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. Bäumler, 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 $N^6$-adenosine Methylation Enzymes
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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-Žugich, 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 N^6-adenosine Methylation Enzymes”
10:20 - 10:40 AM  **BREAK, coffee available in Lobby**  
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**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 Niemiro, 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  Anita Koshy, MD
“Transcriptional Insights into *Toxoplasma gondii*-injected Neurons”

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

2:20 - 2:30 PM  Jennifer Lising Roxas, PhD
“Enteropathogenic *Escherichia coli* Dynamically Regulates Host Mitochondrial Morphology and Function”

2:35 - 3:25 PM  Plenary Lecture, Andreas J. Bäumler, PhD
*UC Davis School of Medicine*
“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
*Please visit our sponsors.*

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

5:10 PM Poster Awards & Drawing for iPad

*The poster session is sponsored by:*

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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. Bäumler, 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 Doorslaer¹,²,³,⁴ and Samuel K. Campos²,³,⁵

¹School of Animal & Comparative Biomedical Sciences, ²Cancer Biology Graduate Interdisciplinary Program, ³Department of Immunobiology, ⁴BIO5 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

¹Department 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\textsuperscript{1,2}, Jason Buehler\textsuperscript{1,2}, Kristen Maness\textsuperscript{3}, Scott Terhune\textsuperscript{4}, and Felicia Goodrum\textsuperscript{1,2}

\textsuperscript{1}Department of Immunobiology, \textsuperscript{2}BIO5 Institute, \textsuperscript{3}Department of Cellular and Molecular Medicine, University of Arizona, Tucson, AZ, \textsuperscript{4}Medical 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. Uhrlahub,1,2,4, Kathryn E. McGovern1,3,4, Anita A. Koshy1,3,4, and Janko Nikolich-Žugich1,2,3*

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 \textit{in vitro} and \textit{in vivo}

\textit{Grace M. Niemiro}\textsuperscript{1}, Douglass Diak\textsuperscript{6}, Forrest L. Baker\textsuperscript{6}, Kyle A. Smith\textsuperscript{2}, Tiffany M. Zuniga\textsuperscript{3}, Preteesh Mylabathula\textsuperscript{6}, Gustavo Durazo\textsuperscript{4}, Michael Seckeler\textsuperscript{5}, Emmanuel Katsanis\textsuperscript{1}, and Richard J. Simpson\textsuperscript{1,3}

\textsuperscript{1}Department of Pediatrics, \textsuperscript{2}Department of Physiology, \textsuperscript{3}Department of Nutritional Sciences, University of Arizona, \textsuperscript{4}Clinical and Translational Sciences, University of Arizona, \textsuperscript{5}Cardiology, Banner Hospital, Tucson, AZ, \textsuperscript{6}Health 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, γδ+ 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 γδ+, 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 γδ 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 HNF4α. Work by others demonstrated that c-Src was responsible for phosphorylating HNF4α 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 HNF4α1 at three Y residues. The result is that phosphorylated HNF4α1 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 Sylejmani¹,⁵, Gayatri Vedantam¹,²,³,⁴, and V.K. Viswanathan¹,²,³

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 (ΔespZ) have swollen, rounded mitochondria, often within double membranes; infection with the EspH-deficient strain (ΔespH) 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.
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<td>EspZ Transmembrane Domain 2 Residues Facilitates Self-association and Contribute to Virulence of Attaching and Effacing Pathogens</td>
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<td>Dakota Reinartz</td>
<td>The Role of AIM2 in Head and Neck Squamous Cell Carcinoma Carcinogenesis</td>
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<td>25</td>
<td>Tyler Ripperger</td>
<td>Transcriptional Modulation of Antibody-Mediated Immunity</td>
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<td>Bryan Roxas</td>
<td>Phylogenomic and Phenotypic Characterization of the Emerging Clostridioides (Clostridium) difficile Ribotype 106 strains</td>
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<td>Mary Salliss</td>
<td>The role of bacterial metabolites, glycochenodeoxycholate (GCDC) and phenyllactate (PLA) on microbe-microbe and microbe-host interactions in the cervicovaginal microenvironment</td>
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<td>Max Wegner</td>
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