



College of Medicine – Tucson
Department of Immunobiology

Fifteenth Annual **Frontiers in Immunobiology & Immunopathogenesis Symposium**

Plenary Speakers

Jennifer L. Gommerman, Ph.D.
University of Toronto

*Understanding Multiple Sclerosis Progression using
Translational and Reverse-translational Approaches*

Andreas J. Bäuml, Ph.D.
UC Davis School of Medicine

*Gastrointestinal Host-Pathogen Interaction in the
Age of Microbiome Research*

Ian J. Mohr, Ph.D.

New York University Langone Health

*Control of Innate Immunity by rRNA Accumulation & RNA
Modification by N⁶-adenosine Methylation Enzymes*

SPONSORS

Gold

ThermoFisher
S C I E N T I F I C

Anonymous

Silver



Scientists Helping Scientists™ | WWW.STEMCELL.COM

Agenda - Frontiers in Immunobiology & Immunopathogenesis Symposium

Morning Activities

BIO5 Institute, Room 103

- 7:45 - 8:15 AM** **Registration & Continental Breakfast**
- 8:20 - 8:40 AM** **Welcome Announcements**
Janko Nikolich-Zugich, MD, PhD
Professor and Department Head, Immunobiology
Co-Director, Arizona Center on Aging
- Jennifer Barton, PhD*
Director, BIO5 Institute
- Michael Abecassis, MD, MBA*
Dean, University of Arizona College of Medicine-Tucson

Session I: Virology

Moderator: Yuecheng Xi

- 8:40 - 8:50 AM** *Robert Jackson, PhD*
“Attenuation of Cytosolic DNA Sensing by Human Papillomaviruses in Keratinocytes”
- 8:55 - 9:05 AM** *Yanmei Hu, MS*
“Discovery of Influenza polymerase PA-PB1 Interaction Inhibitors using an *in vitro* Split-Luciferase Complementation-based Assay”
- 9:10 - 9:20 AM** *Sebastian Zeltzer, PhD*
“Human Cytomegalovirus Viral Latency Protein pUL138 Activates WDR48-USP1 Signaling Axis to Suppress Fanconi Anemia DNA Repair Pathway”
- 9:25 - 10:15 AM** **Plenary Lecture, Ian J. Mohr, PhD**
New York University Langone Health
“Control of Innate Immunity by rRNA Accumulation & RNA Modification by N⁶-adenosine Methylation Enzymes”

10:20 - 10:40 AM **BREAK**, coffee available in Lobby
Please visit our sponsors for a chance to win an iPad

Session II: Immunology

Moderator: Megan S. Molina

10:40 - 10:50 AM *Jennifer Uhrlaub, MS*
“Chronic *Toxoplasma gondii* Infection Blunts the Immune Response to West Nile Virus Increasing Susceptibility”

10:55 - 11:05 AM *Grace Niemi, PhD*
“The Effects of Systemic β -adrenergic Receptor Activation on Immune Cell Mobilization and Cytotoxicity *in vitro* and *in vivo*”

Plenary Lecture, Jennifer L. Gommerman, PhD - Canceled

11:10 – 11:30 AM *Karen Taraszka Hastings, MD, PhD*
“Neoantigen fitness model predicts lower immune recognition of cutaneous squamous cell carcinomas than actinic keratoses”

11:35 – 11:55 AM *Kristian Doyle, PhD*
“The pathophysiology of chronic stroke infarcts: What happens after brain tissue dies?”

12:00 - 1:05 PM **Lunch Sessions with Plenary Speakers**

Immunology Hot Topics

BIO5 Institute, Room 103

Megan S. Molina, student moderator

Virology Hot Topics

MRB, 2nd floor Lunchroom

Yuecheng Xi, student moderator

Bacteriology Hot Topics

MRB, Room 102

Josh Kochanowsky, student moderator

1:10 - 1:30 PM *Al Bothwell, PhD*
“Regulation of Intestinal Tumorigenesis and Metastasis by Wnt Ligands”

Session III: Bacteriology & Parasitology

Moderator: Shuaizhi Li

- | | |
|-----------------------|---|
| 1:35 - 1:55 PM | <i>Anita Koshy, MD</i> “Transcriptional Insights into <i>Toxoplasma gondii</i> -injected Neurons” |
| 2:00 - 2:20 PM | BREAK , coffee available in Lobby <i>Please visit our sponsors for a chance to win an iPad.</i> |
| 2:20 - 2:30 PM | <i>Jennifer Lising Roxas, PhD</i> “Enteropathogenic <i>Escherichia coli</i> Dynamically Regulates Host Mitochondrial Morphology and Function” |
| 2:35 - 3:25 PM | Plenary Lecture, Andreas J. Bäumler, PhD <i>UC Davis School of Medicine</i> “Gastrointestinal Host-Pathogen Interaction in the Age of Microbiome Research” |
| 3:30 – 3:35 PM | Closing Remarks/Acknowledgements |

Afternoon Activities

MRB, Lobby and Room 102

- | | |
|-----------------------|--|
| 3:35 – 3:40 PM | BREAK & Poster Setup <i>Please visit our sponsors.</i> |
| 3:40 - 5:00 PM | Poster Session and Reception |
| 5:10 PM | Poster Awards & Drawing for iPad |

The poster session is sponsored by:

ThermoFisher
S C I E N T I F I C

Plenary Lecture

Control of Innate Immunity by rRNA Accumulation & RNA Modification by N⁶-adenosine Methylation Enzymes

Ian J. Mohr, PhD

New York University Langone Health, New York, New York

Unlike viruses that globally suppress cellular protein synthesis to antagonize innate defenses, host gene expression proceeds during human cytomegalovirus (HCMV) reproduction. Among the numerous cellular genes whose expression is stimulated upon HCMV infection are those involved in ribosome biogenesis and the chemical modification of RNA by N⁶-adenosine methylation. Studies investigating how these host processes impact innate immune responses to control HCMV productive replication will be presented.

Plenary Lecture

Understanding Multiple Sclerosis Progression Using Translational and Reverse-Translational Approaches

Jennifer L. Gommerman, PhD

University of Toronto, Toronto, Ontario, Canada

Evidence from animal models of Multiple Sclerosis (MS) as well as genome-wide association studies and clinical trials in relapsing-remitting MS all point to lymphocytes as being critical mediators of MS pathogenesis. Indeed activated myelin-reactive T lymphocytes are sufficient to passively transfer Experimental Autoimmune Encephalomyelitis (EAE) to naïve mice. We have previously shown that pathogenic encephalogenic T lymphocytes populate the central nervous system (CNS), particularly the sub-arachnoid space of the leptomeninges, during passive EAE. Herein we propose a method for modeling the progressive phase of MS in mice, and we examine how some aspects of this meningeal-resident inflammatory process is recapitulated in human MS tissue. Moreover, taking cues from recent clinical trial data, we find that there are nuanced roles for B lymphocytes in MS/EAE, and that at least some of the B cells that enter the CNS during episodes of neuroinflammation show evidence of microbiota-reactivity. Collectively, our results shed new light on pro- and anti-inflammatory roles for lymphocytes in MS/EAE, and underscore the role of the microbiota in shaping neuroinflammatory processes.

Plenary Lecture

Gastrointestinal Host-Pathogen Interaction in the Age of Microbiome Research

Andreas J. Bäuml, PhD

University of California Davis, School of Medicine, Davis, California

The microbiota is linked to human health by governing susceptibility to infection with enteric pathogens. However, the interplay between the pathogen, the host and its microbiota is highly complex, including host cell manipulation by virulence factors, mucosal immune responses and a diverse gut ecosystem. We discuss a pluralistic approach aimed at integrating viewpoints from different disciplines into a conceptual framework to capture the complexity of bacterial pathogenesis in the age of microbiome research. This approach suggests that the host represents a foundation species that uses its immune system as a habitat filter to shape the gut microbiota. In turn, the gut microbiota protects against ecosystem invasion by opportunistic pathogens through priority effects that are based on niche modification or niche preemption. Frank pathogens can overcome these priority effects by using their virulence factors to manipulate host-derived habitat filters, thereby constructing new nutrient-niches in the intestinal lumen that support ecosystem invasion. The emerging picture identifies pathogens as ecosystem engineers and suggests that virulence factors are useful tools for identifying host-derived habitat filters that balance the microbiota.

Attenuation of Cytosolic DNA Sensing by Human Papillomaviruses in Keratinocytes**Robert Jackson¹, Brittany L. Uhlorn², Kelly M. King¹, Jana Jandova¹, Koenraad Van Doorslaer^{1,2,3,4}, and Samuel K. Campos^{2,3,5}**¹*School of Animal & Comparative Biomedical Sciences*, ²*Cancer Biology Graduate Interdisciplinary Program*,
³*Department of Immunobiology*, ⁴*BIO5 Institute*, University of Arizona, Tucson, AZ

Papillomaviruses are a diverse family of double-stranded DNA viruses that infect epithelial tissues. Persistent infection with a subset of those in the Alphapapillomavirus genus are oncogenic in humans. While their compact genomes encode multi-functional gene products required for a productive viral life cycle, the tumorigenesis phenomenon is largely mediated by potent oncoproteins E6 and E7. Due to their interactions with host cellular pathways, human papillomaviruses (HPVs), such as type 16 and 18, can persist and cause anogenital (e.g., cervical) and a growing number of oropharyngeal (e.g., throat) cancers. We study the role of host innate immune pathways and the viral strategies that evolved to subvert them. Epithelial cells are armed to detect damage/pathogen-associated molecular patterns (DAMPs/PAMPs) with the innate nucleic acid sensor cGAS, which binds cytosolic DNA, synthesizes the second messenger cGAMP, leading to activation of STING, then transcription factors IRF3 and NFκB, and ultimately an anti-viral interferon response. To determine how HPV18 eludes the cGAS/STING pathway we generated two independent HPV18+ clones in primary human foreskin keratinocytes, each maintaining episomal viral DNA at low or high levels. We investigated their responses to stimulation upstream (plasmid DNA transfection) or downstream (exogenous cGAMP treatment) of cGAS. We find that HPV18 suppresses STING/IRF3 activation and cGAMP production upon exogenous DNA transfection, in a viral expression dependent-manner. However, cGAMP activated IRF3 in HPV18+ cells, therefore we hypothesize that HPV18 evades cytosolic DNA sensing, primarily by attenuating the pathway upstream of STING, at the level of cGAS. We used RNA-Seq to further explore how HPV18 impacts host innate immunity at baseline and in response to time-course introduction of exogenous DNA or cGAMP. Differential expression and pathway analysis revealed immune suppressive signatures that differed between the viral clones and due to treatments, indicating a strong relationship between viral expression and immune responsiveness. Next, we are determining the mechanisms of HPV-mediated cGAS attenuation to advance our understanding of how antagonism of cGAS/STING may enable viral persistence.

**Discovery of Influenza Polymerase PA-PB1 Interaction Inhibitors using an
in vitro Split-Luciferase Complementation-based Assay**

Yanmei Hu¹, Jiantao Zhang¹, Nan Wu¹, and Jun Wang¹

¹*Department of Pharmacology and Toxicology, College of Pharmacy, University of Arizona, Tucson, AZ*

The limited therapeutic options and increasing drug-resistance call for next-generation of influenza antivirals. Due to the essential function in viral replication and high sequence conservation among influenza viruses, influenza polymerase PA-PB1 protein-protein interaction becomes an attractive drug target. Here, we developed an *in vitro* split luciferase complementation-based assay to speed up screening of PA-PB1 interaction inhibitors. By screening 10,000 compounds, we identified two PA-PB1 interaction inhibitors, R160792 and R151785, with potent and broad-spectrum antiviral activity against a panel of influenza A and B viruses, including amantadine-, oseltamivir-, or dual resistant strains. Further mechanistic study reveals that R151785 inhibits PA nuclear localization, reduces the levels of viral RNAs and proteins, and inhibits viral replication at intermediate stage, all of which are in line with its antiviral mechanism of action. Overall, we developed a robust high throughput-screening assay for screening broad-spectrum influenza antivirals targeting PA-PB1 interaction and identified R151785 as a promising antiviral drug candidate.

Human Cytomegalovirus Viral Latency Protein pUL138 Activates WDR48-USP1 Signaling Axis to Suppress Fanconi Anemia DNA Repair Pathway

***Sebastian Zeltzer^{1,2}, Jason Buehler^{1,2}, Kristen Maness³, Scott Terhune⁴,
and Felicia Goodrum^{1,2}***

¹Department of Immunobiology, ²BIO5 Institute, ³Department of Cellular and Molecular Medicine, University of Arizona, Tucson, AZ, ⁴Medical College of Wisconsin, Milwaukee, WI

Human cytomegalovirus (HCMV) is beta herpesvirus which infects the majority of the human population. Infection is lifelong with viral reservoirs maintained in latently infected hematopoietic stem cells (HPCs). HCMV replication requires nuclear viral DNA synthesis, and numerous studies have explored the coincidence of viral replication centers with host DNA damage response (DDR) proteins. Broadly, these studies indicate that HCMV induces signaling events consistent with increased Homologous Repair (HR), a high-fidelity DNA repair pathway. However, there are conflicting observations regarding the significance of HR for viral replication, and there is no data regarding the significance of any form of DDR for the regulation of viral latency. The viral protein pUL138 is essential to maintain and establish the latent program, however, little is known regarding how pUL138 regulates these processes. Here we show that pUL138 interacts with WDR48, a scaffold protein that activates ubiquitin specific protease 1 (USP1). USP1 is responsible for the inactivation of the Fanconi Anemia (FA) pathway, an essential mediator of HR. We found that pUL138 activates USP1, thereby suppressing the FA pathway in replicative infection. The significance FA signaling for HCMV replication is unknown, therefore we depleted fibroblasts of essential FA factors: FANCD2 and FANCI, and found diminished viral production in each knockdown, indicating the importance of FA signaling for viral replication. To explore the relevance of the FA pathway in latency, we inhibited USP1 in latently infected HPCs using the USP1 inhibitor C527. Inhibition of USP1 resulted in a loss of latency in WT infected cells, phenocopying HPCs infected with virus lacking pUL138. Together these results suggest an emerging model whereby HCMV coopts the FA DNA repair pathway to mediate viral production and inhibits the same pathway via pUL138-USP1 activity to establish and maintain latency.

Chronic *Toxoplasma gondii* Infection Blunts the Immune Response to West Nile Virus Increasing Susceptibility

**Jennifer L. Uhrlaub^{1,2,4}, Kathryn E. McGovern^{1,3,4}, Anita A. Koshy^{1,3,4},
and Janko Nikolich-Žugich^{1,2,3}**

¹Department of Immunobiology, ²Arizona Center on Aging, ³Department of Neurology, ⁴BIO5 Institute, University of Arizona, Tucson, AZ

Toxoplasma gondii (*T. gondii*), a common brain-tropic parasite, is estimated to chronically infect the central nervous system (CNS) of up to a third of the world's population. A continuous immune response prevents the reactivation of cysts within the CNS and dramatically alters the immune landscape of the brain. Whether, and to what extent, the presence of both the parasite and the immune cells that keep it in check impact the immune response to other neurotropic infections has not been well studied. West Nile virus (WNV) is a mosquito-borne infection with a clinical spectrum ranging from asymptomatic or mild flu-like symptoms to more severe neuroinvasive conditions, including meningitis, encephalitis, acute flaccid paralysis, and death. Leveraging well-characterized mouse models for both *T. gondii* and West Nile virus (WNV), we assessed the impact of chronic *T. gondii* infection on the immune response and outcome to WNV. We determined that granzyme effector function is reduced in WNV-specific CD8 T cells and susceptibility to WNV is increased.

The Effects of Systemic β -adrenergic Receptor Activation on Immune Cell Mobilization and Cytotoxicity *in vitro* and *in vivo*

Grace M. Niemi¹, Douglass Diak⁶, Forrest L. Baker⁶, Kyle A. Smith², Tiffany M. Zuniga³, Preteesh Mylabathula⁶, Gustavo Durazo⁴, Michael Seckeler⁵, Emmanuel Katsanis¹, and Richard J. Simpson^{1,3}

¹Department of Pediatrics, ²Department of Physiology, ³Department of Nutritional Sciences, University of Arizona, ⁴Clinical and Translational Sciences, University of Arizona, ⁵Cardiology, Banner Hospital, Tucson, AZ, ⁶Health and Human Performance, University of Houston, Houston, TX

Graft-versus-host disease (GvHD) is a potentially fatal side effect of hematopoietic cell transplant (HCT), a common treatment for hematological malignancies. β -adrenergic receptor (β -AR) stimulation has been shown to mobilize immune cells with a phenotype that correlates with reduced GvHD. However, the effect of β -AR stimulation on hematological outcomes has not been investigated. 10 healthy participants (4 female) underwent a standard HCT donation regiment (5 daily granulocyte-colony stimulating factor (G-CSF) injections). The day after the last injection, participants completed a 20-minute isoproterenol (ISO) infusion (50ng/kg/min). Cytotoxicity assays were completed against chronic myeloid leukemia cells (K562) and multiple myeloma cells (U266). Cells from rest and ISO were injected into standard-NSG humanized mice and monitored for survival, GvHD, and human cell engraftment. ISO significantly increased total lymphocytes, $\gamma\delta$ + T-cells, natural killer (NK) cells, CD4+ and CD8+ effector memory T-cells, CD8+/CD45RA+ effector memory T-cells, NKG2D+ NK cells, NKG2A+ NK cells, NKG2C+ NK cells, and CD34 progenitor cells compared to rest. ISO decreased proportions of CD4+ T-cells, B cells, CD4+ naïve T-cells, CD8+ central memory T-cells, NKG2A+ NK cells, monocytic-myeloid derived suppressor cells (MDSCs), and polymorphonuclear-MDSCs. ISO increased the proportion of T cells that were CD8+ and $\gamma\delta$ +, NK cells, CD4+ effector memory T-cells, and NKG2D+ NK cells. Immune cells collected during ISO had a significantly higher hemacytotoxicity against K562 and U266 *in vitro*. Mice injected with ISO cells trended to have higher survival compared to rest ($p=0.06$) and lower GvHD scores ($p=0.06$). In conclusion, ISO increases the number and proportion of NK cells and $\gamma\delta$ T-cells, and this may be the mechanism responsible for reduced GvHD, longer survival, and increased hemacytotoxicity. However, these associations are somewhat speculative, and more research is needed to investigate the potential beneficial effect of β -AR stimulation on HCT outcomes.

Regulation of Intestinal Tumorigenesis and Metastasis by Wnt Ligands

Al Bothwell, PhD

Department of Immunobiology, Yale Medical School, New Haven, CT

This work has revealed the remarkable immunoregulatory roles of the Wnt antagonists DKK1 and DKK2. We first identified DKK1 as a critical component of regulatory T cells and then our ability to make a recombinant source of DKK1 showed immunoregulation at physiologic concentrations was a potent driver of Th2 differentiation. Both DKK1 and DKK2 fundamentally regulate anti-tumor responses. In intestinal cancer DKK2 contributes to the decision of cancer stem cells to proliferate or differentiate. Using a mouse AOM-DSS colon cancer tumor model colon polyps were substantially reduced in the absence of DKK2 either in colon epithelial or stem cells. RNAseq analysis of the polyps by Ingenuity Pathway Analysis (IPA) predicted the connection of DKK2 with the transcription factor HNF4 α . Work by others demonstrated that c-Src was responsible for phosphorylating HNF4 α which then results in its degradation. Studies of organoids demonstrate that recombinant DKK2 can induce transcription of the DKK2 gene suggesting an autoregulatory stimulatory pathway. Results suggest DKK2 can activate c-Src by phosphorylation which then phosphorylates HNF4 α 1 at three Y residues. The result is that phosphorylated HNF4 α 1 is then degraded by the proteasome. This leads to increased expression of the stem cell marker Lgr5 in organoids. Finally, organoids with reduced DKK2 which have reduced Lgr5 show very significant reduction of metastasis. Our RNAseq and qRT-PCR studies combined with the IPA analysis lead to the formulation of a unique model which may contribute to the development of some metastatic cancers. Studies of organoids have given insight into mechanisms of tumorigenesis. Comparisons of human patient derived xenografts are ongoing to assess conservation of mechanisms of carcinogenesis and metastasis in humans and mice.

Transcriptional Insights into *Toxoplasma gondii*-injected Neurons

Anita A. Koshy, MD

Department of Immunobiology, Department of Neurology, BIO5 Institute, University of Arizona, Tucson, AZ

Toxoplasma gondii is an obligate intracellular that chronically infects the CNS of up to one-third of the human population. While this chronic CNS infection is asymptomatic in most immune competent people, this tropism for and persistence in the CNS underlies the neurologic disease *Toxoplasma* causes in the immunocompromised. Using a novel mouse model in which CNS cells injected with *Toxoplasma* protein are permanently marked (*Toxoplasma*-Cre system), we determined that *in vivo* neurons are the primary CNS cell with which *Toxoplasma* interacts and infects. Given that *Toxoplasma*— like other intracellular microbes— is dependent on its host cell for survival, this predilection for neurons suggests that *Toxoplasma*'s ability to persist in the CNS depends specifically upon parasite manipulation of the host neurons; yet, the majority of the work on *Toxoplasma*-host cell manipulation has been done *in vitro* and in non-neuronal cells. To address this gap in knowledge, we used laser capture microdissection coupled to RNAseq to isolate and transcriptionally profile *Toxoplasma*-injected neurons (TINs), bystander neurons (neighboring non-*Toxoplasma* injected neurons), and neurons from uninfected mice (controls). Analyses of the resultant data led to both expected (TINs and bystander transcriptomes are consistent with neuroinflammatory responses) and unexpected results (T cells are clustering around TINs?), leading us to reconsider neuron capabilities for immune responses.

Enteropathogenic *Escherichia coli* Dynamically Regulates Host Mitochondrial Morphology and Function

Jennifer Lising Roxas¹, Shylaja Ramamurthy¹, Anusha Harishankar¹, John Scott Wilbur¹, Gresa Sylejmani^{1,5}, Gayatri Vedantam^{1,2,3,4}, and V.K. Viswanathan^{1,2,3}

¹School of Animal and Comparative Biomedical Sciences, ²Department of Immunobiology, ³BIO5 Institute, University of Arizona, ⁴Southern Arizona VA Health Care System, Tucson, AZ, ⁵The University of New Mexico Health Sciences Center, Albuquerque, NM

Enteropathogenic *Escherichia coli* (EPEC), a leading cause of juvenile diarrheal disease mortality, belongs to a family of organisms known as attaching and effacing (A/E) pathogens that intimately attach to host intestinal epithelial cells and efface brush-border microvilli. EPEC, via a type III secretion system, translocates effector molecules into intestinal epithelial cells. Secreted effectors engage various host signaling and structural molecules, alter epithelial cell function, and contribute to pathogenesis. EPEC perturbs host mitochondrial structure and function, but the bacterial proteins responsible, the underlying mechanisms, or their relationships to disease, are unknown. We previously showed that the early-secreted effector EspZ and the late-secreted effector EspH are essential for A/E pathogen virulence. In this study, we explored the intersecting actions of EspZ and EspH on host cell mitochondrial stability, structure and function. We show that the cytoprotective EspZ localizes to host mitochondria and interacts with the mitochondrial fission protein hFis1. Electron microscopy revealed that cells infected with an EspZ-deficient strain (Δ espZ) have swollen, rounded mitochondria, often within double membranes; infection with the EspH-deficient strain (Δ espH) resulted in fused, elongated mitochondria. EspZ prevents DRP1-mediated mitochondrial fragmentation and inhibits mitophagy. The late effector EspH contributes to infection-induced increase in hFis1 levels and, in contrast to EspZ, promotes mitochondrial fission. Thus, EspH promotes, while EspZ protects against, mitochondrial potential loss during infection. Collectively, our observations are consistent with a model whereby early EspZ-dependent mitochondrial stabilization facilitates A/E pathogen colonization, while late EspH-dependent mitochondrial damage and host cell death contributes to pathology and disease severity. Our results suggest that effector-mediated temporally coordinated impacts on host mitochondria may contribute to EPEC pathogenesis.

Poster Titles

| Poster | Author | Abstract Title |
|--------|----------------------|---|
| 1 | Arveen Asghar | The role of Interleukin-6 in age-related frailty syndrome |
| 2 | Forrest Baker | Systemic β -Adrenergic Receptor Activation Augments the ex vivo Expansion and Anti-Tumor Activity of V γ 9V δ 2 T-Cells |
| 3 | Danielle Becktel | Cyclodextrin treatment substantially reduces the chronic inflammatory response following ischemic stroke in adult and aged mice |
| 4 | Christine Bradshaw | TGF β neutralization improves humoral immunity to viral infection in aged mice |
| 5 | Caitlyn Dagenet | The Role of T Follicular Regulatory Cells in Autoimmune Arthritis |
| 6 | Douglass Diak | Lymphocytes collected during exercise decrease leukemia burden in a humanized mouse model compared to lymphocytes collected at rest |
| 7 | Camryn Garza | <i>Parvimonas micra</i> and <i>Finegoldia magna</i> elicit an inflammatory effect in cervical epithelial cells |
| 8 | Karen Hastings | Neoantigen fitness model predicts lower immune recognition of cutaneous squamous cell carcinomas than actinic keratoses |
| 9 | Michael Khanisho | Evaluating the Effects of Chlorhexidine Gluconate and Povidone-Iodine on the Cervicovaginal Epithelium and Health-Associated <i>Lactobacilli</i> Found in Vaginal Microbiome |
| 10 | Shio Kobayashi | A biomimetic five-module chimeric antigen receptor (5MCAR) designed to target antigen-specific pathogenic T cells |
| 11 | Nicole Kummert | Pre-Transplant Bendamustine Conditioning Induces Graft-versus-Leukemia Effect with Lower Graft-versus-Host Disease than Cyclophosphamide in Murine Models |
| 12 | Mark Lee | Evolution has imposed a functional requirement for CD4-Lck association |
| 13 | Maria Love | Evaluation of HIV-Specific T-Cell Responses in HIV-Infected Aging Patients with Controlled Viremia on Long-term Antiretroviral Therapy |
| 14 | Jason Maarsingh | <i>Peptoniphilus</i> , <i>Porphyromonas</i> , and <i>Eggerthella</i> exhibit genus-specific cytotoxic and proinflammatory responses in 3-D human cervical cell models |
| 15 | Anne Macy | Characterization of GILT and MHC Class II in Murine Melanoma Cell Lines for Determination of the Role of the MHC class II Pathway in Melanoma Cells |
| 16 | Sanjay Menghani | A novel copper-dependent antimicrobial against <i>Staphylococcus aureus</i> and <i>Streptococcus pneumoniae</i> |
| 17 | Luwanika Mlera | Host regulation of UL136p33, a modulator of human cytomegalovirus latency and reactivation |
| 18 | Belen Molina | Human Cytomegalovirus Modulates MVB Biogenesis During Infection in Endothelial Cells |
| 19 | Samina Momtaz | Differential remodeling of multivesicular bodies by human cytomegalovirus (HCMV) in endothelial cells and fibroblasts |
| 20 | Melissa Moy | Viral Proteins UL136-33kDa and UL135 Synergistically Promote Human Cytomegalovirus Reactivation from Latency |
| 21 | Preteesh Mylabathula | The Impact Of Simulated Microgravity On The Anti-Tumor Properties Of Human Nk-Cells and $\gamma\delta$ -T Cells In Vivo: IL-2 And Zoledronic Acid Therapy As A Potential Countermeasure |
| 22 | Matthew Ollerton | TFR Reduce HIV-1 Infected TFH in vitro in an IL-2 Dependent Manner |

| | | |
|----|--------------------|--|
| 23 | Shylaja Ramamurthy | EspZ Transmembrane Domain 2 Residues Facilitates Self-association and Contribute to Virulence of Attaching and Effacing Pathogens |
| 24 | Dakota Reinartz | The Role of AIM2 in Head and Neck Squamous Cell Carcinoma Carcinogenesis |
| 25 | Tyler Ripperger | Transcriptional Modulation of Antibody-Mediated Immunity |
| 26 | Bryan Roxas | Phylogenomic and Phenotypic Characterization of the Emerging <i>Clostridioides (Clostridium) difficile</i> Ribotype 106 strains |
| 27 | Mary Salliss | The role of bacterial metabolites, glycochenodeoxycholate (GCDC) and phenyllactate (PLA) on microbe-microbe and microbe-host interactions in the cervicovaginal microenvironment |
| 28 | Kyle Smith | Systemic β 1-Adrenergic Receptor Blockade Augments NK-Cell Mobilization In Response To Acute Exercise In Humans |
| 29 | Lihn Tran | The role of human cytomegalovirus protein UL135 in HCMV-mediated myelosuppression of hematopoietic progenitor cells |
| 30 | Max Wegner | Hypoxia-Inducible Factor 1 α (HIF1 α) limits Human Cytomegalovirus Cell-to-Cell Spread by Attenuating Metabolite Signaling |
| 31 | Alison Williams | A Closer Look At Non-Toxigenic <i>Clostridioides difficile</i> |
| 32 | David Williams | Modeling Human Papillomaviruses from infection to transformation |

Organizing Committee

John Purdy, PhD (chair)
Michael Johnson, PhD
Josh Kochanowsky
Emily Merritt
Shuaizhi Li
Megan S. Molina
Yuecheng Xi
Nicole Swintek
Tonya Fotheringham
Lori Wieland
Vanessa Sophia Gonzalez Verdugo

Further acknowledgement is made for the grateful assistance of:

Tammie Rippberger
Edgar Mendoza - BioCOM

Poster Judges

Joseph Alvin, PhD
Laurie Baert, PhD
Lucas D'Sousa, PhD
Robert Jackson, PhD
Mladen Jergovic, PhD
Sanna Loppi, PhD
Kathryn McGovern, PhD
Grace Niemero, PhD
Kelvin Pond, PhD
Maria Rendon, PhD
Kate Rhodes, PhD
Jennifer Lising Roxas, PhD
Chandrasekaran Sambamurthy, PhD
Sandip Sonar, PhD

Cover photos courtesy of:

*UAHS BioCOM, Images provided by Ian J. Mohr,
Jennifer L. Gommerman, and Andreas J. Bäuml*