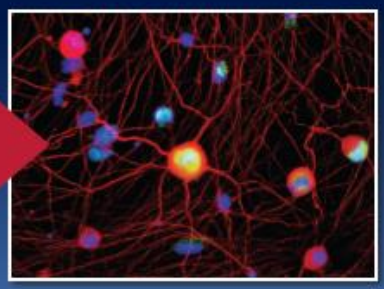

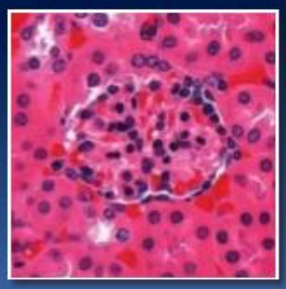

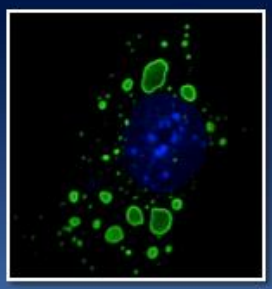

College of Medicine – Tucson
Department of Immunobiology

Twelfth Annual:
Frontiers in Immunobiology & Immunopathogenesis Symposium 2017

Friday March 3rd, 2017

Program and Abstracts

Plenary Speakers

David Leib, Ph.D. *(Virology)*
Dartmouth College

Evgeni Sokurenko, M.D, Ph.D. *(Bacteriology)*
University of Washington

Jonathan Kagan, Ph.D. *(Immunology)*
Harvard Medical School

http://frontiers.immunobiology.arizona.edu/

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Agenda- Frontiers in Immunobiology & Immunopathogenesis Symposium

Morning Activities
BIO5 (Keating Building), 1st Floor Lobby and Room 103

7:45 - 8:15 AM **Registration & Continental Breakfast**

8:20 - 8:40 AM **Welcome Announcements**
Janko Nikolich-Žugich, MD, PhD
Professor and Department Head, Immunobiology
Co-Director, Arizona Center on Aging

Charles Cairns, MD, FACEP, FAHA
Dean, College of Medicine-Tucson
Assistant Vice President, Clinical Research and Clinical Trials
Professor, Emergency Medicine

Jennifer Barton, PhD
Professor, Biomedical Engineering
Interim Director, BIO5 Institute

Session I: Virology
Moderator: Krysta Felix

8:40 - 8:50 AM *Sebastian Zeltzer*
"HCMV: All Roads Lead to the Sorting Endosome"

8:55 - 9:10 AM *Koenraad Van Doorslaer*
BIO5 Institute, and School of Animal & Comparative Biomedical Sciences, University of Arizona, Tucson, Arizona
*"*Use of comparative virology to understand papillomavirus oncogenicity*"*

9:15 - 10:05 AM **Plenary Lecture***, **David Anthony Leib, PhD***
Department of Microbiology and Immunology, Geisel School of Medicine at Dartmouth, Dartmouth-Hitchcock Medical Center, Hanover, New Hampshire
*"*Herpes Simplex Virus and Newton's Third Law"

10:05 - 10:25 AM **BREAK**, *coffee available in Lobby*

Session II: Bacteriology

Moderator: Iris Ma

10:25 - 10:35 AM *Shraddha Tuladhar*
"Determining Toxoplasma Strain-specific CNS immune responses"

10:40 - 10:50 AM *Won Jong Kim*
"The Impact of Autophagy on *Neisseria gonorrhoeae* Infection"

10:55 - 11:10 PM *Michael Johnson*
Department of Immunobiology, University of Arizona, Tucson, Arizona
"Copper operon repressor functions to trigger bacterial "rapid response" system"

11:15 - 12:05 PM **Plenary Lecture**, **Evgeni V. Sokurenko**, **MD, PhD**
Department of Microbiology, University of Washington, Seattle, Washington
"Pandemic of E. coli H30 – "Hannibal Rising" "

Lunch Sessions with Plenary Speakers

Immunology Hot Topics
BIO5 (Keating Building), Room 103
Krysta Felix & Marvin O'Ketch, student moderators

Virology Hot Topics
Medical Research Building, 2nd Floor Lunchroom
Sebastian Zeltzer, student moderator

Bacteriology Hot Topics
Medical Research Building, Room 102
Man Cheong (Iris) Ma, student moderator

Session III: Immunobiology
Moderator: Marvin O'ketch

1:05 - 1:15 PM	Pawel Laniewski, PhD "IL-36γ as a Driver of Mucosal Immune Response against Pathogenic Gonococci in Human 3-D Female Reproductive Tract Models"
1:20 - 1:30 PM	*Heather Thompson, PhD* "Sex Steroid Ablation Restores the Thymus of Aged Animals but Fails to Improve Naïve T Cell Numbers and Function in Peripheral Lymph Nodes"
1:35 - 1:50 PM	*Michael Kuhns, PhD* *Department of Immunobiology, University of Arizona, Tucson, Arizona* *"*Reciprocal TCR-CD3 and CD4 engagement of a nucleating pMHCII stabilizes a functional receptor"
1:50 - 2:10 PM	**BREAK**, *coffee available in Lobby* *Please visit our sponsors for a chance to win an iPad.*
2:10 - 2:20PM	*Deepa R Jamwal, PhD* "TGFβ Signaling in Dendritic Cells is Required for the Maintenance of CD8+CD103+Regulatory T cell Pool"
2:25 - 3:15 PM	**Plenary Lecture*, Jonathan Kagan, PhD*** *Department of Pediatric, Boston Children's Hospital and Harvard Medical School, Boston, Massachusetts* "Initiation of Innate Immunity"
3:20 – 3:25 PM	**Closing Remarks/Acknowledgements**
3:25 – 3:30 PM	**BREAK & Poster Setup** *Please visit our sponsors.*

Evening Activities
Medical Research Building, 1st Floor Lobby and Room 102

3:30 - 5:00 PM	**Poster Session and Reception**
5:10 PM	**Poster Awards & Drawing for iPad**

Plenary Lectures

Herpes Simplex Virus and Newton's Third Law

David Anthony Leib PhD

Department of Microbiology and Immunology, Geisel School of Medicine at Dartmouth, Dartmouth-Hitchcock Medical Center, Hanover, NH

Newton's third law of motion states that "For every action, there is an equal and opposite reaction". The same is true of the pathogenesis of herpesviruses which spend the majority of their lifecycle in a steady, yet active stalemate with their host. Our laboratory studies the intrinsic, innate, and adaptive immune responses to acute and latent herpes simplex virus infection in the nervous system. We also study the mechanisms by which the virus evades and modulates these responses to establish a balance and forge a largely peaceful and lifelong relationship with its host.

Pandemic of E. coli H30 – "Hannibal Rising"

Evgeni V. Sokurenko, MD, PhD

Department of Microbiology, University of Washington, Seattle, WA

*Escherichia coli H*30 is a multidrug-resistant subgroup of the ST131 clonal group of *E. coli* that has emerged in the U.S. and expanded globally in less than two decades. Annually, it is estimated to cause over 1 million urinary tract and bloodstream infections and could be responsible for over 10,000 deaths in the USA alone. Hallmark characteristics of *H*30 include Cipro resistance (*H*30 accounts for a majority of all fluoroquinolone-resistant *E. coli* isolates) and extended-spectrum beta-lactamase (ESBL) production. The clinical significance and pandemic-like expansion of ST131-*H*30 has been the focus of several hundred studies over the past decade. In addition to its antimicrobial resistance phenotype, *H*30 is strongly associated with persistent/recurrent UTI and severe complications, accounting for up to 70% of E. coli sepsis isolates in some patient populations. The economic impact, morbidity, and mortality of infections caused by *H*30 could rival that associated with MRSA. The emergence of this phylogenetically discrete yet actively evolving clonal group provides a unique opportunity by which to study and understand the mechanisms leading to recently emerged bacterial strains that have disseminated globally.

Initiation of Innate Immunity

Jonathan Kagan PhD

Department of Pediatric, Boston Children's Hospital and Harvard Medical School, Boston, MA

The central goal of my research is to understand the earliest events that determine innate immune responses in various multicellular organisms. We aim to create a comprehensive map of the subcellular sites of innate immune signal transduction, and determine how manipulations of early signaling events influence protective immunity. Particular focus is placed on understanding how microbial or self-derived molecules engage pattern recognition receptors, and the functional consequences of this engagement. In this seminar, I will discuss our recent investigations of innate immune signal transduction, with an emphasis on defining how known regulators of signal transduction interact with one another dynamically and functionally to execute effective host defenses.

Invited Lectures

as presented

HCMV: All Roads Lead to the Sorting Endosome

Sebastian Zeltzer, Marco Padillia-Rodriguez, Felicia Goodrum, Julie Donaldson

Cellular and Molecular Medicine, University of Arizona, Tucson Arizona

The ability of a cell to respond and interact with its environment, depends on its concentration of plasma membrane receptors and subsequent endocytic processing events. Here we describe how Human Cytomegalovirus (**HCMV**), a betaherpes virus, alters the ARF6 clathrin independent endocytic (**CIE**) pathway, leading to an aberrant accumulation of internalized surface receptors such as MHCI, CD147, and CD59 in the sorting endosome. Using fluorescent microscopy and live cell imaging, we demonstrate that infection drives the CIE regulator ARF6 to disproportionality interact with the sorting endosome. Further, we demonstrate that expression of a key ARF6 regulator, TRE17, and its ubiquitin specific protease activity, is sufficient to reverse the retention of CIE cargos and decrease ARF6 association with the sorting endosome. Together, this work details a novel means by which HCMV subverts a number of immune receptors by way of dislocation, and provides greater insight into the role ubiquitin protease activity plays in the sorting of endocytic cargos at large.

Use of comparative virology to understand papillomavirus oncogenicity

Robert Burk and Koenraad Van Doorslaer

BIO5 Institute, and School of Animal & Comparative Biomedical Sciences, College of Agriculture & Life Sciences, University of Arizona, Tucson, Arizona

Papillomaviruses establish a persistent infection in the basal epithelia. To complete their life cycle, papillomaviruses need to replicate in terminally differentiated cells. The papillomaviral lifecycle perturbs the normal differentiation cycle of the infected cell, forcing cells to divide far beyond their normal lifespan. It is feasible that the continued insult provided by replicating viruses eventually results in malignant transformation of the infected cell. It is improbable that the ability to cause cancer provides papillomaviruses with an evolutionary advantage. Nonetheless, evolution did select for the viral functions linked to oncogenesis. I hypothesize that the evolution of these viral phenotypes allowed papillomaviruses to adapt to novel environmental niches on the host (e.g. external genitalia vs. cervix). Persistent infection is key to viral oncogenesis; many long-term persisting viruses do not cause cancer. By carefully interrogating the differences between these viruses, I believe it will be possible to elucidate which viral phenotypes are associated with oncogenic progression. As an example, the correlation between HPV E6-induced degradation of a cellular protein and epidemiologically determined HPV oncogenicity was evaluated using a Bayesian statistical approach within a phylogenetic context. Phylogenetic modeling indicates that this phenotype is not specifically correlated with oncogenic risk, but may act as an enabling phenotype. The role of viral phenotypes on HPV fitness and oncogenesis needs to be interpreted in the context of evolution.

Determining *Toxoplasma* strain-specific immune responses

Shraddha Tuladhar[1,2], Yarah Ghotmi[2,3], Apoorva Bhaskara[2], Joseph S. Lagas[2,3], and Anita A. Koshy[1,2,4]

Department of Immunology[1], BIO5 Institute[2], UBRP[3], Department of Neurology[4]; University of Arizona, Tucson, AZ, 85719, USA.

Toxoplasma gondii is an obligate intracellular parasite that infects up to 1/3 of the world's population. Though this persistent infection is asymptomatic in most, in immunocompromised individuals, symptoms of *Toxoplasma* can range from fever to focal neurologic syndromes to death. While the determinants of disease variability are poorly understood, recent human data suggest that the genotype of the infecting *Toxoplasma* strain may influence disease outcomes. Consistent with the human data, *in vitro* data has revealed that strain-specific polymorphic effector proteins injected into host cells can lead to different innate immune responses. Thus, we hypothesize that different *Toxoplasma* strains provoke distinct, strain-specific immune responses, which in turn affect disease severity. To test this hypothesis we compared the CNS immune response of mice infected with either type II or type III parasites, two well characterized, genetically distinct *Toxoplasma* strains. At 3 weeks post infection (wpi), we found that CNS parasite burden was equivalent but type III-infected mice had a more pro-inflammatory CNS immune response as compared to type II-infected mice. Consistent with these findings, our flow cytometry analysis of immune cells isolated from the spleen and brain of 3 wpi mice showed that type III-infected mice had a significantly lower numbers of alternatively activated macrophages (AAMs) and regulatory T cells as compared to type II-infected mice. Combining the prior *in vitro* data with our preliminary data, lead us to propose the following model: early on, type II-infection induces a pro-inflammatory response which gives rise to a compensatory anti-inflammatory response that will ultimately hinder the host from eliminating CNS parasites. Conversely, type III-infection elicits a less inflammatory response early on. This leads to subtle increase in parasite burden, which then drives a more pro-inflammatory response as parasites disseminate to the CNS, resulting in an immune response that more effectively clears CNS parasites. We are currently working on testing this model.

Sex Steroid Ablation Restores the Thymus of Aged Animals but Fails to Improve Naïve T Cell Numbers and Function in Peripheral Lymph Nodes

Thompson, H., ***Uhrlaub, J., Jergovic, M., White, S., Smithey, M., Nikolich-Zugich, J.***

Department of Immunobiology and the Arizona Center on Aging, College of Medicine, University of Arizona, Tucson, AZ 85724

Infections remain amongst the leading causes of morbidity and mortality among the elderly (>65 years of age). Unfortunately, the elderly who are vulnerable to infection also respond poorly to vaccines. Underlying these poor immune responses are age-related changes in the immune and other organ systems. In the T cell lineage, changes with age occur in hematopoietic stem and progenitor cells, thymic stroma, and in the balance between naïve and memory T cells. To assess the power of thymic rejuvenation to address one or more of these changes, we used Degarelix to block sex steroid production in old mice (>18 mths). Consistent with previous reports we found that thymic cellularity was increased to adult levels (<4 mths). Using Rag2pGFP mice we observed a 3-fold increase in recent thymic emigrants in the blood in Degarelix treated old mice compared to untreated old mice, but that increase did not translate into increased naïve T cell numbers within the lymph nodes. To assess the impact of the changes of Degarelix-associated thymic rejuvenation upon functional immunity, we infected treated and untreated mice with the West Nile virus (WNV), which inflicts significant mortality to older humans and mice. When infected at day 42 post Degarelix treatment, treated old mice showed no improvement in survival compared to untreated aged mice in response to West Nile Virus. To examine whether the above lack of immune protection may have been due to recently described trafficking defects in draining lymph nodes, we analyzed stromal and hematopoietic cell subsets from collagenase digested lymph nodes from adult and old mice. We found a 4.2-fold reduction in hematopoietic cells and 2.3-fold reduction in stromal cells in the old lymph nodes. Moreover, within the stromal populations we found a 1.5-fold decrease in fibroblastic reticular cells and a 3.8-fold decrease in lymphatic endothelial cells. These data suggest that Degarelix-associated thymic rejuvenation did not improve peripheral lymph node defects in aging, and that it, alone, may be insufficient to restore protective immunity.

Copper operon repressor functions to trigger bacterial "rapid response" system

Michael Johnson

Department of Immunobiology, College of Medicine, The University of Arizona, Tucson, AZ

Despite being in contact with bacteria since antiquity, copper is broadly toxic to bacteria. Copper toxicity is seen in practice where copper surfaces and tools significantly reduce nosocomial infections. Furthermore, during host mediated nutritional immunity (sequestering essential metals while bombarding bacterial with toxic metals), macrophages kill engulfed targets using copper. To understand how bacteria evolved to interact with toxic metals, which includes why copper is toxic and how bacteria overcome copper stress, we are using *Streptococcus pneumoniae* as our model system. *S. pneumoniae*, causative agent of pneumonia, meningitis, and otitis media, contains a copper export system which consists of an operon repressor, a copper chaperone, and a copper exporter. We have found that when copper enters *S. pneumoniae*, it needs to be the oxidized copper (II) form, but to exit, it must be in the reduced copper (I) form. Thus, *S. pneumoniae* require a constant stream of electrons while under copper stress. The copper operon promotor sequence exists intact before a sugar import system and is upregulated in a microarray of *S. pneumoniae* under copper stress. We have shown that this sugar import system can import both reducing and non-reducing sugars. Reducing sugars, such as glucose, can act to donate electrons such as in Benedicts Test (which uses detection of copper II to copper I to reveal reducing sugars in urine). Mutations to this sugar import system reduce the bacteria's ability to survive copper stress implying that this system could be directly responsible to suppling the electrons needed to properly export copper. This finding suggests that bacterial repressors play multifunctional roles in toxin "rapid response" systems.

IL-36γ as a Driver of Mucosal Immune Response against Pathogenic Gonococci in Human 3-D Female Reproductive Tract Models

Paweł Łaniewski, James Baker, Melissa Herbst-Kralovetz

Department of Basic Medical Sciences, College of Medicine-Phoenix, University of Arizona, Phoenix, AZ

IL-36γ is an immunoregulatory cytokine, which belongs to the IL-1 family. It is expressed by epithelial cells of the skin, respiratory tract and gut. Recently, we have shown that IL-36γ, as well as its receptor (IL-36R) and receptor antagonist (IL-36Ra), are also expressed by epithelium lining the female reproductive tract (FRT) and is differentially induced by microbial products in a TLR-dependent fashion. Gonorrhea, the sexually transmitted disease (STD), which is caused by *Neisseria gonorrhoeae*, remains a major health problem in the United States with nearly 400,000 cases reported annually. The overall objective is to determine how IL-36γ impacts gonococcal infection in the lower FRT. Here we test the hypothesis that IL-36γ promotes and amplifies inflammation at this mucosal site following infection with *N. gonorrhoeae*. To determine the host response to infection, we utilized our well-characterized 3-D bioreactor-derived human vaginal and endocervical epithelial cell models. Differentiated 3-D FRT models were infected with pathogenic *N. gonorrhoeae* or commensal *Lactobacillus crispatus* strains for 24 h at various MOIs. Gene expression in the 3-D cells was determined by quantitative real time PCR and gene expression arrays. Cell supernatants were also collected and analyzed by ELISA and Luminex cytometric bead arrays. IL-36γ expression was significantly increased in both 3-D models following gonococcal infection, but not endogenous vaginal lactobacilli. The response to gonococcal infection was dose-dependent. Secretion of IL-36γ was confirmed in the cell supernatants. However, no differences in IL-36R and IL-36Ra expression were observed following infection with *N. gonorrhoeae*. Production of proinflammatory cytokines, chemokines and antimicrobial peptides, which are also triggered by IL-36γ, were detected following gonococcal infection. The IL-36γ-driven inflammation also impacted epithelial barrier function and integrity. Multiple genes encoding components of tight junctions, gap junctions, focal adhesions were significantly decreased following gonococcal infection. As evidenced by gene array analysis, *N. gonorrhoeae* also altered expression of genes involved in facilitating immune cell migration (*ITGAL*, *ICAM1*) in the same fashion as IL-36γ treatment in absence of infection. In conclusion, we demonstrate that IL-36γ is induced in the lower FRT following infection with *N. gonorrhoeae*, but not commensal vaginal bacteria. In addition, we demonstrate that IL-36γ is an important driver/regulator of mucosal inflammation, which may contribute to disruption of epithelial barrier integrity in the lower FRT.

Unraveling Defects in Stromal Cells and Fibrosis in Aged Lymph Nodes

Thompson, H., ***Jeftic, I., Padilla-Torres, J., Nikolich-Zugich, J.***

Department of Immunobiology and the Arizona Center on Aging, College of Medicine, University of Arizona, Tucson, AZ 85724

Infections remain amongst the leading causes of morbidity and mortality among the elderly (>65 years of age). Unfortunately, the elderly who are vulnerable to infection also respond poorly to vaccines. Underlying these poor immune responses are age-related changes in the immune and other organ systems. In the T cell lineage, changes with age occur in hematopoietic stem and progenitor cells, thymic stroma, and in the balance between naïve and memory T cells. Recently, the role of lymph nodes in maintaining naïve T cells in the periphery has become more appreciated. We hypothesized that stroma cell subsets were dysregulated with age. To understand the role of these cells, we collagenase digested lymph nodes from adult (<4 mth) and old mice (>18 mth). We found a 4.2-fold reduction in hematopoietic cells and 2.3-fold reduction in stromal cells in the old lymph nodes. Within the stromal populations we found a 1.5-fold decrease in fibroblastic reticular cells and a 3.8-fold decrease in lymphatic endothelial cells. In preliminary data, we performed cytokine-chemokine qPCR mini-arrays on whole lymph nodes from old and adult mouse to determine what genes that are dysregulated with age. We found genes upregulated in old lymph nodes were dominated by those involved in fibrosis. These genes included TGF-β superfamily members, TGF-β and Mstn (both increased more than 4-fold in old lymph nodes), as well as Th2 cytokines that are associated with fibrosis, including IL-13, IL-4, and IL-5 (all increased more than 50-fold in old lymph nodes compared to adults). We then hypothesized that old lymph nodes were more fibrotic. To determine if old lymph nodes were more fibrotic we used histochemical stains to characterize fibrosis (picrosirus red and trichrome). Both these stains showed increased staining for fibrosis in old compared to adult lymph nodes. To unravel these defects further, we used heterochronic (adult and old) parabiosis. We found lymph node stromal cells and hematopoietic cells were decreased in the adult compared to adult isochronic (same age) controls. We also saw increased trichrome staining in adult mice that had undergone heterochronic parabiosis. We conclude that aged lymph nodes have both decreased stromal cells, hematopoietic cells, and increased fibrosis. From these results, our current hypothesis is that increased fibrosis blocks normal communication between stromal and hematopoietic cells and that this dysregulation in the lymph nodes contributes to age related declines in T cell function and immune response.

Reciprocal TCR-CD3 and CD4 engagement of a nucleating pMHCII stabilizes a functional receptor macrocomplex

Caleb R. Glassman, Heather L. Parrish, and Michael S. Kuhns

Department of Immunobiology, The University of Arizona College of Medicine, Tucson, AZ 85724, USA.

$CD4^+$ T cells convert the time that T cell receptors (TCRs) interact with peptides embedded within class II major histocompatibility complex molecules (pMHCII) into signals that direct their development, activation, differentiation, and execution of effector functions. It is well established that TCRs relay information to intracellular signaling motifs of the associated CD3 subunits, and broadly accepted that CD4 recruits the kinase Lck to those motifs upon coincident detection of pMHCII. However, the data defining the role of CD4 in T cell activation is often conflicting, and thus the mechanics by which it works with the TCR-CD3 complex to facilitate pMHCII recognition remains enigmatic. In one model the TCR and CD4 bind pMHCII independently in a V-like orientation, while in another CD4 interacts with a composite surface formed by the TCR-CD3 complex bound to pMHCII. Our data show that as the duration of TCR-pMHCII interactions increase, so do CD4 interactions with MHCII. Likewise, CD4 increases TCR confinement to pMHCII. Importantly, this occurs via reciprocal interactions involving membrane distal and proximal CD4 ectodomains. Altogether, the data indicate that a precisely assembled macrocomplex functions to reliably convert TCR-pMHCII confinement into reproducible signals that orchestrate adaptive immunity.

TGFβ Signaling in Dendritic Cells is Required for the Maintenance of $CD8^+CD103^+$ Regulatory T cell Pool

Deepa R Jamwal[1], Rajalakshmy Ramalingam[2], Monica T. Midura-Kiela[1], Fayez K. Ghishan[1], and Pawel R. Kiela[1]

[1]*Department of Pediatrics, College of Medicine, University of Arizona, Tucson, Arizona*
2Roche Tissue Diagnostics, Tucson, Arizona

Dendritic cells (DC) are professional antigen presenting cells that play an instrumental role in shaping immune response or immunological tolerance depending upon the local milieu. Abnormal DC function can directly disrupt immune tolerance resulting in autoinflammatory diseases including Inflammatory Bowel Disease. TGFβ plays a key role in regulating mucosal immune responses to imprint tolerance and facilitate resolution. $Cre^+TGFbR2^{\Delta DC}$ mice, a model that mimics TGFβ resistance in DCs, develop spontaneous multi-organ autoinflammatory phenotype with colitis. However, the exact mechanisms and contribution of $CD4^+$ and $CD8^+$ cells to inflammation remain under investigation. **Aim:** To evaluate the role of TGFβ signaling in DCs on T cell compartments with particular emphasis on recently described immunosuppressive $CD8^+CD103^+$ T cells, capable of suppressing experimental colitis. **Methods:** Cre^- or Cre^+ *Rag1*$^{-/-}$xTGFbR2$^{\Delta DC}$ mice were adoptively transferred with total $CD4^+$, or $CD8^+$, or $CD3^+$ T cells from naïve WT mice and monitored for 8 weeks. Mesenteric lymph nodes (MLNs) and colonic lamina propria (LP) were evaluated for the phenotype of DCs and T cell subsets by flow cytometry. Proliferation of naïve ($CD62L^+CD69^-$) $CD8^+CD103^+$ T cells was tracked in co-culture with DCs from MLNs of Cre^+ or Cre^- mice, or during 96 hr treatment with Concanavlin A (ConA) with different combinations of IL15, IL7 and TGFβ. **Results:** In naïve WT mice, 98% of LP $CD8^+CD103^+$ T cells represented tissue resident memory T cells ($CD62L^-CD69^+$), whereas in MLN 90% of $CD8^+CD103^+$ T cells were naïve ($CD62L^+CD69^-$) consistent with regulatory phenotype. In Cre^+ *Rag1*$^{-/-}$xTGFbR2$^{\Delta DC}$ mice, both $CD4^+$ and $CD8^+$ T cells were indispensable for development of colitis. Contrary to Cre^- recipients, Cre^+ *Rag1*$^{-/-}$ xTGFbR2$^{\Delta DC}$ mice with colitis showed a significant contraction in the MLN population of naïve $CD8^+CD103^+$ T cells. *In vitro*, CD8+CD103+ cells lost CD103 expression within 24 hrs. ConA,IL15/IL7 was sufficient for CD8+CD103- cells to proliferate. However, cells treated with ConA, IL15/IL7 and TGFβ regained CD103 expression by day3. In a co-culture with $F4/80^-CD11c^+MHC\text{-}II^{hi}$ MLN DCs from Cre^- or Cre^+ mice, $CD8^+CD103^+$ T cells sorted from MLNs of naïve mice, proliferating T cells lost the expression of CD103, and regained it in daughter populations only when cultured with control Cre- MLN DC. **Conclusion:** Our data suggests that TGFβ signaling in DCs is required for the maintenance of regulatory pool of $CD8^+CD103^+$ T cells in the MLN, and that their depletion and CD8+ T cell activation may be prerequisite for the development of autoinflammatory disease in mice with DC-specific deletion of TGFbR2.

Notes:

Poster Abstract Titles

Poster #	Author name	Abstract Title
1	Alane Blythe Dy	Genetic Variation in Surfactant Protein A2 Results in Differential Regulation of Eosinophils
2	Brittany L Forte	Human Papillomavirus May Exploit Golgi Vesiculation to Evade the cGAS/STING Pathway During Mitosis-Dependent Infection
3	Carla M Cabral	Defining neuron-Toxoplasma gondii interactions in vivo & over time
4	Chandrasekaran Sambamurthy	Toxoplasma gondii-neuron interactions in vivo: a transciptomics approach
5	Christy A Harrison	Epithelial Na+/H+ exchange promotes homeostasis in the gut microbiome and protects against the development of auto-immune colitis
6	Daniel A Powell	Determining Mechanisms of Protection in a Live Attenuated Coccidiodes Vaccine
7	Deepa R Jamwal	TGFβ Signaling in Dendritic Cells is Required for the Maintenance of CD8+CD103+ Regulatory T cell Pool
8	Heather Linn Thompson	Sex Steroid Ablation Restores the Thymus of Aged Animals but Fails to Improve NaÃ¯ve T Cell Numbers and Function in Peripheral Lymph Nodes
9	James M Baker	Impact of Sex Hormones on IL-36Î³ and Epithelial Barrier Markers in 3-D Human Vaginal and Cervical Epithelial Models
10	Jameson K. Gardner	IL-36gamma creates a HSV-2 resistant environment in 3-D human vaginal epithelial cell and mouse models
11	Jason C Buehler	Sensing the cellular environment for the establishment of HCMV latency
12	Jennifer L Uhrlaub	Influence of immune evasion mechanisms on CD8 T cell responses to poxvirus infection in a vulnerable population
13	Katrina T Lichauco	Transition State as a Possible Model for Inherent Recognition of Peptide:MHC by the TCR
14	Ken Johnson	Cross-presentation of gp100 melanoma antigen
15	Kimberly Young	Sex differences in microglia morphology, complement receptor 3 subunits (CD11b/CD18), and cytokines in the adult healthy brain among male, female, and post-menopause model mice
16	Krysta M Felix	Segmented Filamentous Bacteria Confer Protection Against a Lung Infection Through Innate Mechanisms
17	Liz A Dahlmann	ZIKV Hijacking of Host Lipid Metabolism
18	Man Cheong Ma	A mouse model for Neisseria asymptomatic colonization and persistence
19	Maria A Rendon	Transcriptional regulation of the type IV pilus in Neisseria elongata
20	Mark S Lee	Direct evidence for conformational change in CD3ζζ in the TCR-CD3 Complex
21	Michael S Kuhns	Reciprocal TCR-CD3 and CD4 engagement of a nucleating pMHCII stabilizes a functional receptor macrocomplex
22	Mladen Jergovic	Impaired priming of cytotoxic T cells due to cell-extrinsic factors underlies the age-related vulnerability to infection with intracellular pathogens
23	Nicole E Behrens	HIV and Aging: Viral and Immunological Features Associated with Aging in HIV-Infected Older Patients
24	Oscar A Mendez	Semi-automated quantification of Toxoplasma-CNS host cell interactions
25	Pawel Laniewski	IL-36gamma as a Driver of Mucosal Immune Response against Pathogenic Gonococci in Human 3-D Female Reproductive Tract Models

26	Sebastian L Zeltzer	HCMV: All Roads Lead to the Sorting Endosome
27	Shraddha Tuladhar	Determining Toxoplasma strain-specific CNS immune responses
28	Vivian Nguyen	Developing a Model of Mixed Dementia to Untangle Causality in Aged Wildtype and Transgenic APP Mice
29	Won Jong Kim	The Impact of Autophagy on Neisseria gonorrhoeae Infection

Organizing Committee

Joyce Wu, PhD (chair)
John Purdy, PhD
Krysta Felix
Marvin O'ketch
Man Cheong (Iris) Ma
Jordyn Rippberger
Dragana Nikolich-Žugich
Nicole Swinteck

Further acknowledgement is made for the grateful assistance of:
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Debra Bowles- BioCOM
Margrit McIntosh - BioCOM
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Heather Thompon, PhD
Heather Bronnimann, PhD
Jason Buehler, PhD
Deepa R Jamwal, PhD
Donna Collins-McMillen, PhD

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