennsylvania, Philadelphia, Pennsylvania

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