St. Jude Children's Research Hospital
ALSAC • Danny Thomas, Founder

Pediatric Infectious Diseases Society

17th Annual
St. Jude/PIDS
Pediatric Infectious Diseases Research Conference

Program & Abstracts

March 9–10, 2018

Marlo Thomas Center
For Global Education And Collaboration
St. Jude Children's Research Hospital

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17th Annual St. Jude/PIDS
Pediatric Infectious Diseases Research Conference
St. Jude Children's Research Hospital
March 9-10, 2018
Memphis, Tennessee

St. Jude Children's Research Hospital
Department of Infectious Diseases
262 Danny Thomas Place, Mailstop 320
Memphis, TN 38105-3678

Pediatric Infectious Diseases Society
1300 Wilson Boulevard
Suite 300
Arlington, VA 22209

March 7, 2018

Dear Colleagues:

On behalf of St. Jude Children's Research Hospital and the Pediatric Infectious Diseases Society, we welcome you to Memphis, Tennessee for the 17th Annual St. Jude/PIDS Pediatric Infectious Diseases Research Conference!

This year, nearly 200 faculty, fellows, and students will attend the meeting. The program features a "Frontiers in Infectious Diseases" symposium, with presentations from leading investigators in infectious diseases and microbiology. In addition, a series of career development workshops will be available, including sessions on career paths in public health, private practice, investigative research, and outcomes research. Perhaps most important, attendees will have an opportunity to present their original research to colleagues in pediatric infectious diseases, with over 60 novel case reports and original science abstract publications in basic and clinical science, epidemiology, laboratory diagnostics, and health outcomes. We are also pleased to announce that, through the generosity of St. Jude Children's Research Hospital, these original science abstracts will be published as a supplement in the *Journal of the Pediatric Infectious Diseases Society* (JPIDS) along with a summary of conference proceedings and selected invited reviews.

We look forward to an excellent meeting and to meeting each of you over the next few days.

Sincerely,

Elaine I. Tuomanen, MD, FPIDS
Chair, Department of Infectious Diseases
St. Jude Children's Research Hospital

Buddy Creech, MD, MPH, FPIDS
Chair, Research Affairs Committee,
Pediatric Infectious Diseases Society
Director, Vanderbilt Vaccine Research Program

Research Affairs Committee

Anne Blaschke, MD, PhD
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William Muller, MD, PhD, FPIDS
Sallie Permar, MD, PhD
Octavio Ramilo, MD, FPIDS
David Rosen, MD, PhD
Jessica Snowden, MD
Stephen Spector, MD
Joshua Wolf, MBBS

PROGRAM AT A GLANCE

Wednesday, March 7	
6:00 – 8:00 P.M.	**Satellite Registration** Westin Memphis Beale Street Hotel
Friday, March 9	**Marlo Thomas Center for Global Education and Collaboration (GEC)**
7:30 A.M.	**Breakfast**
8:00 A.M.	**Welcome and Announcements** Elaine Tuomanen, MD, St. Jude Children's Research Hospital Buddy Creech, MD, MPH, Vanderbilt University School of Medicine Paul W. Spearman, MD, Cincinnati Children's Hospital Medical Center
8:10 – 11:30 A.M.	**Frontier Lectures in Infectious Diseases** *Using systems serology to understand mother to child antibody transfer*—Galit Alter, PhD, Harvard University *Novel therapeutic strategies from study of host-pathogen interaction*—George Liu, MD, PhD, Cedars-Sinai Medical Center *The Future of HIV Therapeutics; Towards Sustained ART-Free Remission and Cure*—Deborah Persaud, MD, Johns Hopkins University *Multidrug resistant Enterococci, a problem foreordained by events of the Paleozoic era*—Michael Gilmore, PhD, Harvard University
11:30 A.M. – 12:30 P.M.	**2018 John H. Erskine Lecture in Infectious Diseases** *Listeria monocytogenes*: *From basic mechanisms of pathogenesis to cancer immunotherapy*—Daniel Portnoy, PhD, University of California, Berkeley
12:30 – 1:30 P.M.	**Lunch – Roundtable with Faculty**

1:30 – 4:00 P.M.	**Oral Abstract Presentations by Fellowship Award Recipients** • *Dissecting mechanisms of granulomatous response in chronic bacterial infections*—Trung Pham, MD, PhD, Stanford University, Stanford, CA • *Development of a consensus DNA vaccine for seasonal H3N2 influenza viruses*—Amelia Keaton, MD, The Children's Hospital of Philadelphia, PA • *Antibiotic exposure and progression of respiratory viral infections in transplant recipients*—Chikara Ogimi, MD, Seattle Children's Hospital, Seattle, WA • *IgE-mediated allergic stimulation inhibits monocyte antiviral responses to alter virus-induced T cell priming by influenza and rhinovirus*—Regina Rowe, MD, University of Texas Southwestern Medical Center, Dallas, TX **Oral Abstracts by Meeting Attendees** • **(19131)** *Maternal immunization with* ΔgD-2 *protects neonatal mice against lethal HSV challenge*—Carol Kao, MD, Albert Einstein College of Medicine • **(19115)** *Pediatric Unexplained Encephalitis Study (PUES): Next-generation sequencing as a diagnostic modality*—Julia Haston, MD, Emory University • **(19122)** *CRISPR screening reveals TNF as a mediator of IFN-g induced macrophage death countered by autophagy*—Anthony Orvedahl, MD, PhD, Washington University • **(19164)** *Genotypic and phenotypic diversity within the neonatal HSV-2 population*—Lisa Akhtar, MD, PhD, Children's Hospital of Philadelphia • **(19110)** *Correlating infection phenotypes with tissue viral distribution in congenital Zika virus infection*—Emma Mohr, MD, PhD, University of Wisconsin
4:00 – 5:00 P.M.	**Poster Presentations**
6:30 P.M.	**St. Jude/PIDS Reception and Dinner** National Civil Rights Museum

Saturday, March 10	Marlo Thomas Center for Global Education and Collaboration (GEC)
7:30 A.M.	**Breakfast**
8:00 – 9:30 A.M.	**Keynote Address** *Life after respiratory syncytial virus*—Fernando Polack, MD, Fundacion, INFANT, Buenos Aires, Argentina **Luminary in Pediatric Infectious Diseases** *Vaccines and vaccine safety*—Larry K. Pickering, MD, Emory University School of Medicine
9:45 A.M. – 12:30 P.M.	**Career Development Presentations** *The changing face of Peds ID*—Janet Gilsdorf, MD, University of Michigan, Ann Arbor **Career Pathways Brief Talks** Introduction: Scott James, MD, University of Alabama, Birmingham and Jessica Snowden, MD, University of Arkansas for Medical Sciences • Basic Science: George Liu, MD, PhD, Cedars-Sinai Medical Center • Outcomes Research: Grace Lee, MD, MPH, Stanford University • Private Practice: Stephanie Stovall, MD, Golisano Children's Hospital of Southwest Florida • Public Health at a Local Level: Kari Simonsen, MD, University of Nebraska **Career Panel Discussion** (11:30 AM – 12:30 PM)
12:30 – 2:00 P.M.	**Lunch and Career Paths Breakout Discussions** **K Awards/Loan Repayment Program** Scott James, MD and Jessica Snowden, MD **How Jobs are Pieced Together: Elucidating the Black Box of Peds ID FTE's** Kari Simonsen, MD **How Grants are Graded: Understanding the NIH Criteria** Betsy Herold, MD, Albert Einstein College of Medicine and John Williams, MD, Children's Hospital of Pittsburgh
2:00 – 3:30 P.M.	**Global Health Session** Moderators: Miguela Caniza, MD, St. Jude Children's Research Hospital and Miriam Laufer, MD, University of Maryland School of Medicine Part 1: Reflections on careers in global health Vaccines: Edwin Asturias, MD, University of Colorado —Polio and Fernando Polack, MD – RSV Part 2: Global health jeopardy Combination of trivia and cases collected from local and international settings

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General Information

Location of Conference

Marlo Thomas Center for Global Education and Collaboration (GEC)
St. Jude Children's Research Hospital
262 Danny Thomas Place
Memphis, TN 38105-3678

Headquarter Hotel Information
Westin Memphis Beale Street Hotel
170 Lt. George W. Lee Avenue
Memphis, TN 38103
(901) 334-5900

Registration Hours – Marlo Thomas Center for Global Education and Collaboration – St. Jude Children's Research Hospital campus

Wednesday, March 7: 6:00 – 8:00 p.m. (Satellite/Wine Reception – Tennessee Ballroom at the Westin Hotel)
Friday, March 9: 7:00 a.m. – 5:30 p.m.
Saturday, March 10: 7:00 a.m. – 4:00 p.m.

Poster Session

The Poster Session will take place on Friday, March 9, from 4:00 to 5:00 p.m. in the foyer of the GEC building.

Travel Stipend Information

Recipients of travel stipends must check in at the registration desk each day to verify attendance. Checks can be picked up at the registration desk on the last day of the conference.

Mothers' Room

St. Jude Children's Research Hospital offers several Mothers' Rooms around the campus for conference registrants. These rooms can be used to breastfeed your infant or to express and store milk while at the conference. The closest rooms to use during conference hours are the Mothers' Room located at the Chili's Care Center and Danny Thomas Research Center. Please seek staff assistance at the Registration Desk before attempting to use this facility.

Shuttle Bus Service

Shuttle bus service is available for registrants to St. Jude Children's Research Hospital from the Westin Memphis Beale Street Hotel. Registrants are responsible for providing their own transportation to any other destination while in Memphis. The following shuttle schedule will be posted at the registration desk.

Date	Time	Pick-up Site	Destination
Friday, March 9	7:00, 7:15, and 7:30 a.m.	Westin Memphis Beale Street Hotel	St. Jude
	5:15 and 5:30 p.m.	St. Jude	Westin Memphis Beale Street Hotel
	6:00 p.m.	St. Jude	National Civil Rights Museum
	6:15 and 6:45 p.m.	Westin Memphis Beale Street Hotel	National Civil Rights Museum
	8:30 and 9:00 p.m.	National Civil Rights Museum	Westin Memphis Beale Street Hotel and St. Jude
Saturday, March 10	7:00, 7:15 and 7:30 a.m.	Westin Memphis Beale Street Hotel	St. Jude
	3:30 p.m.	St. Jude	Westin Memphis Beale Street Hotel and Airport

Friday Night Event

There will be a tour and dinner at the National Civil Rights Museum on Friday, March 9. The museum is located at 450 Mulberry Street and is within a short distance from the Westin Memphis Beale Street Hotel**.** A virtual tour of the museum will be taken shortly upon arrival. Dinner will begin after the tour. Roundtrip shuttle bus services will be provided from the Westin and St. Jude. One bus will depart from St. Jude at 6:00 p.m. Another bus will pick up from the Westin at 6:15 and 6:45 p.m. Both buses will return to St. Jude and the Westin beginning at 8:30 and 9:00 p.m.

Internet Access

St. Jude Children's Research Hospital offers wireless Internet access through HopeNet.

Staff Contact Information
For onsite assistance, please contact Christy Phillips (202) 306-9545 or Brandi Kirby (901) 595-2641.

Continuing Medical Education Information

Educational Objectives
After attending this educational conference, you should be able to:

- Discuss and use new interventions and vaccines for pediatric and prenatal infections
- Compare and contrast different career options in pediatric infectious diseases

Please note that session objectives will be presented during the conference as appropriate.

Accreditation Statement

This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of St. Jude Children's Research Hospital and the Pediatric Infectious Diseases Society. St. Jude Children's Research Hospital is accredited by the ACCME to provide continuing medical education for physicians.

Credit Designation
St. Jude Children's Research Hospital designates this live activity for a maximum of 13.5 *AMA PRA Category 1 Credits*™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Acknowledgement
This live activity is supported by an educational grant from Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc. The opinions expressed at this conference are those of the faculty and do not necessarily reflect the opinions of the CME provider, joint provider, or commercial supporter.

Evaluation and Credit Certificates
To claim *AMA PRA Category 1 Credit™* or attendance credit for this activity, you must attend the sessions and complete the evaluation by following the instructions below:

- Go to cme@stjude.org
- Sign in using the email address you used when registering for this conference and your password (if you do not remember your password or have not previously used this system, click "forgot my password" and follow the instructions; St. Jude employees should choose "Sign in with your St. Jude ID")
- Click the MyCME button on the top right of your screen and choose Evaluations and Certificates
- Click "Complete Evaluation" by *St. Jude/PIDS Pediatric Infectious Diseases Research Conference* to complete the evaluation for this activity
- You will then be returned to the Evaluations and Certificates screen to print, download, or email your certificate.

If you have questions about claiming your CME or attendance certificate, please contact us at cme@stjude.org. If you registered after February 28, 2018, or on site, it may take 48 hours to have your information added to the CME system so that you may claim credit.

Disclosure of Financial Relationships
All individuals in a position to control the content of this CME activity (such as faculty, presenters, and planners) were asked to complete a statement regarding all relevant financial relationships between themselves or their spouse/partner and any commercial interest (defined by the ACCME as "any entity producing, marketing, re-selling, or distributing health care goods and services consumed by, or used on, patients"). St. Jude CME has reviewed and resolved any conflicts of interest that were identified.

All relevant relationships are disclosed on the CME Addendum; no other speakers, planners, or other individuals with control over content have disclosed relevant financial relationships with commercial interests for themselves or their spouse/partner.

Session 1. Frontiers in Infectious Diseases

Friday, 8:00 – 11:30 a.m.

GEC Auditorium

Using systems serology to understand mother to child antibody transfer
GALIT ALTER, Harvard University, Boston, Massachusetts

Novel therapeutic strategies from study to host-pathogen interaction
GEORGE LIU, Cedars-Sinai Medical Center, Los Angeles, California

Major pathogens utilize broad arrays of strategies to overcome host defenses and cause infection. In this presentation, I will describe the pathogenic mechanisms associated with several bacterial factors, including the staphylococcal golden pigment, PBP2a, and GBS hyaluronidase. I will demonstrate how interaction of the pathogenic factors with host immune cells leads to worse infectious outcome and will suggest novel approaches to combat infections based on knowledge of the molecular mechanisms.

The future of HIV therapeutics, towards sustained ART-free remission and cure
DEBORAH PERSAUD, Johns Hopkins University, Baltimore, Maryland

A latent reservoir for HIV-1 resides within memory CD4+ T cells of HIV-1-infected individuals. This stable reservoir precludes virus eradication and cure with current antiretroviral treatment (ART) strategies, rendering antiretroviral treatment life-long. However, a single case of HIV-1-cure in an infected adult, the "Berlin patient", who following treatment of acute myeloid leukemia that included bone marrow transplantation with HIV-1-resistant, CCR5delta32 homozygous cells, has remained off ART for 10 years without return of replicating HIV-1, has transformed the field of HIV-1-therapeutics towards a goal of ART-free HIV-1 remission and cure for infected individuals. Similarly, the single case of 27 months of ART-free remission in a perinatally-infected child, the "Mississippi baby, following very early ART administered within 48 hours of birth, has provided proof-of-concept for ART-free remission in perinatal infection. The novel treatment strategies under clinical investigation towards ART-free remission and cure in pediatric and adult populations will be discussed. The mechanisms underlying the recalcitrant nature of the latent reservoir and their differences in perinatal and adult infection will also be discussed.

Multidrug resistant enterococci, a problem foreordained by events of the Paleozoic era
MICHAEL GILMORE, Harvard University, Boston, Massachusetts

Enterococci are among the most widely distributed core components of gut flora in animals from invertebrates and insects to mammals. This led us to speculate that an ancestral *Enterococcus* colonized the last common ancestor, and was vertically disseminated as new host species evolved. Despite being numerically minor constituents of the gut microbiota, enterococci emerged among the vanguard of multidrug resistant hospital adapted pathogens. Interestingly this happened twice: in *Enterococcus faecalis*, and in the distantly related species *E. faecium*. This raises two questions: 1) What are the core properties of enterococci that make them nearly universal components of gut consortia of such a diverse range of animals? and 2) Why, among the great diversity of gut microbes, did enterococci repeatedly emerge to become leading causes of multidrug resistant hospital acquired infection? With antibiotic resistance now a leading global public health threat, there is a compelling need to understand the underlying biology and genetics that led to their hospital adaptation.

To determine the core traits of enterococci that both enable them to inhabit animals with diverse gut physiologies and diets, and predisposed them to adapt and proliferate in the modern hospital ecology, we selected 25 enterococcal species representing all major phylogenetic branches of the genus. We examined them in detail for phenotype, genotype, and where possible, correlated that with host association. We further compared these traits to those of both commensal and multidrug resistant strains of the most common human

associated species, *E. faecalis* and *E. faecium*. We found that the enterococci acquired the ability to withstand episodic desiccation and starvation, among other stressors, and that speciation is largely driven by changing carbohydrates available in the gut of new hosts. Calibration of divergence indicates that enterococci arose commensurate with the terrestrialization of animals, and parallels their radiation, including gaps as occurred during the Permian Extinction. In adapting to cycles of deposition on land, the enterococci acquired traits that positioned them well for survival and adaptation to the modern hospital environment.

Session 1b. 2018 John H. Erskine Lecturer in Infectious Diseases
Friday, 11:30 a.m. – 12:30 p.m.

GEC Auditorium

***Listeria monocytogenes*: From basic mechanisms of pathogenesis to cancer immunotherapy**
DANIEL PORTNOY, University of California, Berkeley

Listeria monocytogenes is an intracellular pathogen that is able to escape from a phagosome, grow rapidly in the host cell cytosol and exploit the host actin cytoskeleton to spread from cell-to-cell without leaving the intracellular environment. This provides a cell biological explanation for the requirement of cell-mediated immunity to clear the infection; i.e., T-cells must be generated that can recognize infected cells and kill the cell. Infection with *L. monocytogenes* leads to the development of a robust cell-mediated immune response which provides the rationale for using attenuated strains of *L. monocytogenes* as vaccine vectors. Recombinant strains that express and secrete tumor antigens can stimulate the immune system to recognize and kill the tumor cells.

Lunch – Roundtable with Faculty
Friday, 12:30 – 1:30 p.m.

GEC Foyer and Board Room

Session 2. Oral Presentations by PIDS and PIDS-St. Jude Fellows and Meeting Attendees
Friday, 1:30 – 4:00 p.m.

GEC Auditorium

Dissecting mechanisms of granulomatous response in chronic bacterial infections
TRUNG PHAM, Stanford University, Stanford, California

Infectious granulomas are dynamic microstructures comprised of immune cells and pathogen and are thought to be an important immune response to control chronic latent infections. Mounting evidence suggests granulomatous formation can also serve as a mechanism for pathogen persistence. To study function and regulation of granulomatous response in chronic bacterial infection, we use a latent murine *Salmonella* infection model with fully virulent bacteria. We find granuloma morphologic maturation temporally coincides with resolution of T-cell IFNγ response and reduction of bacterial burden to low chronic stage levels. In lymphoid tissues such as the spleen of chronically infected mice, *Salmonella* bacilli persist within granuloma microstructures that are predominated by CD11b+CD11c+Ly6C+ myeloid cells. Flow cytometric analyses of splenocytes identify CD11b+CD11c+Ly6C+ cells as granuloma macrophages. *Salmonella* infection dynamically modulates granuloma macrophage number, phenotype and functions. To define the mechanisms regulating infectious granulomas during chronic *Salmonella* infection, we disrupt TNFα signaling in infected mice using anti-TNFα monoclonal antibody. TNFα neutralization markedly alters CD11bCD11cLy6C+ macrophage polarization and granuloma cellular organization. Furthermore, mice treated with anti-TNFα antibody exhibit increased bacterial tissue burden and concurrent expansion of IFNγ-producing T cells. Together our preliminary findings support a model in which TNFα signaling regulates granuloma organization and controls bacterial persistence in chronic *Salmonella* infection via a CD11b+CD11c+Ly6C+ macrophage-dependent mechanism.

Development of a consensus DNA vaccine for seasonal H3N2 influenza viruses
AMELIA KEATON, The Children's Hospital of Philadelphia, Philadelphia, Pennsylvania

Seasonal influenza is a significant cause of illness worldwide, with children being particularly vulnerable to illness and complications. Available flu vaccines typically induce strain-specific immune responses and therefore must be updated and administered annually. Here we report on the development of a novel seasonal H3N2 influenza vaccine based on synthetic DNA-encoded hemagglutinin (HA) antigens. Initial studies using an animal model indicate that this vaccine platform induces a broadly reactive immune response to a variety of H3N2 viral strains.

Antibiotic exposure and progression of respiratory viral infections in transplant recipients
CHIKARA OGIMI, Seattle Children's Hospital, Seattle, Washington

The objective of this study was to investigate the effect of antibiotic use on the disease progression of subsequent respiratory viral infections (parainfluenza virus, respiratory syncytial virus or human metapneumovirus) in allogeneic transplant recipients. We identified 90 patients (84 adults and 6 children) with first respiratory viral infection during the initial 100 days post-transplantation (6/2008-2/2016). Among these, 33 progressed to lower respiratory tract disease (LRTD). Number of antibiotic-days in the 21 days before viral onset was associated with progression to LRTD after adjusting for neutropenia frequency, steroid use, and either lymphopenia or monocytopenia in Cox proportional hazard models, but use of specific antibiotic classes was not. Cumulative antibiotic exposure immediately prior to viral onset appears to be an important risk factor for the disease progression for some respiratory viruses in transplant recipients.

IgE-mediated allergic stimulation inhibits monocyte antiviral responses to alter virus-induced T cell priming by influenza and rhinovirus
REGINA ROWE, University of Texas Southwestern Medical Center, Dallas, Texas

Respiratory virus infections are highly associated with the development and exacerbation of allergic diseases. Deficient antiviral responses by antigen presenting cells, such as monocytes and dendritic cells, are hypothesized as potential mechanisms underlying this link. Using an *ex vivo* model of primary human monocytes, we evaluated the effects of IgE-mediated allergic stimulation on multiple monocyte functions. Allergic stimulation inhibited virus-induced 1) upregulation of MHC I and II, and co-stimulatory molecules CD80 and CD86, 2) type I interferon release, and 3) CD4 Th1 priming after both influenza A virus and rhinovirus (RV) exposure. Interestingly after RV-exposure, monocytes promoted significant Th2 differentiation, which was further enhanced by IgE-mediated stimulation.

These data suggest that allergic stimulation impairs monocyte innate antiviral responses and results in altered virus-driven T cell differentiation. Modulation of monocyte antiviral inflammatory responses represents another mechanism by which allergic stimulation may contribute to virus-induced exacerbations of allergic disease. An understanding of the molecular mechanisms involved in these processes is ongoing and may identify potential therapeutic targets to prevent allergic disease development, progression, or exacerbation.

Maternal immunization with ΔgD-2 protects neonatal mice against lethal HSV challenge
CAROL KAO, Albert Einstein College of Medicine, Bronx, New York

We hypothesize that maternal transfer of antibodies (Abs) that elicit antibody dependent cellular cytotoxicity (ADCC) will protect neonates from HSV infection. To test this, we compared immunogenicity and efficacy of two vaccine candidates: (i) a single-cycle virus deleted in glycoprotein-D (ΔgD-2) that induces high-titer Abs that activate the Fc gamma receptor (FcγR) to elicit ADCC; and (ii) recombinant gD-2 protein with Alum and MPL adjuvants (rgD-2), which induces primarily neutralizing Abs, but not ADCC. ΔgD-2 completely protects adult mice against vaginal or skin challenge with clinical isolates of HSV-1 or 2 and prevents viral latency; rgD-2 provides partial protection, does not prevent latency and failed in clinical trials (ELife, 2014, JCI Insight, 2016, JID, 2017). Methods: C57Bl/6 female mice were vaccinated with ΔgD-2 or control uninfected cell lysates or with rgD-2. Mice were mated one week after and pups challenged intranasally with

a lethal dose of HSV on days 1, 7 or 14. To distinguish the roles of transplacental vs. colostrum Abs, mother-pup pairs were switched at birth. Results: Pups born and nursed by ΔgD-2-immunized mice were protected compared to rgD-2 ($p=0.01$, Day 7) or control-immunized mice ($p< 0.001$, Days 7 or 14, Fisher's exact test). Maternal ΔgD-2 immunization also prevented or decreased viral dissemination to liver, lung, kidney, brain or trigeminal ganglia. Similar levels of HSV-specific Abs were detected in serum of neonates born to ΔgD-2 or rgD-2-immunized mice, but only the former exhibited ADCC. Protection decreased if pups were challenged on Day 1 or were nursed with control mice and correlated with a decline in total HSV-specific and ADCC Abs. Studies are in progress to compare age-related changes in FcγR expression and ADCC activity. Conclusion: Maternal ΔgD-2 vaccination provides significant protection against neonatal infection, which correlates with ADCC.

Pediatric Unexplained Encephalitis Study (PUES): Next-generation sequencing as a diagnostic modality

JULIA HASTON, Emory University, Atlanta, Georgia

Background: Encephalitis can be associated with focal and global neurologic dysfunction resulting in significant morbidity and mortality in children. Although viruses are often implicated, an etiology is not identified in the majority of cases. Next-generation sequencing (NGS) is a high-throughput metagenomics-based sequencing technique that may enhance the detection of novel or low frequency pathogens.

Methods: Hospitalized immunocompetent patients aged 6 months-18 years with encephalitis of unidentified etiology were eligible for enrollment. Demographic, historical, and clinical information was obtained using a standardized questionnaire. Serum or plasma and cerebrospinal fluid (CSF) samples were subjected to NGS. Pathogens were identified by comparing the nucleic acid sequences from the samples with online databases of sequences.

Results: Twenty patients aged 6 months-17 years and 50% female were enrolled from 2013 to 2017. NGS identified non-human nucleic acid sequences of significant frequency in 4 patients. Those identified by NGS were Cladosporium bantiana in CSF, Mycoplasma bovis in CSF, and Parvovirus B19 in both plasma and CSF, as well as tobacco mosaic virus which was a presumed contaminant. Two organisms diagnosed by conventional CSF PCR, Neisseria meningitidis and Balamuthia mandrillaris, were identified in the NGS data in retrospective analysis. One additional patient was found to have positive IgM serology for LaCrosse virus, but molecular diagnosis was not achieved.

Conclusion: We describe a prospective cohort analysis evaluating NGS as a diagnostic tool for children with unexplained encephalitis. Limitations of NGS in this study included detection of contaminants and difficulty distinguishing low levels of pathogen nucleic acid sequences from background. However, multiple putative pathogens were identified suggesting that NGS could facilitate pathogen discovery and diagnosis.

CRISPR screening reveals TNF as a mediator of IFN-g induced macrophage death countered by autophagy

ANTHONY ORVEDAHL, Washington University, St. Louis, Missouri

Optimal host immunity rests on a delicate balance between sufficient control of microbial proliferation and limiting injury from the ensuing inflammatory response. Host determinants of this balance are incompletely understood. The cytosolic pathway of autophagy performs important immune functions including degradation of invading pathogens and limiting inflammation. We previously showed that mice lacking autophagy genes in myeloid cells developed hyperinflammation, even under naive conditions. Cells and tissues from autophagy-deficient mice exhibited elevated transcriptional signatures for interferon gamma (IFN-g) and tumor necrosis factor (TNF) pathways. Importantly, the mechanisms driving hyperinflammation in these mice remain obscure. Turning to an in vitro system, we found that cells with CRISPR deletion of autophagy genes were hypersensitive to IFN-g induced cell death. Genome-wide CRISPR screening revealed that TNF pathway genes are required to mediate IFN-g induced death of autophagy gene-deficient cells. Further screen validation and mechanistic experiments will be presented. These studies have potential implications for

improving clinical outcomes in inflammatory states such as sepsis and infections that are controlled by IFN-g and TNF.

Genotypic and phenotypic diversity within the neonatal HSV-2 population
LISA AKHTAR, The Children's Hospital of Philadelphia

Neonates infected with herpes simplex virus (HSV) at the time of birth can have different clinical courses. Approximately half display manifestations limited to the skin, eyes, or mouth (SEM disease, 45%). However, others develop invasive infections that spread systemically (disseminated, 25%) or to the central nervous system (CNS, 30%); both of which are associated with significant morbidity and mortality. The viral and/or host factors that predispose a neonate to invasive forms of HSV infection are not known. To define viral diversity within the neonatal population we evaluated ten HSV-2 isolates cultured from neonates with a range of clinical presentations. To assess viral fitness independent of host immune factors, we measured viral growth characteristics of each isolate in cultured cells and found that they displayed diverse *in vitro* phenotypes. Isolates from neonates with CNS disease were associated with larger average plaque size and enhanced spread through culture, with isolates derived directly from the cerebrospinal fluid (CSF) exhibiting the most robust growth characteristics. We then sequenced the complete viral genomes of all ten neonatal HSV-2 isolates. We found extensive inter-host variation between isolates distributed throughout the HSV-2 genome. Furthermore, we assessed intra-host variation and found that each HSV-2 isolate contained minority variants, with two viral isolates containing ten-fold higher levels of allelic variation than other neonatal isolates or comparable adult isolates. Several HSV-2 proteins including glycoprotein I (gI, US7), gK (UL53), and viral proteins UL20, UL24, and US2 contained variants that were found only in neonatal isolates associated with CNS disease. These genes encode viral proteins known to contribute to cell-to-cell spread and neurovirulence in mouse models. This study represents the first-ever application of comparative pathogen genomics to neonatal HSV disease.

Correlating infection phenotypes with tissue viral distribution in congenital Zika virus infection
EMMA MOHR, University of Wisconsin, Madison, Wisconsin

Congenital Zika syndrome is a constellation of birth defects that occurs following in utero infection with Zika virus (ZIKV). We hypothesized that higher viral loads and broader viral tissue distribution in the fetus was associated with tissue histopathology and the development of ZIKV-associated birth defects. We developed a nonhuman primate (NHP) model that accurately models human disease with prolonged maternal viremia, vertical transmission, fetal tissue dissemination and birth defects. We infected rhesus macaques with 10^4 PFU of a Puerto Rican ZIKV isolate (PRVABC59) in the first trimester and assessed viral tissue distribution and birth defects in their offspring. We delivered fetuses in the second trimester (n=4) and near term in the third trimester (n=5) to evaluate viral tissue distribution and birth defects. We assessed liveborn infants with a postnatal evaluation that included an ophthalmologic exam, hearing evaluation, electroretinography, visually-evoked cortical potentials, brain magnetic resonance imaging and neurodevelopmental assessment. Viral RNA was quantified in fetal and infant tissues by QRT-PCR, and for infectious virus by positive- and negative-strand RNA in-situ hybridization. Two dams experienced adverse pregnancy outcomes, with a second trimester stillbirth and a miscarriage near term. Three of 5 infants delivered near term underwent comprehensive postnatal evaluations; one of these experienced respiratory distress requiring noninvasive positive pressure ventilation. One stillborn infant had severe ocular defects consisting of a choroidal coloboma, anterior segment dysgenesis and retinal dysplasia, along with widely disseminated ZIKV infection. Thus, from a total of nine pregnancies evaluated in the study, two had adverse clinical outcomes. Fetal and infant viral tissue distribution analyses are underway to positively correlate viral tissue distribution and ZIKV-associated birth defects.

Session 3. Poster Session

Friday, 4:00 – 5:00 p.m.

GEC Foyer

Session 4. Keynote Address

Saturday, 8:00 – 8:45 a.m.

GEC Auditorium

Life after respiratory syncytial virus
FERNANDO POLACK, Fundacion, INFANT, Buenos Aires, Argentina

Respiratory syncytial virus (RSV) is the main cause of hospitalization in infants worldwide. Every year, more than 3 million infants and young children experience severe presentations and an estimated 100,000 children die around the world due to RSV. However, this serious situation is likely to change soon. A number of preventive interventions against the virus are undergoing evaluation in clinical trials and the landscape of respiratory infections may be very different in the next decade.

In this presentation, we will discuss the potential impact of RSV prevention on pediatric hospitalizations, explore whether the virus will be replaced in the respiratory tract by other pathogens, consider the consequences of RSV vaccines for recurrent wheezing and asthma, identify subpopulations that may respond poorly to vaccination, and estimate the impact of a successful vaccine on infant mortality at the hospital and in the community in the developing world.

Session 5. Luminary in Pediatric Infectious Diseases

Saturday, 8:45 – 9:30 a.m.

GEC Auditorium

Vaccines and vaccine safety
LARRY PICKERING, Emory University, Atlanta, Georgia

Below are the following learning objectives:

- Clarify differences between FDA licensure of and ACIP recommendations for vaccines
- Examine why differences between the two agencies exist
- Review vaccine successes
- Highlight lessons learned from making vaccine recommendation
- Emphasis the role of mentoring in each step of the vaccine development process

Session 6. Career Development Presentation

Saturday, 9:30 –10:15 a.m.

GEC Auditorium

The changing face of Peds ID
JANET GILSDORF, University of Michigan, Ann Arbor, Michigan

Infections have plagued humans since the dawn of time and early physicians recognized patterns of infection and attempted to cure them with remedies we would today consider useless. Eventually, through excellent biomedical research, ID doctors have acquired valuable tools to prevent and/or treat many infections.

As our mastery of the worst infectious diseases evolved, so did the spectrum of research conducted by ID physicians. While we still have much to learn about the basic pathophysiology of, and host responses to,

infections, we also need to know about management of highly immune-compromised patients, effective utilization of medical therapies, and methods to protect patients in our hospitals from infection. To this end, many new opportunities exist in the ways PID physicians are trained, and ithe positions they assume following fellowship.

Session 7. Career Pathways Brief Talks/Panel Discussion

Saturday, 10:15 a.m. – 12:30 p.m.

GEC Auditorium

The career pathways session will bring together several faculty members with differing career paths. Each panelist will provide a short talk that introduces the audience to their field and touch on the pros/cons of this career option. After all panelists have given their short talk, they will join each other on stage and answer questions related to career development. There will be a few facilitated questions, but the majority of the discussion will be driven by attendees' questions.

- Basic Science: **GEORGE LIU**, Cedars-Sinai Medical Center, Los Angeles, California
- Outcomes Research: **GRACE LEE**, Stanford University, Stanford, California
- Private Practice: **STEPHANIE STOVALL**, Golisano Children's Hospital of Southwest Florida
- Public Health at the Local Level: **KARI SIMONSEN**, University of Nebraska, Omaha, Nebraska

Session 8. Career Paths Breakout Discussions

Saturday, 12:30 – 2:00 p.m.

GEC Conference Rooms

K Awards/Loan Repayment Program
SCOTT JAMES, University of Alabama, Birmingham and JESSICA SNOWDEN, University of Arkansas for Medical Sciences, Little Rock, Arkansas

In this session, faculty will discuss the structure and requirements of the NIH's K series awards (Career Development), as well as the Loan Repayment Program. In addition, Drs. James and Snowden will provide advice on avoiding specific application pitfalls, tips for highlighting the strengths of your application and answer questions from participants on both programs.

How Jobs are Pieced Together: Elucidating the Black Box of Peds ID FTE's
KARI SIMONSEN, University of Nebraska, Omaha, Nebraska

In this session, faculty will discuss the structure of physician salaries, otherwise known as FTE (Full Time Equivalent). We will provide advice on strategies for building the position you need now and in the future, as well as tips on handling common conflicts and concerns. This will be an interactive discussion with ample opportunities for questions.

How Grants are Graded: Understanding the NIH Criteria
BETSY HEROLD, Albert Einstein College of Medicine and JOHN WILLIAMS, Children's Hospital of Pittsburgh, Pittsburgh, Pennsylvania

In this session, faculty will discuss NIH study section scoring and demonstrate a typical study section discussion of a K award. Topics included will be study section procedures, general approach to reading grants, ways to avoid specific grant pitfalls, and the importance of a clear presentation of your proposed work.

Session 9. Global Health Session
Saturday, 2:00 – 3:30 p.m.

GEC Conference Rooms

Moderators: MIGUELA CANIZA, St. Jude Children's Research Hospital, Memphis, Tennessee, and MIRIAM LAUFER, University of Maryland School of Medicine, Baltimore, Maryland

Part 1: Reflections on careers in global health

- Polio Vaccines – **EDWIN ASTURIAS**, University of Colorado, Aurora, Colorado
- RSV – **FERNANDO POLACK,** Fundacion, INFANT, Buenos Aires, Argentina

Part 2: Global health jeopardy

- Combination of trivia and cases collected from local and international settings

Poster Presentations
Friday, 4:00 – 5:00 p.m.

GEC Foyer

Bacterial

#19089 An outbreak of pediatric brucellosis in Dallas (Presenter: Linda Hassouneh)

#19099 Diagnosis of a spinal epidural abscess in a neonate by Whole-body magnetic resonance imaging (Presenter: Su Jin Joo)

#19100 Ceftazidime for neutropenic fevers in children: Is it time for a change? (Presenter: Muayad Ali)

#19102 Novel RT-PCR assay for detection of *Kingella kingae* in children with osteoarticular infections (Presenter: Theresa Madigan)

#19105 Signs of sepsis do not predict SBI or respiratory viral infection rates in febrile infants ≤90 days (Presenter: Erin Nicholson)

#19106 Disseminated multidrugresistant *A. Baumanii* infection (Presenter Alicia Chang)

#19109 Nasopharyngeal bacterial colonization and AOM in children with RSV acute respiratory infection (Presenter Alejandro Diaz)

#19144 Clinical characteristics of *S. aureus* bloodstream infections in children (Presenter: Gregory Fricker)

#19112 Clinical course and antibiotic dosing in healthy vs non-healthy weight children with osteomyelitis (Presenter: Joel Waddell)

#19116 Microbiology and susceptibility pattern of skin isolates from patients with epidermolysis bullosa (Presenter: Satja Issaranggoon Na Ayuthaya)

#19118 Predisposing characteristics of pediatric patients for ESBL related urinary tract infections (Presenter: Omayma Amin)

#19119 Comparison of bioMerieux and Roche procalcitonin tests in children with community-acquired pneumonia (Presenter: Sophie Katz)

#19127 Impact of drug resistant bacterial colonization in pediatric hematopoietic cell transplant patients (Presenter: Patrick Gavigan)

#19129 Broad based microbial identification and quantification in low sample volume (Presenter: Mridu Sinha)

#19132 Retapamulin activity against mupirocin-resistant isolates from pediatric patients with MRSA (Presenter: Ami Patel)

#19134 Healthcare associated infections among pediatric patients with neutropenia and cancer in Honduras (Presenter: Pamela Zacasa)

#19142 Surveillance of central line-associated bloodstream infections (CLABSI) and non-

CLABSIs in a pediatric cancer center in a low-resource setting (Presenter: Jose Francisco Mendez)

#19143 Microbiological characteristics of *S. aureus* isolates in a pediatric hospital in Bogota, 2008-2017 (Presenter: Ivan Gutierrez)

#19149 Human monocytic ehrlichiosis (HME): Mimicker or cause of hemophagocytic lymphohistiocytosis (HLH)? (Presenter: Shaina Hecht)

#19161 Infections in children with acute myeloid leukemia receiving antimicrobial prophylaxis in a tertiary (Presenter: Jennia Acebo)

Host Responses

#19091 Increase in distal airway mucus-producing Clara-cells during primary *Pneumocystis* infection (Presenter: Sergio Vargas)

#19101 Kikuchi-Fujimoto disease in children: The forgotten disease (Presenter: Muayad Alali)

#19107 The human antibody response to Enterovirus-D68 iblonfection (Presenter: Matthew Vogt)

#19126 Induction of Muc5b and Muc5ac via STAT6/FoxA2 pathway during *Pneumocystis* primary infection (Presenter: Diego Rojas)

#19135 Human thoracic duct lymph carries CXCR5++ PD-1++ Tfh to the peripheral blood (Presenter: Laura Vella)

#19137 Reactive hemophagocytic lymphohistiocytosis associated with re-initiation of highly active antiretroviral therapy (Presenter: Melissa Campbell)

#19152 False positive fourth generation combined HIV antigen/antibody immunoassays in patients (Presenter: Julia Haston)

Other

#19087 Utility of anaerobic and fungal cultures in clinically ill oncologic patients (Presenter: Madan Kumar)

#19088 A genomic snapshot of the *Acinetobacter baumannii* lineages circulating in hospitals from Honduras (Presenter: Julio Zuniga)

#19117 Identification of practices for febrile neutropenia in hemto oncology units in Latin America (Presenter: Mario Melgar)

#19125 Towards a unified fever and neutropenia management protocol for Central America and the Caribbean (Presenter: Mario Melgar)

#19128 Impact of time-lapse b/w onset of fever and first I/V Ab in pediatric cancer patients with FN (Presenter: Saadia Anwar)

#19136 Epi info as a toll in antimicrobial stewardship program at a pediatric hospital in Bogoto, Colombia (Presenter: Ivan Gutierrez)

#19160 Evaluation of posaconazole plasma concentrations in pediatric patients (Presenter: Alyssa Berganini)

#19163 Evaluation of the Accelerate Pheno™ and its potential clinical impact (Presenter: Jack Schneider)

Other Pathogens

#19096 A pathology diagnosis of cutaneous *Rhizopus* species infection in an adolescent with type 1 diabetes (Presenter: Monika Dietrich)

#19097 Osteomyelitis due to *Mycobacterium goodii* in an adolescent - case report (Presenter: Alejandro Diaz)

#19108 Cryptococcal brain access in a six-year-old girl (Presenter: Jeffrey Coote)

#19113 Delayed wound healing due to fusariosis following trauma (Presenter: Janitzio Guzman)

#19114 *Mucor indicus* necrotizing fasciitis in post-stem cell transplant pediatric patient (Presenter: Deborah Bloch)

#19133 Congenital toxoplasmosis in a premature infant, associated with maternal ingestion of deer meat (Presenter: Patricia Pichilingue-Reto)

#19147 Ocular toxocariasis among patients referred for suspicion of retinoblastoma (Presenter: Iván Tinoco Martín)

#19148 Visceral toxocariasis among patients at a pediatric cancer center hospital in Memphis (Presenter: Iván Tinoco Martín)

#19153 Predictive value of noninvasive diagnosis of primary *Pneumocystis* infection in infants at autopsy (Presenter: Sergio Vargas)

#19158 Identification and characterization of *Mycobacterium tuberculosis* effector proteins (Presenter: Sujittra Chaisavaneeyakorn)

#19162 Intestinal parasites in pediatric cancer patients in south of Mexico (Presenter: Enid Alejandra Nava)

Viral

#19069 A unique case of sudden death from pneumonia with superimposed bacterial infection and herpes virus (Presenter: Amit Reddy)

#19083 Hospital acquired viral respiratory infections (HA-VRI) in a tertiary neonatal intensive care unit (Presenter: Claudette Poole)

#19086 Comparison of RSV hospitalized children with and without an underlying medical condition (Presenter: Einas Bataseh)

#19094 Assessing health-related quality of life of pediatric HIV patients in Tabarre, Haiti (Presenter: Aileen Aldrich)

#19103 Acute viral gastroenteritis illness severity by pathogen and rotavirus vaccine status (Presenter: Lubna Hamdan)

#19130 Missed opportunity encounters for early diagnosis of adolscents with HIV infection (Presenter: Lorraine James)

#19146 Use of cyclin-dependent kinase inhibitors to modulate gammaherpesvirus reactivation (Presenter: Joy Hazleton)

#19155 Humoral immune correlates of protection from postnatal CMV acquisition (Presenter: Frances Saccoccio)

#19156 Reception and barriers to long acting injectable ARV for PrEP in adolescent & trainees (Presenter: Aderonke Adefisayo)

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Notes

Notes

Notes

- Entrance Gates
- Patient Only Parking
- Marlo Thomas Center
- Kay Kafe
- Mail Services Center
- Docks

- (A) Danny Thomas/ALSAC Pavilion
- (B) Patient Care Center
- (C) Richard C. Shadyac ALSAC Tower
- (D) Danny Thomas Research Center
- (E) Donald P. Pinkel, MD, Research Tower
- (F) Central Energy Plant
- (G) AutoZone Garage 1
- (H) Incinerator/Hazardous Waste
- (I) Chili's Care Center
- (IA) Kay Research & Care Center
- (J) 545 Danny Thomas Place
- (K) 595 Building
- (L) 567 Danny Thomas Place
- (M) 505 Building
- (N) 305 Building
- (O) Tamer-Rashid (ALSAC HQ)
- (OA) ALSAC HQ Addition
- (OE) Domino's Event Center
- (P) AutoZone Garage 2
- (Q) Kmart St. Jude Life Center
- (R) Longinotti Building
- (S) Barry Building
- (T) Tri Delta Place
- (U) ALSAC Gift Shop
- (V) St. Jude GMP Facility
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- (X) 160 Shadyac Avenue
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- (Z) Data Center
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