



College of Medicine – Tucson
Department of Immunobiology

Seventeenth Annual

Virtual Frontiers in Immunobiology & Immunopathogenesis Symposium

March 11, 2022

Registration: January 12, 2022 — March 11, 2022

Abstract Submission: January 12, 2022 — February 18, 2022

Plenary Speakers

Erika Pearce, Ph.D. (Immunologist)

Johns Hopkins University School of Medicine

Bloomberg Distinguished Professor

Bloomberg~Kimmel Institute for Cancer Immunotherapy

“Mitochondria Shape Shifting in the T Cell Response”

Breck Duerkop, Ph.D. (Bacteriologist)

University of Colorado School of Medicine

Assistant Professor, Department of Immunology & Microbiology

Co-Director, Microbiology Graduate Program

“Enterococcal Phage-Host Interactions and Mechanisms of Resistance”

Daniel DiMaio, M.D., Ph.D. (Virologist)

Yale School of Medicine

Waldemar Von Zedtwitz Professor of Genetics

Professor of Molecular Biophysics and Biochemistry

Professor of Therapeutic Radiology

Deputy Director, Yale Cancer Center

“The Unique Cell Biology of Human Papillomavirus Entry”



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Immunobiology



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Welcome to the 17th Annual Frontiers in Immunobiology & Immunopathogenesis Symposium. We have remained virtual this year and returned to the Accelevents online platform (<https://www.accelevents.com/e/2022IMBSymposium>). On the website, you will be able to find the schedule, see our sponsor Thermo Fisher Scientific, share the registration link (in case you want to pass the word along to your friends/colleagues) and enter the event on the day of. When you enter the event, you will be able to see all the event links to the talks, poster sessions, and interactive plenary speaker lunch sessions that will occur throughout the day.

This symposium will feature great scientific talks that will hopefully lead to many thought-provoking moments. The symposium is headlined by our plenary speakers Drs. Daniel DiMaio, Erika Pearce, and Breck Duerkop. We will also feature six trainee talks and three University of Arizona faculty talks.

Like last year, we will have two 45-minute poster sessions. To view the Poster Sessions on Accelevents, go to “Workshops” and click on “Read More” under the description, to view all the Zoom links for the Poster Presenters. Posters should also be available to download to view independently as presenters will be sending them through the Chat on Zoom (as one might look from afar before approaching a poster).

Lastly, there will be a door prize of an iPad. For each session you interact with our sponsor Fisher Scientific, you will get a number that you can use to enter the raffle (Note: the winner needs to be present at the end for the Poster Award session). The only way to get the ticket numbers for the raffle is by attending the sponsor session and/or the poster session. That means the more you interact, the better your chances!

We encourage you to explore the Accelevents website during the Pre-Event access starting at 7:30 AM. If you have any questions or issues, please click “Lounge” on the left panel and go to the “Help Desk”; where we will be standing by to help you. Please access the Symposium through Accelevents. In the event of a site issue, please join via Zoom for the Main Stage Sessions <https://arizona.zoom.us/j/86347048277> Password: 2022.

In closing, we truly hope you enjoy the 17th Annual Frontiers in Immunobiology & Immunopathogenesis Symposium. We hope you leave inspired and encouraged that we can overcome all sorts of barriers to facilitate the production and discourse of great science.

Sincerely,

Michael, Justin, and the 2022 Symposium Committee Team

Agenda - Frontiers in Immunobiology & Immunopathogenesis Symposium

8:30 - 8:45 AM

Welcome Announcements

Janko Nikolich-Žugich, MD, PhD

Professor and Department Head, Immunobiology

Director, Aegis Consortium for Pandemic - Free Future

Co-Director, Arizona Center on Aging

Michael M. I. Abecassis, MD, MBA

Dean, College of Medicine – Tucson

Professor, Departments of Surgery and Immunobiology

Jennifer Barton, PhD

Director, BIO5 Institute

Professor, Biomedical Engineering

Session I

Moderator: Kristen Zarrella

8:45 - 9:35 AM

Plenary Lecture, Daniel DiMaio, MD, PhD

Yale Cancer Center

"The Unique Cell Biology of Human Papillomavirus Entry"

9:40 - 9:50 AM

Pierce Longmire

"Human Cytomegalovirus Hijacks Host Translesion Synthesis Repair for Genome Integrity"

9:55 – 10:05 AM

Rob Jackson

"HPV16's Differentiation-Dependent Viral Lifecycle In 3D Oral Epithelia"

10:10 – 10:30 AM

Mike Worobey, PhD

"The Huanan Market Was the Epicenter Of SARS-Cov-2 Emergence"

10:35 – 10:45 AM

Break/ Sponsors

Session II

Moderator: Tyler Ripperger

- 10:45 - 10:55 AM** *Kristen Zarrella*
“HCMV Latency Protein UL138 Modulates STAT1 Signaling for Latency”
- 11:00 – 11:50 AM** **Plenary Lecture, *Erika Pearce, PhD***
Johns Hopkins University School of Medicine
“Mitochondria Shape Shifting in the T Cell Response”
- 11:55 – 12:40 PM** *Poster Session I/ Sponsors*
- 12:45 - 1:45 PM** ***Lunch Sessions with Plenary Speakers***

Session III

Moderator: Tyler Ripperger

- 1:50 – 2:10 PM** *Tara Carr, MD*
“Connecting Environment to Asthma Risk”
- 2:15 – 2:25 PM** *Lucas D’souza*
“Genetic Evidence That Uptake of The Fluorescent Analog 2NBDG Occurs Independently of Known Glucose Transporters”
- 2:30 – 2:40 PM** *Caleb Kim*
“The Unique TCR C α Domain Regulates T-Cell Development, Homeostasis, And Responses to Agonist Peptide-MHC”
- 2:40 – 2:50 PM** ***Break/ Sponsors***

Session IV

Moderator: Dakota Reinartz

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|-----------------------|--|
| 2:50 – 3:40 PM | Plenary Lecture, <i>Breck Duerkop, PhD</i>
<i>University of Colorado School of Medicine</i>
“Enterococcal Phage-Host Interactions and Mechanisms of Resistance” |
| 3:45 – 3:55 PM | <i>Kate Rhodes</i>
“Identification of Host Interaction Factors Required for Asymptomatic Carriage of Neisseria” |
| 4:00 – 4:20 PM | <i>Katri Typpo, MD, MPH</i>
“Modulation of the Gut-Lung Axis to Improve Pediatric Acute Lung Injury” |
| 4:25 – 5:05 PM | <i>Poster Session II/ Sponsors</i> |
| 5:20 PM | <i>Poster Awards</i> |

Plenary Lecture

Enterococcal Phage-Host Interactions and Mechanisms Of Resistance

Breck Duerkop, PhD

*Assistant Professor Co-Director, Microbiology Graduate Program Department of Immunology & Microbiology
University of Colorado School of Medicine*

My laboratory studies bacterial viruses (bacteriophages or phages) and their interactions with their hosts. The long-term goal of my laboratory is to understand how phages and other forms of mobile DNA contribute to host-microbe interactions and their overall impact on human health. This seminar will highlight our work that explores the mechanisms of phage interactions with multidrug resistant enterococci and how phage resistance impacts enterococcal fitness, leading to diverse phenotypes that include antibiotic resistance, anti-phage restriction, intestinal colonization, and polymicrobial interactions.

Plenary Lecture

The Unique Cell Biology of Human Papillomavirus Entry

Daniel DiMaio, MD, PhD

*Waldemar Von Zedtwitz Professor of Genetics and Professor of Molecular Biophysics and Biochemistry and of
Therapeutic Radiology Deputy Director Yale Cancer Center*

During human papillomaviruses entry, the cytoplasmic protein complex known as retromer sorts incoming virions into a series of vesicular compartments that comprise the retrograde transport pathway leading to the nucleus. To access the retromer and other cytoplasmic entry factors from the endosome lumen, a cell-penetrating peptide (CPP) on the C-terminus the L2 minor capsid protein mediates protrusion of L2 through the endosome membrane into the cytoplasm. We isolated four artificial proteins named traptamers that inhibit different entry steps and used them to further dissect HPV entry and trafficking. One traptamer stimulates ubiquitination of L2 and diverts incoming virus to the lysosome, whereas the others act downstream by preventing sequential passage of the virus through retrograde compartments. Further studies revealed that exit of HPV from the endosome requires cycling of Rab7 between GTP- and GDP-bound forms and that the L2 CPP mediates transient insertion of L2 into the endosome membrane, which is stabilized by retromer-L2 binding. These results define the retrograde trafficking route taken by HPV during entry, reveal the role of Rab7 in this process, and show that retromer plays a role in CPP-mediated membrane insertion.

Plenary Lecture

Mitochondria Shape Shifting in The T Cell Response

Erika Pearce, PhD

*Bloomberg Distinguished Professor Bloomberg Kimmel Institute for Cancer Immunotherapy Johns Hopkins
University School of Medicine*

Pearce's research group uses a variety of approaches to address key questions in immune cell metabolism, and how this impacts immunity to infection and cancer. Her work is focused on the role of metabolism in regulating the development and function of T cells, white blood cells that play a central role in the body's immune response. Having found that structural changes to the mitochondria affect the mitochondrial metabolism, and that this metabolism fuels the differentiation, survival, and function of T cells, Pearce is now working to develop ways to make better, long-lived T cells that will improve immune therapies against tumors, pathogens, and other diseases.

Human Cytomegalovirus Hijacks Host Translesion Synthesis Repair for Genome Integrity***Pierce Longmire¹, Sebastian Zeltzer, PhD², Marek Svoboda³, Giovanni Bosco, PhD³, and Felicia Goodrum, PhD⁴****¹Graduate Program in Molecular Medicine, University of Arizona, Tucson, AZ, ²BIO5 Institute, University of Arizona, Tucson, AZ ³Department of Molecular and Systems Biology, Dartmouth Geisel School of Medicine, Hanover, NH, ⁴Department of Immunobiology, University of Arizona, Tucson, AZ*

Human cytomegalovirus (HCMV) persists through lifelong infection in a majority of the global population. The mechanisms by which HCMV toggles between latent and replicative states are poorly defined. Understanding virus-host interactions behind these mechanisms is key to developing novel strategies to control reactivation from latent infection in the context of immune suppression, where HCMV reactivation is a significant cause of morbidity and mortality. Through studying host proteins that contribute to the replicative cycle, we find that the host DNA processivity factor, proliferating cell nuclear antigen (PCNA), re-localizes to viral DNA synthesis. Infection induces monoubiquitination (mUb) of PCNA and recruits host translesion synthesis (TLS) polymerases to replication centers, consistent with activation of the TLS repair pathway for DNA lesion bypass. We find that TLS polymerases specialized in insertion of nucleotides across lesions restrict virus replication. In contrast, the TLS polymerase complex that extends past lesions is required for optimal virus replication. Strikingly, depletion of either group of TLS polymerases causes increased viral genome rearrangements, demonstrating a role for these host polymerases in regulating viral genome integrity. We are investigating how viral protein UL138, a latency determinant, modulates this pathway through interaction with ubiquitin specific peptidase 1 (USP1). This work provides the first insights how HCMV hijacks specialized host polymerases and DNA damage response (DDR) pathways to regulate virus replication and genome stability. We anticipate that this work will provide mechanistic insight into the role of the DDR in infection and its role in the establishment and maintenance of latency.

HPV16's Differentiation-Dependent Viral Lifecycle In 3D Oral Epithelia

Robert Jackson, PhD¹, Esha V Rajadhyaksha², Caitlyn E Flores¹, David Williams³, and Koenraad Van Doorslaer, PhD^{1,2,3,4}

¹School of Animal & Comparative Biomedical Sciences, ²College of Medicine; College of Science, ³Cancer Biology and Genetics Graduate Interdisciplinary Programs, ⁴Department of Immunobiology, BIO5 Institute

Human papillomavirus (HPV) infections cause 5% of human cancers worldwide. In addition to cervical cancer, oncogenic HPVs are responsible for an epidemic number of oral cancers. HPV16 causes nearly all these HPV-associated oral cancers in men and women. The reasons for this increased incidence are poorly understood and confounded by a lack of adequate experimental systems for studying the full viral lifecycle. As the complete HPV lifecycle requires differentiation of stratified epithelia, we use 3D tissue culturing to grow physiologically relevant epithelia for studying pathogen-host interactions. Due to their availability, foreskin keratinocytes are often used but may not be suitable for modelling distinct anatomic sites of natural HPV infection and carcinogenesis (e.g., cervical and oral epithelium). To identify tissue-specific characteristics that would impact HPV infection, we grew 3D epithelia from foreskin, cervix, and tonsil-derived primary cells (three independent donors each). Histological validation of the 3D stratified squamous epithelia identified differences between these tissues (e.g., mucosal cytoplasmic clearing). Bulk RNA-seq revealed 1,238 differentially-expressed genes (likelihood ratio test, P-adj < 0.01) and unique transcriptional profiles for each tissue type. Functionally, these genes have over-representation of tissuespecific transcription factors, epithelial differentiation markers, and innate immune processes. This demonstrates that tissue origin may influence the outcome of HPV infection, highlighting a need to use relevant tissue models to study the lifecycle of these viruses. We want to understand HPV16's unique role in causing oral cancers and how its differentiation-dependent lifecycle is regulated in oral tissues. Using our characterized oral cells, we established HPV16 genomes into two independent donor clones. Following differentiation, cells were processed for single cell RNA-seq to examine covarying viral and host genes to identify host pathways active in spatially distinct differentiation stages of the multi-layered 3D oral epithelium. Overall, understanding the interplay between host differentiation and HPV16's lifecycle will identify key host factors regulating oral HPV infection.

HCMV Latency Protein UL138 Modulates STAT1 Signaling for Latency

Kristen Zarrella¹, Sebastian Zeltzer², Pierce Longmire¹, Meaghan Hancock³, Donna Collins-McMillen², Jason Buehler⁴, Justin Reitsma⁵, Jay Nelson³, Scott Terhune⁵, and Felicia Goodrum, PhD¹

¹Department of Immunobiology, ²BIO5 Institute, University of Arizona, Tucson, Arizona, ³Vaccine and Gene Therapy Institute, Oregon Health and Science University, Beaverton, OR, ⁴Imanis Life Sciences, Rochester, MN, ⁵Microbiology and Immunology, Medical College of Wisconsin, Wauwatosa, WI,

Human Cytomegalovirus (HCMV) is a beta herpesvirus that persists in majority of the population through the establishment of viral latency. HCMV latency occurs in hematopoietic cells through complex virus-host interactions that remain incompletely defined. We have identified UL138 as an HCMV gene that is required for latency in hematopoietic cells. Towards understanding how UL138 functions in latency, we have identified an interaction between UL138 and ubiquitin specific peptidase-1 (USP1) and its scaffold protein, WDR48. WDR48 activates the deubiquitinase activity of USP1, which functions to downregulate the DNA damage response, while sustaining an antiviral innate immune response. Both pathways can lead to the cell mounting an inflammatory response to viral infection. We have shown that UL138 activates USP1 activity and loss of USP1 results in a loss of latency, marked by viral replication in hematopoietic cells in the absence of a stimulus for reactivation. USP1 has been reported to sustain signal transducer and activator of transcript 1 (STAT1) phosphorylation (pSTAT1) and an interferon response. Therefore, we hypothesized that UL138 through USP1 sustains pSTAT1 for the establishment of latency. We have found that HCMV infection stimulates phosphorylation and activation of STAT1 in a UL138-, USP1-, and TBK1- dependent manner during a productive infection. Further, the ISG response downstream of pSTAT1 is enhanced and sustained in the presence of UL138. Additionally, the kinase of STAT1, JAK1, represses viral replication during latency in hematopoietic cells. We are exploring the importance of UL138-mediated pSTAT1 induction and the innate response in suppressing virus replication for latency establishment. We have further evidence that UL138 is also directing USP1-mediated modulation of the DDR, which implies an intriguing co-regulation of these host pathways. By defining the mechanisms underlying HCMV persistence and its regulation of the innate immune response, we will gain insight into how to target this virus.

Genetic Evidence That Uptake of The Fluorescent Analog 2NBDG Occurs Independently of Known Glucose Transporters

Lucas D'souza, PhD¹, Stephen Wright, PhD², and Deepta Bhattacharya, PhD¹

¹Department of Immunobiology, and ²Department of Physiology, University of Arizona, Tucson, AZ

The fluorescent derivative of glucose, 2-Deoxy-2-[(7-nitro-2,1,3-benzoxadiazol-4-yl)-amino]-D-glucose (2NBDG), is a widely used surrogate reagent to visualize glucose uptake in live cells at single cell resolution. Using a model of CRISPR-Cas9 gene editing in 5TGM1 myeloma cells, we demonstrate that ablation of the glucose transporter gene Slc2a1 abrogates radioactive glucose uptake but has no effect on the magnitude or kinetics of 2NBDG import. Extracellular 2NBDG, but not NBD-fructose was transported by plasma cells into the cytoplasm suggesting specific activity that is unlinked to glucose import and that of chemically similar compounds. RNA-Seq analysis of primary plasma cells and the 5TGM1 myeloma cell line revealed expression of other candidate glucose transporters. Yet, deletion of these transporters individually or in combination with one another also had no impact on 2NBDG uptake. Ablation of the genes in the Slc29 and Slc35 families of nucleoside and nucleoside sugar transporters as well as the ATP-binding cassette (ABC) transporter family also failed to impact 2NBDG import. Thus, cellular uptake of 2NBDG is promoted by an unknown mechanism and is not a faithful indicator of glucose transport.

The Unique TCR C α Domain Regulates T-Cell Development, Homeostasis, And Responses to Agonist Peptide-MHC

Caleb Kim¹, Heather Parish PhD¹, and Michael Kuhns PhD¹

¹*Department of Immunobiology, University of Arizona, Tucson, Arizona*

T-cells play a critical role in adaptive immune responses upon recognition of peptide-antigens presented in the context of MHC molecules (pMHC). They do this using the T-cell receptor (TCR), which is made up of extracellular, transmembrane and intracellular regions. The major building block of the extracellular regions are immunoglobulin (Ig) domains, which consist of two β sheets pressed together with a hydrophobic core. For example, the variable (V) regions of the TCR α and TCR β chains consist of Ig-folds. Likewise, the TCR α constant region (C) consists of an Ig-fold. However, the TCR β C region (C α) is missing the top β sheet of its Ig-like structure and instead has two loosely-associated top strands (C and F strands) on its surface. Previous results suggests that this evolutionarily conserved region mediates TCR multimerization and impacts signaling in vitro, but the functional significance of this region in vivo has yet to be characterized. In this study, we made transgenic OTII Rag1^{-/-} C57Bl/6 mice with or without mutations on the C α C-strand to determine what the fitness cost is for mice bearing mutated C α domain. Our results suggest that mutating this region increases TCR tonic signaling, and positive selection in the thymus, thymic output into the periphery, and homeostatic survival of T-cells in the periphery. We interpret our data as evidence that the unique C α domain plays a role in fine-tuning TCR signaling.

Identification Of Host Interaction Factors Required for Asymptomatic Carriage of Neisseria

Katherine Rhodes, PhD^{1,2}, Man Cheong Ma, PhD³, Maria Rendon, PhD^{1,2}, Rodrigo Arana, Magdalene So, PhD^{1,2}

¹Department of Immunobiology, ²Bio5 Institute, University of Arizona, Tucson, AZ., and ³Boehringer-Ingelheim, Fremont, CA

Human adapted commensal *Neisseria* are common members of the mucosal microbiota, however, the processes by which they shape and maintain a niche in their natural host are not well understood. By examining the function of host interaction factors conserved in commensal and pathogenic *Neisseria* during host carriage, we may elucidate the bacterial mechanisms driving commensalism and asymptomatic infection within the genus. Here, we performed an in vivo screen of a transposon mutant library of *Neisseria musculi*, a commensal of wild-caught mice that persistently and asymptotically colonizes the oral cavity and gut of laboratory mice. Through this method we identified approximately 500 candidate genes required for long-term host interaction. Among these are homologs encoding virulence factors of *Neisseria meningitidis* and *Neisseria gonorrhoeae*, as well as many genes of unknown function found in both human and animal adapted *Neisseria*. We validated a set of candidate genes encoding the biosynthesis of a polysaccharide capsule, which is widely considered a *N. meningitidis* virulence factor, by construction and in vivo testing of a defined mutant which cannot produce capsule. Results show that the mutant colonized but failed to persist long term in mice. Our findings highlight the utility of the *Nmusmouse* model for as a tool for discovery, and represent a first step in the characterization of novel host interaction factors conserved across the genus.

The Huanan market was the epicenter of SARS-CoV-2 emergence

Michael Worobey, PhD

Department Head, Ecology and Evolutionary Biology, Professor, Ecology and Evolutionary Biology, Professor, BIO5 Institute, Professor, Genetics - GIDP, University of Arizona

Despite strong epidemiological links and the documented presence of SARS-CoV-2 susceptible animals, the role of the Huanan Seafood Wholesale Market in the COVID-19 pandemic remains controversial. Using spatial analyses we show that the earliest known COVID-19 cases diagnosed in December 2019 were geographically distributed near to, and centered on, this market. This distribution cannot be explained by high densities of elderly people at greater risk of symptomatic COVID-19. This pattern was stronger in cases without, rather than with, identified epidemiological links to the Huanan market, consistent with SARS-CoV-2 community transmission starting in the surrounding area. By combining spatial and genomic data, we show that both the two early lineages of SARS-CoV-2 have a clear association with the Huanan market. We also report that live mammals, including raccoon dogs, were sold at the market in late 2019 and geospatial analyses within the market show that SARS-CoV-2-positive environmental samples were strongly associated with vendors selling live animals. Together, these analyses provide strong evidence for the emergence of SARS-CoV-2 via the live wildlife trade and identify the Huanan market as the unambiguous epicenter of the COVID-19 pandemic.

Connecting Environment to Asthma Risk

Tara Carr, MD

*Associate Professor, Medicine - (Clinical Scholar Track, Associate Professor, Otolaryngology
Director, Adult Allergy Program, Director, Allergy & Immunology Fellowship Program, University of Arizona*

The Binational Early Asthma and Microbiome Study is an NIAID-funded program project grant that builds on our group's observations that children of Mexican descent living in Tucson, AZ have a fourfold higher risk of asthma than children living just across our border, in Nogales, Mexico. This cohort will recruit pregnant women from our clinical partners in both Nogales and Tucson. Human and environmental samples will be collected in the prenatal, perinatal periods and through the first two years of the baby's life. Microbiome and immunology testing will establish relationships between environment, personal microbial characteristics, immune development, and health outcomes, specifically a surrogate marker of asthma in early life, T2 wheeze. Partners at UCSF will perform in depth assessment of bacterial function and identification of potentially protective bacterial products. Mouse models will study maternal to fetal microbial transmission complemented by environmental exposures in asthma risk and pathobiology. BEAMS hopes to identify asthma-protective factors in Mexico that would provide novel tools toward prevention of asthma and related diseases.

Modulation of the Gut-Lung Axis to Improve Pediatric Acute Lung Injury

Katri Typpo, MD, MPH

Associate Professor, Pediatrics, University of Arizona, Tucson, AZ

Severe Pediatric Acute Respiratory Distress Syndrome (PARDS) is a life-threatening condition with high mortality (33%). Novel therapies to improve mortality in this condition are critical. Multiple retrospective studies from our group and others have demonstrated an association between early enteral nutrition (EEN) and decreased mortality in children with PARDS, but mechanisms for this association are unclear. Crosstalk between the lung and gut microbiome is a potential mechanism by which EEN may reduce PARDS mortality. Diet can rapidly alter the relative abundance of beneficial butyrate-producing commensal gut bacteria to increase fecal butyrate. In animal models of ARDS, butyrate pre-treatment decreases lung inflammation and injury. We hypothesize, that in severe PARDS, EEN increases relative abundance of butyrate producing gut commensals, thereby increasing fecal butyrate concentrations with downstream effects of reducing acute lung inflammation and injury. EEN is a novel pathway to improve clinical outcomes in these children. Precise nutritional therapies which are titrated and targeted to preserve gut commensal organisms and increase butyrate concentrations may offer great potential for improving outcomes of pediatric ARDS. Future clinical trials are needed to prospectively test if ‘microbiome therapy’ can improve ventilator free days and mortality from pediatric ARDS.

Poster Titles

Poster Session 1

Poster	Author	Abstract Title
1	Meredythe Durckel	Testing The Activity of A Novel Copper-Dependent Antimicrobial Against Streptococcus Pyogenes
2	Jordan Gunning	The Immune Phenotype of Radiation Damaged Salivary Glands
3	Ian Kline	Human Cytomegalovirus Promotes de novo Phosphatidylcholine Synthesis
4	Ann Macy	Role of GILT and MHC class II in melanoma cells on regulating the anti-tumor immune response
5	Melissa Moy	Complex Interplay Between UL136-33kDa and UL135 Coordinate the Switch Between Human Cytomegalovirus Latency and Reactivation
6	Stephanie Tribble	The Innate Immune Sensor, AIM2, Promotes Type 2 Inflammation by Enhancing Tuft Cell Differentiation in the Small Intestine
7	Stephanie Rosenberg	Informing Influenza Vaccine Design: Effect of Cytokine Neutralization on T cell Proliferation
8	Matthew Ollerton	Germinal Center B Cells Augment HIV Replication in T Follicular Helper Cells

Poster Session 2

Poster	Author	Abstract Title
9	Trevor Tankersley	Dysregulation of the Immune System: The Role of the Gut Microbiota in Immune System Function
10	Bao Thai	Overcoming the Barriers in Differentiation of Stem Cells to B Lymphocytes
11	Isabelle Tobey	HPV Inhibition of the cGAS/STING Pathway
12	Dakota Reinartz	Investigating the Role of AIM2 in Head and Neck Squamous Cell Carcinoma
13	Maxwell Wegner	Human Cytomegalovirus Alters Extracellular Metabolic Programming
14	David Williams	Single Cell Sequencing Identifies Protein Arginine Methyltransferase 1 as A Regulator Of Persistent HPV Infection In Primary Cervical Keratinocytes
15	Maria Love	Genetic Characterization of HIV-1 vpr Gene from HIV-Infected Older Individuals on Long Term Antiretroviral Therapy
16	Sanna Loppi	Brain Metabolism Is Altered For At Least 12 Weeks Following Stroke
17	Emily Merrit	Using 3-D, whole neuron reconstructions to probe neuronal capabilities for parasite clearance

Organizing Committee

Michael Johnson, PhD (chair)

Justin Wilson, PhD

Danielle Becket

Christine Bradshaw

Jennifer Bray

Christopher Coplen

Tonya Fotheringham

Vanessa Gonzalez

Polly Haffner

Henrik O'Brien

Dakota Reinartz

Tyler Ripperger

Kristen Zarrella

Further acknowledgement is made for the grateful assistance of:

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Poster Judges

Lucas D'souza

Martha Dua-Awereh

Rob Jackson

Mladen Jergovic

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Rebekah Mokry

Maria Rendon

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