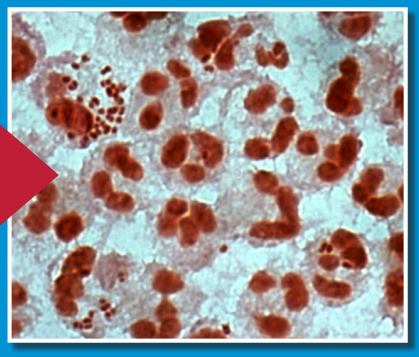
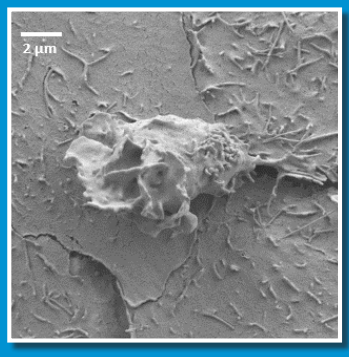
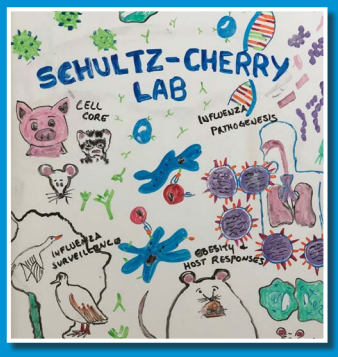






College of Medicine – Tucson
Department of Immunobiology

Thirteenth Annual:

Frontiers in Immunobiology & Immunopathogenesis Symposium 2018

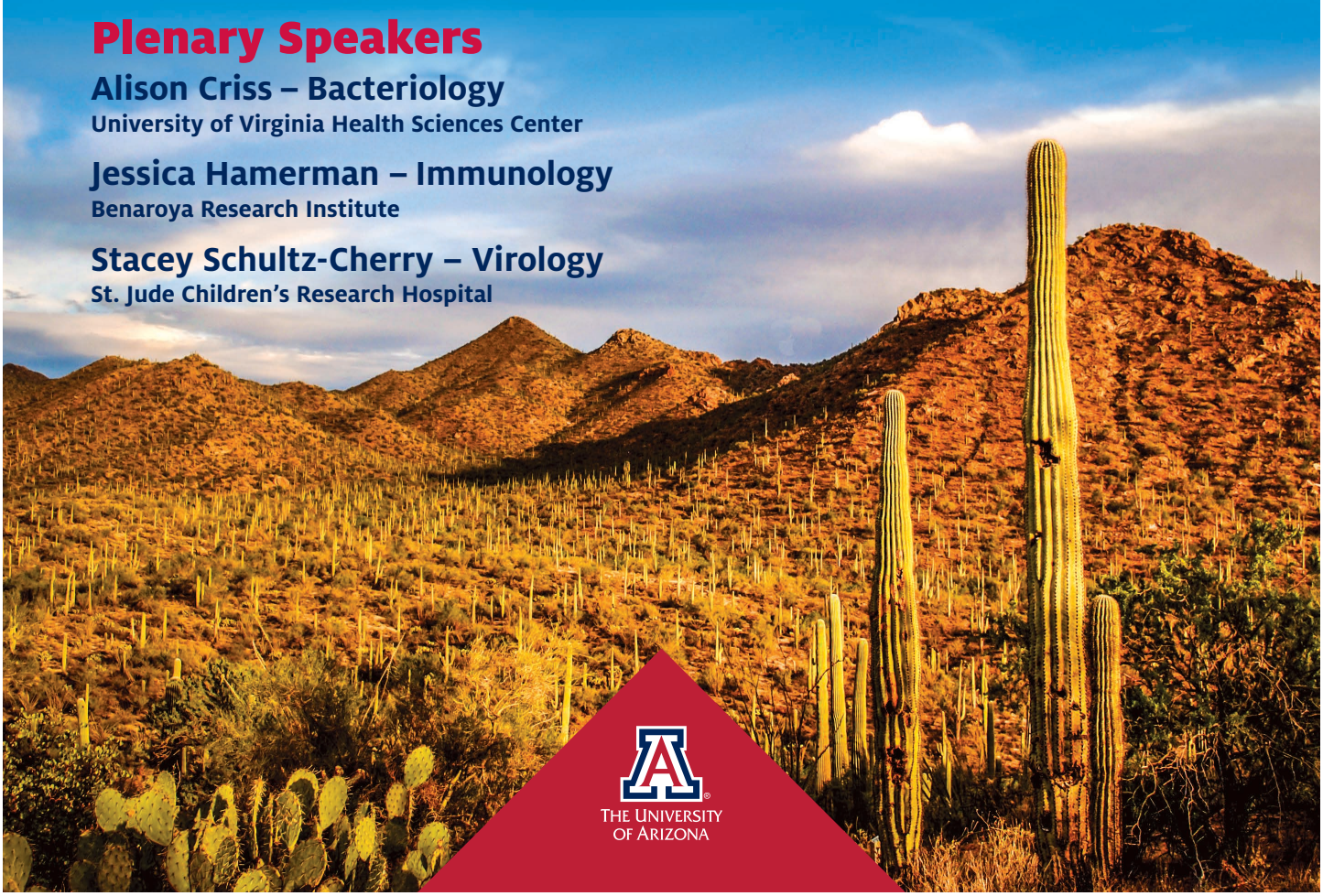
Friday, March 9, 2018

Plenary Speakers

Alison Criss – Bacteriology
University of Virginia Health Sciences Center

Jessica Hamerman – Immunology
Benaroya Research Institute

Stacey Schultz-Cherry – Virology
St. Jude Children's Research Hospital



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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
Professor, Emergency Medicine

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

Session I: Bacteriology
Moderator: Man Cheong (Iris) Ma

8:40 - 8:50 AM *Maria A Rendon, PhD*
"Transcriptional control of the type IV pilus in *Neisseria elongata*"

8:55 - 9:15 AM *Raina M. Maier, PhD*
Interim Director, Institute of the Environment
Department of Soil, Water and Environmental Science, University of Arizona, Tucson, Arizona
"The Earth's Microbiome and Human Health"

9:20 - 10:10 AM **Plenary Lecture**, ***Alison Criss, PhD***
Department of Microbiology, Immunology, and Cancer Biology at University of University of Virginia Health Sciences Center, Charlottesville, Virginia
"Neutrophilic inflammation in the pathogenesis of *Neisseria gonorrhoeae* infection"

10:15 - 10:35 AM **BREAK**, *coffee available in Lobby*
Please visit our sponsors for a chance to win an iPad.

Session II: Virology

Moderator: Jacob Zbesko

10:35 - 10:45 AM *Jameson Gardner*
"Depo-Provera and β-estradiol regulate IL-36γ in the vaginal microenvironment and impact HSV-2 pathogenesis"

10:50 - 11:00 AM *Donna Collins-McMillen, PhD*
*"*Do Novel Immediate Early Promoters Drive Human Cytomegalovirus Reactivation from Latency?"

11:05 - 11:55 PM **Plenary Lecture**, ***Stacey Schultz-Cherry, PhD***
Infectious Diseases Department, St. Jude Children's Research Hospital, Memphis, Tennessee
"Have Swab Will Travel; Looking for Influenza in All the Fun Places"

12:00 - 1:00 PM **Lunch Sessions with Plenary Speakers**

Immunology Hot Topics
BIO5 (Keating Building), Room 103
Nico Contreras, student moderator

Virology Hot Topics
Medical Research Building, Room 102
Jacob Zbesko, student moderator

Bacteriology Hot Topics
Medical Research Building, 2nd Floor Lunchroom
Man Cheong (Iris) Ma, student moderator

Session III: Immunobiology
Moderator: Nico Contreras

1:05 - 1:15 PM *Lydia Meador, PhD*
"Inflammation induces GILT expression in human melanoma"

1:20 - 1:30 PM *Mladen Jergovic, PhD*
*"*Expression of Ly6C defines a subpopulation of naïve CD8 T cells with a rapid effector function which is expanded under 'non SPF' conditions"

1:35 - 1:55 PM *Deepta Bhattacharya, PhD*
Department of Immunobiology, University of Arizona, Tucson, Arizona
*"*Good memories and bad ones of flavivirus infections"

2:00 - 2:20 PM **BREAK**, *coffee available in Lobby*
Please visit our sponsors for a chance to win an iPad.

2:20 - 2:30PM *Krysta Felix*
"P2x7 Purinergic Receptor Expression on T cells Controls Autoimmune Arthritis"

2:35 - 3:25 PM **Plenary Lecture**, ***Jessica Hamerman, PhD***
Benaroya Research Institute at Virginia Mason, Seattle, Washington
"Toll-like receptors, Monocyte Differentiation, and Macrophage Activation Syndrome"

3:30 – 3:35 PM **Closing Remarks/Acknowledgements**

3:35 – 3:40 PM **BREAK & Poster Setup**
Please visit our sponsors.

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

3:40 - 5:00 PM **Poster Session and Reception**

5:10 PM **Poster Awards & Drawing for iPad**

Plenary Lecture

Neutrophilic inflammation in the pathogenesis of *Neisseria gonorrhoeae* infection

Alison Criss, PhD

Department of Microbiology, Immunology, and Cancer Biology at University of Virginia Health Sciences Center, Charlottesville, Virginia

The obligate human bacterial pathogen *Neisseria gonorrhoeae* causes gonorrhea, one of the most common and debilitating diseases worldwide. The clinical hallmark of acute gonorrhea is the recruitment of neutrophils to the site of infection. Despite their abundance and their numerous antimicrobial properties, neutrophils are unable to clear *N. gonorrhoeae* during human infection. Over the past nine years, we have sought to define the mechanisms used by *N. gonorrhoeae* to recruit neutrophils to mucosal sites yet resist killing by them. For these studies, we use *ex vivo* model systems with primary human neutrophils, polarized epithelial cells from tissues that are naturally infected with *N. gonorrhoeae,* and infectious bacteria. Epithelia that are infected apically with *N. gonorrhoeae* coordinate the migration of neutrophils in the physiologically relevant basolateral-to-apical direction, using proinflammatory and chemotactic lipid mediators. These findings open the possibility of modulating the neutrophilic inflammatory response to *N. gonorrhoeae,* a major contributor to the tissue damage and resulting consequences such as infertility, ectopic pregnancy, and pelvic inflammatory disease. Once in a tissue setting, *N. gonorrhoeae* uses two nonexclusive, complementary approaches to survive from neutrophils. First, *N. gonorrhoeae* encodes gene products that defend against neutrophils' antimicrobial products, such as reactive oxygen species, proteases, and antimicrobial peptides. Second, *N. gonorrhoeae* subverts the ability of neutrophils to produce and release these antimicrobial products. Specifically, we found that certain bacterial variants co-opt a "silent" phagocytic pathway in adherent, chemokine-treated primary human neutrophils, which allows *N. gonorrhoeae* to avoid delivery into toxic phagolysosomes and inhibit the oxidative burst. Identifying the bacterial virulence properties and neutrophil signaling pathways used by *N. gonorrhoeae* to survive exposure to neutrophils can reveal new targets to combat gonorrhea, which are desperately needed with rising rates of infection and increasing resistance to antibiotics.

Plenary Lecture

Have Swab Will Travel; Looking for Influenza in All the Fun Places

Stacey Schultz-Cherry, PhD

Infectious Diseases Department, St. Jude Children's Research Hospital, Memphis, Tennessee

Influenza is a leading cause of morbidity and mortality worldwide. Each year as the new flu season starts, we hope that our vaccines will be effective against the currently circulating viruses knowing that we can't always predict what Mother Nature will throw at us.2018 is the perfect time to reflect on this being the 100-year anniversary of the worst influenza pandemic recorded; the 1918 "Spanish flu" outbreak. But where do these novel influenza viruses come from; how do we know what emerging strains pose a risk to humans; how do we protect ourselves against them; and what people may be at an increased risk of acquiring these viruses? My talk will focus on working on influenza at a global public health level from traipsing through the jungles "looking" for emerging influenza viruses to decisions about the strains that go in our vaccines and why we are concerned about the obesity pandemic in regard to influenza disease and prevention.

Plenary Lecture

Toll-like receptors, Monocyte Differentiation, and Macrophage Activation Syndrome

Jessica Hamerman, PhD

Benaroya Research Institute at Virginia Mason, Seattle, Washington

Cytopenias are an important clinical problem associated with acute inflammatory disease and infection. We show that specialized phagocytes that internalize red blood cells develop in TLR7-driven inflammation and during mouse malaria infection, associated with both severe anemia and thrombocytopenia. TLR7 signaling caused development of inflammatory hemophagocytic cells (iHPC) that resemble splenic red pulp macrophages (RPM), but are a distinct population derived from Ly6C^{hi} monocytes independently of the RPM lineage-defining transcription factor Spi-C. These iHPC were responsible for anemia and thrombocytopenia in TLR7-overexpressing mice, which have a severe Macrophage Activation Syndrome (MAS)-like disease. iHPC were also found during blood stage malaria infection in mice, associated with severe anemia and thrombocytopenia. Our findings uncover a novel mechanism by which TLR signaling specifies monocyte fate during inflammation, and identify a new population of phagocytes responsible for anemia associated with inflammation and infection.

Transcriptional control of the type IV pilus in *Neisseria elongate*

***María A Rendón*[1], *Man Ma Cheong*[1], *and Magdalene So*[1]**

[1]*The BIO5 Institute and Department of Immunobiology, University of Arizona, Tucson AZ, 85721*

The type IV pilus (Tfp) is a complex surface structure present on bacteria and archaea. All animal and human species of the *Neisseria* genus produce Tfp. In pathogenic *Neisseria*, Tfp is required for colonization, and it mediates important functions such as host-cell signaling, twitching motility, and DNA uptake. Tfp is a complex machine containing at least 21 proteins, with PilE comprising the main subunit of the fiber.

Commensal and pathogenic *Neisseria* have evolved different mechanisms to regulate expression of *pilE*. In pathogenic *Neisseria*, *pilE* transcription requires sigma factor RpoD, while in commensal *Neisseria* it requires sigma factor RpoN and activator Npa. We have identified the sensor kinase, Nps, which works in conjunction with Npa to regulate *pilE*. An in-frame deletion of *nps* abolished *pilE* transcription, PilE production, and piliation. We determined that Nps and Npa form a two-component system, by introducing point mutations in the activation sites of Nps (H325) and Npa (D58) and observing their effects on PilE expression. Finally, we determined that RpoN and Nps/Npa control transcription only of *pilE*. The other Tfp genes are transcribed from an RpoD promoter.

Why have commensal and pathogenic *Neisseria* evolved two different regulatory mechanisms to control *pilE* expression? It has been shown that when the Tfp machinery malfunctions, this could lead to a buildup of PilE in the membrane or to pili fibers in the periplasm; in both cases the accumulation is toxic to the bacteria. Pathogenic *Neisseria* avoid this toxicity by cleaving PilE into a soluble form that is released into the medium. We speculate that commensal *Neisseria* evolved the RpoN/Nps/Npa regulatory system to sense and control PilE buildup. It is also possible that commensal and pathogenic *Neisseria* require the use of Tfp under different environmental conditions.

Identifying the signal(s) that control *pilE* expression will ultimately allow us to understand how *Neisseria* adapts to its microenvironment, and ultimately identify the timing of Tfp expression and the niche(s) where Tfp expression is required.

The Earth's Microbiome and Human Health

Raina M. Maier, PhD

Interim Director, Institute of the Environment, Department of Soil, Water and Environmental Science
University of Arizona, Tucson, AZ

The Earth's microbiome and the human microbiome are both impacted by everything we do, from mining to burning of fossil fuels to large-scale agriculture. Scientists still barely understand these impacts and in particular, we don't know how the environmental microbiome interacts with the human microbiome to cause or protect from disease. Several examples will be discussed to illustrate the potential consequences of large-scale changes in the Earth's microbiome on human health as well as the challenges in understanding the Earth microbiome.

Depo-Provera and β-estradiol regulate IL-36γ in the vaginal microenvironment and impact HSV-2 pathogenesis

Jameson Gardner[1,2], Thessaly Alexander[1], and Melissa Herbst-Kralovetz[1,3]

[1]Department of Basic Medical Sciences, College of Medicine – Phoenix, University of Arizona, Phoenix, AZ;
[2]Molecular and Cellular Biology Graduate Program, School of Life Sciences, Arizona State University, Tempe, AZ;
[3]Department of Obstetrics and Gynecology, College of Medicine – Phoenix, University of Arizona, Phoenix, AZ

Recently, the widely-used contraceptive Depo-Provera (DMPA) has been linked to an increased risk of sexually transmitted infection acquisition (e.g. HSV-2 and HIV). However, a fundamental gap exists in the understanding of specific mechanisms whereby DMPA use increases risk for HSV-2 infection. DMPA is a synthetic progestin that induces a long-lasting progesterone dominant environment in the female reproductive tract (FRT) that may repress host defense mechanisms. IL-36γ is a novel regulator of mucosal inflammation in the FRT, and we have shown that IL-36γ is induced in response to HSV-2 infection. A recent study reported that IL-36γ was the only cytokine upregulated in cervicovaginal secretions from HSV-2 seropositive DMPA users, suggesting that IL-36γ may be a link between DMPA use and HSV-2 infection. We hypothesize that DMPA suppresses IL-36γ expression in the vaginal environment, thereby increasing susceptibility to genital HSV-2 infection. We found that DMPA treatment in mice decreased expression of all IL-36 family members (α, -β, -γ, -Ra, and –R) in the lower FRT, while β-estradiol treatment increased expression. The IL-36 cytokines were also expressed in the upper FRT, albeit at lower levels relative to the lower FRT, suggesting that the IL-36 cytokines play an important role in host defense mechanisms in the lower FRT. To evaluate the impact of IL-36γ on HSV-2 pathogenesis, we used an intravaginal challenge model and treated mice with DMPA to enhance susceptibility to infection. Mice treated with IL-36γ 4h prior to lethal challenge exhibited a delay in disease onset, less severe disease, decreased vaginal viral replication, increased disease resolution and overall survival. IL-36γ treatment increased levels of pro-inflammatory cytokines in vaginal tissue and lavages 4h after treatment, and neutrophil recruitment was enhanced and corresponded with increased levels of chemokines. Together, we demonstrate that IL-36γ is hormonally regulated in the FRT and that treatment with IL-36γ promotes the transient induction of immune mediators and neutrophil recruitment in the vaginal microenvironment that protected against genital HSV-2 disease and increased viral clearance after lethal challenge.

Do Novel Immediate Early Promoters Drive Human Cytomegalovirus Reactivation from Latency?

Donna Collins-McMillen[1], Mike Rak[2], Nat Moorman[3], and Felicia Goodrum[1,2,4]

[1]BIO5 Institute, University of Arizona, Tucson, Arizona; [2]Department of Cellular & Molecular Medicine, University of Arizona, Tucson, Arizona; [3]Department of Microbiology & Immunology, Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina; [4]Department of Immunobiology, Department of Molecular and Cellular Biology, University of Arizona Cancer Center, University of Arizona, Tucson, Arizona

Reactivation of human cytomegalovirus (HCMV) from its latent state causes severe disease in immunocompromised individuals. In addition, mounting evidence suggests that the cost of persistent HCMV infection is high, even for healthy individuals, as HCMV is becoming associated with age-related pathologies such as cardiovascular disease and immune dysfunction. There is a critical need to determine the molecular basis for HCMV latency so that treatment strategies targeting the latent infection can be developed. Following HCMV infection, crucial, but poorly defined, checkpoints govern the cascade of viral gene expression, the progression of the infectious cycle, and the transition between lytic replication and latency. The HCMV immediate early (IE) protein IE2-86-kDa (IE2) arises from activation of the canonical major immediate early promoter (MIEP) and is critical for the initiation and progression of the replicative cycle in fibroblasts. Latent infection of undifferentiated hematopoietic cells is also marked by episodes of low-level IE gene expression, although these typically fail to result in reactivation and the production of viral progeny. Further, despite low level IE gene expression detected in these cells, the MIEP is known to be silent in latency-associated hematopoietic cell types. Together, these findings indicate that additional viral and/or cellular checkpoints, apart from the initial transcription of IE genes from the MIEP, must be negotiated to trigger lytic reactivation and productive replication in latency-associated cell types. We have identified two novel viral promoters in the MIE locus (referred to as intronic promoters or iPs). These promoters drive a second wave of IE2 expression during HCMV infection of fibroblasts, but appear to be dispensable for viral replication in this cell type. In contrast, we show that these iPs (and not the MIEP) activate IE genes in latency-associated hematopoietic cells, offering a potential explanation for how reactivation can be triggered in cell types where the MIEP is silent. We propose that the iPs function as a regulatory switch that allows HCMV to transition between replicative and latent states by providing a more stringent means of controlling IE2 expression in response to cellular cues.

Inflammation induces GILT expression in human melanoma

Lydia R. Meador[1,2], Hari Menon[1], Haiyan Cui[2], Denise J. Roe[2,3], David J. DiCaudo[4], and Karen Taraszka Hastings (pubmed index as Hastings KT)[1,2]

[1]University of Arizona College of Medicine, Phoenix, AZ, United States; [2]University of Arizona Cancer Center, University of Arizona, Tucson, AZ, United States; [3]Mel and Enid Zuckerman College of Public Health, University of Arizona, Tucson, AZ, United States; [4]Mayo Clinic, Scottsdale, AZ, United States.

T cell-mediated immunity can produce durable anti-melanoma responses resulting in improved survival of metastatic melanoma patients. Gamma-interferon-inducible lysosomal thiol reductase (GILT) is critical for MHC class II-restricted presentation of multiple melanoma antigens to CD4+ T cells and facilitates cross-presentation on MHC class I for activation of CD8+ T cells. Our prior study found that GILT expression is increased in melanocytes of malignant melanoma specimens compared with benign nevi. To determine whether GILT expression is associated with inflammation, expression in halo nevi specimens was compared to nevi without lymphocytic infiltrate by immunohistochemistry. A halo nevus is a benign nevus with a lymphocytic infiltrate which leads to regression of the nevus. GILT, but not MHC class II, expression was increased in melanocytes of halo nevi compared to nevi without lymphocytic infiltrate. Analysis of a publicly available gene expression profiling cohort of 457 cutaneous melanoma specimens revealed that GILT expression was associated with IFN-γ, TNF-α, and IL-1β expression in human melanoma. In vitro exposure of human melanoma cell lines to IFN-γ, TNF-α, or IL-1β induced GILT expression in melanoma cell lines, which lacked GILT expression at baseline. Vemurafenib, a BRAF inhibitor used in the treatment of metastatic melanoma, enhanced IFN-γ-induced GILT and MHC class II expression in a melanoma cell line. Together these data demonstrate that inflammation, alone or in combination with a current therapeutic agent, induces high levels of GILT expression in human melanoma.

Expression of Ly6C defines a subpopulation of naïve CD8 T cells with a rapid effector function which is expanded under 'non SPF' conditions

Mladen Jergovic[1], Jennifer Uhrlaub[1], Heather Thompson[1], Megan Smithey[1], and Janko Nikolich-Žugich[1]

[1]*Department of Immunobiology and the University of Arizona Center on Aging, University of Arizona College of Medicine – Tucson, Arizona, USA*

The composition of the peripheral naïve T cell pool in humans and rodents is governed by complex homeostatic mechanisms. Insightful rodent studies have elucidated mechanisms of peripheral T cell homeostasis and identified stimulation by homeostatic cytokine IL-7 and T cell receptor (TCR) engagement by self-peptide– MHC complexes as key signals for naïve T cell maintenance. However, all of these studies have been performed on animals housed under specific pathogen free (SPF) conditions, in almost complete absence of infection and inflammation. Here we investigate the peripheral naïve CD8 T cell pool in adult C57BL/6 mice cohoused with pet shop mice hosting a variety of infections. We show a novel subpopulation of naïve T cells with rapid effector function characterized by expression of a single memory marker Ly6C and expanded under inflammatory conditions during 'non SPF' housing or acute viral (West Nile Virus) and bacterial (Listeria monocytogenes) infection. We show that the extended phenotype of these Ly6C positive naïve cells is $Eomes^{hi}BCL\text{-}2^{hi}CD5^{hi}SCA1^{hi}$. Even more surprisingly we show long lasting increase in number of naïve CD8 T cells in the lymph nodes of 'non SPF' mice due to expansion of the Ly6C positive subpopulation. We are currently in the process of aging mice under 'non SPF' conditions and we will present results on the role of inflammation in homeostasis of naïve T cells relative to the age of mice.

Good memories and bad ones of flavivirus infections.

Rachel Wong[1], Justin Richner[2], Haiyan Zhao[2], Mark J. Shlomchik[3], Michael S. Diamond[2], Daved H. Fremont[2], and Deepta Bhattacharya[1]

[1]Department of Immunobiology, College of Medicine, University of Arizona, Tucson, AZ; [2]Department of Pathology and Immunology, Washington University in St. Louis School of Medicine, St. Louis, MO; [3]Department of Immunology, University of Pittsburgh School of Medicine, Pittsburgh, PA

Members of the flaviviridae family include a number of globally relevant pathogens such as West Nile (WNV), Japanese Encephalitis (JEV), Dengue, and Zika viruses. Upon clearance of a primary flavivirus infection, both neutralizing and non-neutralizing antibody specificities are generated and maintained. After subsequent infection with heterologous flaviviruses, these antibodies can play either protective or pathogenic roles by blocking or enhancing infection. To define the cells capable of responding to secondary heterologous infections, we utilized a WNV and subsequent JEV vaccination model. Using tetramer reagents directed at the potently neutralizing lateral ridge epitope on envelope protein domain III (DIII) of WNV or JEV, we found that memory B cells were more antigenically diverse and cross-reactive than were long-lived plasma cells. To determine how this memory B cell diversity is generated and subsequently utilized, we generated a novel mouse model in which deletion of activation induced cytidine deaminase (AID) is temporally driven by hCD20-CreERT2. Deleting AID during an ongoing germinal center reaction against WNV did not affect the antigen specificity or quantity of memory B cells or bone marrow plasma cells. Thus, a BCR affinity threshold is not responsible for excluding cross-reactive cells from the bone marrow plasma cell compartment. We next examined whether this diverse memory B cell pool following WNV vaccination was dependent on additional affinity maturation in a recall response against JEV. Deletion of AID, and therefore additional affinity maturation, during the memory phase following WNV vaccination did not alter the cellular response to JEV compared to wild type mice. Thus, cross-reactive B cells selectively enter the memory B cell compartment and are excluded from the long-lived plasma cell compartment through a B cell receptor affinity-independent mechanism. This diverse memory B cell pool can subsequently respond to a heterologous challenge without additional affinity maturation.

P2x7 Purinergic Receptor Expression on T cells Controls Autoimmune Arthritis

Krysta Felix*[*,1], Fei Teng[*,1], Nhan Tran[1], Ivan Jaimez[1], Heqing Ma[1], and Hsin-Jung Joyce Wu[1,2]

[*]These authors contributed equally to this work, [1]Department of Immunobiology, College of Medicine, the University of Arizona, Tucson, AZ; [2]Arizona Arthritis Center, College of Medicine, the University of Arizona, Tucson, AZ

Arthritis affects about 1% of the population, including an estimated 54.4 million people in the U.S. Genetics account for about 12-15% of the risk of developing arthritis, but other risk factors including age, gender, smoking, and obesity also play a large role. In addition, recent evidence supports the idea that the gut microbiota can influence the development of autoimmune arthritis. One of the ways that crosstalk between the microbiota and the host can occur is through a purinergic receptor called P2x7, which senses extracellular ATP. P2x7 can also mediate the response to other danger signals like serum amyloid A (SAA), which is upregulated in the presence of some microbes, including Segmented Filamentous Bacteria (SFB). We investigated the role of P2x7 in the development of arthritis through crosstalk with the gut microbiota, using a mouse model of spontaneous autoimmune arthritis, the K/BxN model. In the absence of P2x7, we found that the mice developed more severe disease, as measured by change in ankle thickness, which was further worsened by colonization with SFB, a commensal species we have previously shown to increase autoimmune arthritis in K/BxN mice. To determine the cellular requirement for P2x7, we transferred either B or T cells lacking P2x7 to mice lacking these cells. We found that P2x7 deficiency in B cells made no impact on arthritis development, while P2x7 deficiency in T cells contributed to worsened disease. P2x7 deficiency results in increased Tfh cell numbers in the Peyer's Patches (PPs) due to decreased apoptosis, which has been shown previously. We additionally found that decreased apoptosis results in increased Tfh cells in the spleens in mice receiving P2x7 deficient T cells, which may further contribute to the development of arthritis. Furthermore, we found that TIGIT, a receptor that has been associated with T cell exhaustion, was upregulated on P2x7 deficient Tfh cells, but not on non-Tfh cells, in both the PPs and the spleen. TIGIT+ cells proliferated more than TIGIT- cells. Overall, these data suggest that T cell-intrinsic P2x7 plays an important role in the control of autoimmune arthritis, potentially by modulating TIGIT levels on Tfh cells.

Poster Titles

Poster	Author	Abstract Title
1	Amanda Chung	Liquefaction of the brain following stroke shares multiple characteristics with atherosclerosis and mediates secondary neurodegeneration in an osteopontin dependent mechanism
2	Brittany L Forte	Evasion of the cGAS/STING Pathway by Human Papillomavirus During Mitosis-Dependent Infection
3	Deepa R Jamwal	Intestinal Epithelial MHCII expression modulates the course of autoimmune and infectious colitis in a mouse model of conditional I-AB knockout
4	Rachel Wong	Good memories and bad ones of flavivirus infections.
5	Donna K. Collins-McMillen	Do Novel Immediate Early Promoters Drive Human Cytomegalovirus Reactivation from Latency?
6	Ellen M Wilkinson	Vaginal biogenic amines alter inflammatory mediators and mucin production but do not alter vaginal epithelial cell viability or morphology
7	Geetanjali Gupta	Resident Memory CD8+ T (CD8+ TRM) Cells for Adoptive Cell Therapy
8	Jameson Gardner	Depo-Provera and β-estradiol regulate IL-36γ in the vaginal microenvironment and impact HSV-2 pathogenesis
9	Jason Buehler	Diminishment of EGF Receptor and Downstream Signaling as CMV Replication Progresses
10	Jennifer Uhrlaub	Cell-specific inhibition of SMAD2/3 restores lymph node cellularity and germinal center function in aged mice responding to acute chikungunya virus (CHIKV) infection.
11	Joshua Kochanowsky	Role of Rop16 in Toxoplasma Encystment
12	Katrina T Lichauco	A Possible Model for Inherent Recognition of Peptide:MHC by the TCR
13	Kevin Ferro	Reactive oxygen species and immune memory: the role of superoxide dismutases for immune priming in the red flour beetle, Tribolium castaneum
14	Krysta Felix	P2x7 Purinergic Receptor Expression on T cells Controls Autoimmune Arthritis
15	Liz A Dahlmann	Sterol O-Acyltransferase Activity is Required for Enveloped Virus Infection
16	Lydia R Meador	Inflammation induces GILT expression in human melanoma
17	Man Cheong Ma	Function of polysaccharide capsule in commensal Neisseria
18	Maria A Rendon	Transcriptional control of the type IV pilus in Neisseria elongata
19	Masahiro Ito	Investigating lactobacilli species-specific differences and impact on immune barrier properties of the human vaginal epithelia
20	Megan S Molina	Bendamustine Conditioning Induces Phenotypic and Functional Changes in Host Antigen-Presenting Cells Which May Confer Protection Against GvHD
21	Mladen Jergovic	Expression of Ly6C defines a subpopulation of naïve CD8 T cells with a rapid effector function which is expanded under 'non SPF' conditions
22	Nicole E Bradley	Reduction in Terminally Differentiated T cells in Virologically Controlled HIV-Infected Older Patients
23	Pawel Laniewski	Functional Impact of Sneathia amnii on the Female Reproductive Tract Epithelium
24	Sebastian L Zeltzer	HCMV Alters Sorting Fate of Internalized Receptors
25	Thessaly Alexander	Impact of IL-36γ Treatment on HSV-2 Replication in the Female Reproductive Tract
26	Vanessa Figliuolo da Paz	Downregulation of Disabled homolog 2 (Dab2) expression by microbial components in dendritic cells in inflammatory bowel disease contributes to dendritic cells function and intestinal inflammation
27	Victoria Gershuny	Mathematical models for expansion of the adaptive immune system on re-exposure to antigen
28	Wonjong Kim	Killing of Neisseria gonorrhoeae by Neisseria DNA

Organizing Committee

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Katherine Rhodes, PhD

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