

**March 29-30, 2018**

**The University of Iowa**

**College of Public Health**

**Iowa City, Iowa**

**Great Plains Emerging Infectious**

**Diseases Conference**

**GREAT PLAINS EMERGING INFECTIOUS DISEASES CONFERENCE**

March 29th and 30th, 2018

University of Iowa College of Public Health

Iowa City, Iowa

WELCOME to the seventh-annual Great Plains Emerging Infectious Diseases Conference sponsored by the University of Iowa College of Public Health, the University of Iowa Center for Emerging Infectious Diseases (CEID), the Midwest Center of Excellence Vector-Borne Disease, and the Department of Epidemiology.

We hope that this conference will serve to bring together public health professionals, researchers, faculty, and students in microbiology, infectious diseases, and related fields working in the Great Plains and Midwestern states. The GPEID Conference highlights basic, applied, epidemiological, and translational research in biomedical and veterinary disciplines. Major topics may include but are not limited to zoonotic and vector-borne diseases including a special keynote recognition of the 100th anniversary of the Spanish influenza pandemic, global health, healthcare-associated infections, antimicrobial resistance, molecular diagnostics and epidemiology, public health preparedness, and science communication.

We thank you for your participation and look forward to the many opportunities for intellectual exchange over the next two days and into the future.



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3:30-4:00p Registration and time to hang posters, CPHB Atrium

4:00-4:50p Keynote address, N110 CPHB

  *Dr. Richard Webby, PhD*

*Current Influenza Threats; What Might GPEID 3018 Be Remembering?*

5:00-6:00p Poster presentations and Reception, CPHB Atrium

6:00-7:00p Networking, CPHB Atrium

FRIDAY, MARCH 30, 2018

7:30-8:00a Registration and breakfast (doors open at 6:30 a.m.), Atrium / C217 CPHB

8:00-8:15a Introduction and Welcome, Christine Petersen CEID Director

8:15-9:15a Research session I – Vector Borne Diseases, N110 CPHB

 *Kai Rogers*

 *Angela Toepp*

 *Brittany Fuller*

 *Deborah A. Hudman*

9:15-9:20a Break, C217 CPHB

9:20-10:20a Breakout session (choose one)

*Ethics and Challenges of Quarantine*

*Moderators: Drs. Jorge Salinas, Jovana Davidovic, and Robin Paetzold (N110 CPHB)*

*Lessons Learned from Recent Emerging Zoonoses – From Influenza A to Zika,*

*Moderators: Drs. Christine Petersen and Lyric Bartholomay (C217AB CPHB)*

10:20-10:30a Break with Coffee Available, C217 CPHB

10:30-11:30a Research session II – Bacterial Diseases, N110 CPHB

 *Geneva Wilson*

 *Wesley Hottel*

 *Corey Parlet*

 *Aaron Miller*

11:30a-1:30p Lunch, C217 CPHB

1:30-2:30p Discussion session, N110 CPHB

 *Close Range: Distant Disease on the Move*

 *A Discussion with Pulitzer Prize-winning Journalist Mark Johnson*

 *Milwaukee Journal Sentinel health/science reporter*

2:30-3:30p Q & A with keynote speakers, S106AB CPHB

3:30-4:00p Networking, Atrium / C217 CPHB

4:00-5:00p Keynote address, N110 CPHB

 *Bobbi Pritt, MD*

 *Ticks, Teamwork, and Emerging Pathogens*

5:30-8:00 Dinner and Networking, Atrium / C217 CPHB

This year’s keynote speakers are **Dr. Richard Webby, PhD, and Dr. Bobbi Pritt, MD.**

**Dr. Richard Webby, PhD**

*Member, Department of Infectious Diseases*

*Director, WHO Collaborating Center for Studies on the Ecology of Influenza in Animals*

*St. Jude Children’s Research Hospital, Memphis, TN*

**Keynote Address**

*“Current Influenza Threats; What Might GPEID 3018 Be Remembering?”
Thursday, March 29th, 4:00-5:00 p.m.
Room N120 CPHB*

Richard Webby, PhD, received his PhD from the University of Otago, New Zealand before making the move to Memphis. He has a basic research program that focuses on influenza viruses at the human-animal interface with a goal to understand pandemic emergence and risk. This work involves virologic and serologic surveillance activities in animal and human populations to determine the prevalence of influenza viruses present with further laboratory-based research to understand the mechanisms behind various viral phenotypes. Data collected through the above activities feeds into the WHO GISRS system for risk assessment of circulating influenza viruses and, where appropriate, subsequent pandemic preparedness activities.

**Dr. Bobbi Pritt, MD**

*Professor, Laboratory Medicine and Pathology*

*Director, Clinical Parasitology Laboratory*

*Co-Director, Vector-Borne Diseases Laboratory Services*

*Mayo Clinic, Rochester, MN*

**Keynote Address**

*“Ticks, Teamwork, and Emerging Pathogens”*

*Friday, March 30th, 4:00-5:00 p.m.*

*Room N120 CPHB*

Bobbi Pritt, MD, MsC serves as the Vice Chair of Education for her department and the Program Director of the Mayo Clinic MD Microbiology fellowship. She is board certified in Anatomic and Clinical Pathology and Medical Microbiology, and holds a Master’s degree in Medical Parasitology from the London School of Hygiene and Tropical Medicine and a Diploma in Tropical Medicine and Hygiene from the Royal College of Physicians in London. Dr. Pritt’s major areas of academic interest are laboratory detection of infectious diseases, medical education, and infectious disease pathology. During the past 10 years, she has worked closely with colleagues at the CDC, state and public health laboratories, and state universities to describe and characterize two new tick-borne human pathogens found in the Midwestern United States. She continues to collaborate with the CDC and public health laboratories to proactively identify new tick-borne pathogens in the United States.

**Breakout Sessions:**

**Ethics and Challenges of Quarantine**

Moderators: Drs. Jorge Salinas, Jovana Davidovic, and Robin Paetzoid

**Lessons Learned from Recent Emerging Zoonoses – From Influenza A to Zika**

Moderators: Drs. Christine Petersen and Lyric Bartholomay

**Discussion Session:**

**Close Range: Distant Disease on the Move a Discussion with Pulitzer Prize-winning Journalist Mark Johnson**

*Milwaukee Journal Sentinel health/science reporter*

**Wesley Hottel, MS**

University of Iowa, Department of Epidemiology

Genomic similarity of *Legionella pneumophila* isolated from routine monitoring of hospital premise plumbing systems

**Deborah A. Hudman, MS**

A.T. Still University, Department of Microbiology and Immunology

Prevalence of Tick-Borne Pathogens in Northeast Missouri

**Aaron C. Miller, PhD**

University of Iowa, Department of Epidemiology

Real-time influenza forecasting using thermometer-recorded and user-reported symptom information

**Corey Parlet, PhD**

University of Iowa, Department of Microbiology and Immunology

Enhanced neutrophil effector responses conferring protective anti-MRSA immunity correspond with CD4+ T cell expansion in the skin and germinal center B cell formation in the skin draining lymph nodes

**Kai Rogers**

University of Iowa, Department of Microbiology and Immunology

Acute *Plasmodium* infection promotes resistance to Ebola virus via type 1 immunity

**Patricia Storlie, PhD**

Wartburg College, Department of Biology

Designing a CRISPR/Cas9 Toolkit for Major Surface Protease (MSP) Knock-out in *Leishmania tarentolae*

**Angela Toepp, MS**

University of Iowa, Department of Epidemiology

Impact of maternal and sibling *Leishmania infantum* infection status on vertical transmission of Leishmaniosis

**Geneva Wilson, MS**

University of Iowa, Department of Epidemiology

Measuring the Bioaerosol Concentration of *C. difficile* spores in Infected Patient’s Rooms during Select Activities

**Tiffany Y. Borbón**

University of Iowa, Department of Microbiology and Immunology

Bacterial co-infection in murine cutaneous leishmaniasis

**Eric Kontowicz, MS**

University of Iowa, Department of Epidemiology

Flooding and Influenza in Iowa

**Mitchell Lefebvre**

University of Iowa, Department of Microbiology and Immunology

Radiation attenuated sporozoite vaccination combined with subunit vaccination induces protective CD8 T cell responses against Plasmodium in mice

**Kurayi Mahachi, MS**

University of Iowa, Department of Epidemiology

Ticks near and far: A look at the global distribution of ticks and tick-borne diseases in the United States Ethiopia and India

**Marie Ozanne, MS**

University of Iowa, Department of Biostatistics

Visceral Leishmaniasis in Brazil: a Quest for a Reproductive Number

**Avery Peace, PhD**

University of Wisconsin, Department of Pathobiological Sciences

Metagenomic assessment of viruses of black-legged ticks (*Ixodes scapularis*) in Wisconsin

**Austin Rau**

University of Iowa, Department of Geographical & Sustainability Sciences

Landscape Genetics of H3N2 Influenza in Minnesota: 2012-2013

**Breanna M. Scorza, PhD**

University of Iowa, Department of Epidemiology

Characterization of circulating Natural Killer cells in canines exposed to tick-borne infections

**Kevin Tsai, MPH**

University of Iowa, Occupational and Environmental Health

Enteric Pathogen Contamination Investigation of Infant Weaning Foods in Low-income Neighborhoods of Kisumu, Kenya

**Diogo G. Valadares**

University of Iowa, Internal Medicine

T cell exhaustion during murine cutaneous and visceral leishmaniasis

**Rahul Vijay, PhD**

University of Iowa, Department of Microbiology and Immunology

Unexpected ˜Immunoblockade” following Co-stimulation during experimental malaria

**Tiffany Y. Borbón**; Gwendolyn Clay; Breanna Scorza; Alan Sariol; Yani Chen; Bayan Zhanbolat; Fayyaz Sutterwala; and Mary E. Wilson

University of Iowa

Cutaneous leishmaniasis (CL) is caused by *Leishmania* protozoa leading to localized skin lesions and ulceration. In some human infections, response to treatment is best initiated after ulceration. Furthermore, ulceration introduces bacteria from skin microbiota, and predisposes to secondary bacterial infection. *Staphylococcus aureus* is a bacterium often cultured from CL lesions. We hypothesized that skin-derived bacteria activate inflammatory responses that contribute to outcome of *Leishmania* infection.

We injected C57BL/6 mouse ears with *L. major*, *S. aureus*, or both intradermally, and monitored lesion volume, histologic inflammatory response, microbial burden (qPCR) and antigen-induced expression of immune mediators in lymph node cells 1 and 4 weeks post-infection (p.i.) (RT-qPCR, bioplex assay, intracellular cytokine stain). Results showed a two-fold greater lesion size in co-infected ears than ears injected with either pathogen alone (p<0.0001, p<0.001 at 3 days, 3 weeks). However, microbial burdens were unchanged between co-infected vs. singly infected groups. Co-infected lesions had significantly more neutrophils than ears infected with L. major alone (1.5-fold, p<0.01). *Leishmania* antigen-stimulated LN cells from co-infected mice released more IL-17A than singly infected mice (p<0.001). These differences occurred though there was no detectable S. aureus DNA remaining in ears. However, IL-17A was produced by γδ T cells at 1 week p.i., and produced by Th17 cells at 4 weeks p.i. These data suggest IL-17A underlies the enhanced neutrophilic infiltrate and larger lesion size caused by S. aureus co-infection of L. major lesions, but the IL-17A source differs between early (γδ T cells) vs. late (Th17 cells) infection.

**Wesley Hottel;** Valerie Reeb; Nancy Hall; Lucy DesJardin

University of Iowa

Whole genome sequencing (WGS) was performed on *Legionella pneumophilia* (Lp) strains isolated from various locations of hospital premise plumbing systems as part of routine monitoring in order to better understand strain diversity over time. 46 Lp isolates from various locations in two facilities were analyzed; Facility A predominately isolated Lp serogroup (sg) 1 (sampled 2012-2016) and Facility B with predominately Lp sg 4 (sampled 2013-2016). The selection of Lp isolates to sequence represented different collection dates to determine if a dominant lineage was observed over time and location within the facility. wgMLST analysis of Illumina MiSeq Next Generation Sequence (NGS) data showed that there were two commonly found sequence type (ST) populations in Facility A. One cluster belonged to ST36 known to be associated with various outbreaks in the U.S. and a separate cluster of ST1 the most common ST among sporadic disease and environmental Lp isolates globally. Resfinder identified a beta-lactam resistance gene blaOXA-29 in the ST1 genomes. A BLASTn analysis indicated that this DNA sequence is associated with the Paris strain plasmid (pLPP) which appeared to be stable in isolates at this facility over time. Facility B isolates had >98% identical alleles using a *Legionella* wgMLST scheme and belonged to ST378. A SNP-based typing scheme revealed that although these strains were all closely related some facility locations had sub-clusters that persisted over time. This work allows for better characterization of *Legionella* species that colonize hospital plumbing systems and may inform what actions are needed when *Legionella* is isolated.

**Deborah A Hudman**

A.T. Still University

We evaluated *Amblyomma americanum* (lone star tick) and *Dermacentor variabilis* (American dog tick) in northeast Missouri for the presence of *Borrelia,* *Ehrlichia,* and *Rickettsia* bacteria and Heartland virus. We collected actively questing ticks from four state owned conservation areas in Adair County Missouri. Two of those conservation areas were managed and the other two have not been managed for the past ten years. We screened 436 individual adult lone star ticks and infection rates were 6% for *B. lonestari,* 19% for *E. chaffeensis,* 3% for *E. ewingii,* 36% for *R. amblyommatis,* and 1% for *R. montanensis*. In the 189 individual American dog ticks infection rates were 18% for *E. chaffeensis,* 15% for *E. ewingii,* 5% for *R. amblyommatis,* and 3% *for R. montanensis*. In addition we screened 20 adult pools (5 adults per pool n=100) and 30 nymphal pools (25 nymphs per pool n=750) for the Heartland virus which was not detected. Prevalence of tick-borne pathogens were more or less similar between managed vs unmanaged sites however tick numbers were nearly double in unmanaged sites which makes the argument that some form of management may reduce the risk of potential disease transmission. Understanding the presence and epidemiology of these causative (*E. chaffeensis, E. ewingii,* and Heartland virus) and suspected (*B. lonestari*, *R. amblyommatis,* and *R. montanensis*) agents in Missouri should increase awareness of potential tick-borne disease in the medical community.

**Eric Kontowicz**

University of Iowa

Influenza is an important and reoccurring public health issues in the state of Iowa given our large swine population and other related agricultural practices. Despite having annual vaccination campaigns influenza still poses a public health threat. Cases of influenza are often sporadic in nature and the and the annual case number can vary from year to year; but on average it is expected that 2% of outpatient visits will be related to influenza like illness. Factors that can cause influenza relate illness to rise above these numbers is of public health and epidemiological importance. The main factors that often influence influenzaâ€™s infectivity are often driven the virusâ€™ genome and the mutations that can occur in this genome. There are many different host and environmental factors that can influence influenzaâ€™s infectivity as well. The primary aim of this study will be to evaluate if there is any association between flooding events that have occurred in Iowa with an increase in Influenza cases in the following flu season. A secondary aim of the study will be to evaluate if there is any effect of flooding events on influenza strain variability seen in the state of Iowa. The hypothesis for this study is that flooding events will be both significantly and positively associated with an increase in influenza incidence and an increase in strain variability. Influenza cases and strain data will be taken from the Iowa State Hygienic Lab and flooding events will be identified via literature review.

**Mitchell Lefebvre**

University of Iowa

The Plasmodium species are the causative agent of malaria a devastating mosquito born disease that is responsible for millions of infections and hundreds of thousands of deaths annually. Anti-malarial therapies suffer from emerging parasite drug resistance and there are no readily deployable protective vaccines despite decades of research. Sporozoites the infectious form of the parasite can be attenuated with radiation (RAS) and injected intravenously (IV) to induce protective immunity in mice and humans. Unfortunately this vaccination strategy is not field practical as it requires multiple large IV doses. Other vaccination strategies using subcutaneous and intradermal delivery of subunit vaccines are more readily administrable in the field but induce short lived non-sterilizing immune responses. An “ideal” malaria vaccine would induce immunity at the asymptomatic liver stage preventing development of the symptomatic blood stage. We use understanding of the immunological requirements for protection from Plasmodium in mice to guide human vaccination efforts. Here we sought to determine if combining RAS immunization with subunit vaccines could generate highly protective memory CD8 T cells. Our data show that a single RAS vaccination primes Plasmodium-specific CD8 T cells that offer modest protection against malaria infection. A subunit vaccine consisting of recombinant Listeria monocytogenes expressing a specific Plasmodium target epitope boosts RAS-induced CD8 T cell numbers specific to that epitope and depending on the epitope confers sterilizing immunity upon rechallenge. My results suggest that RAS vaccination can be optimized by combination with subunit vaccines in mice to generate protective memory CD8 T cell responses against Plasmodium.

**Kurayi Mahachi**

University of Iowa

In recent years studies have highlighted increased occurrences of tick-borne diseases both nationally and globally. Outside of higher income countries the primary methodology for the diagnosis of tick-borne diseases in both human and animal subjects has been microscopy. However recent studies have noted that microscopy may only identify approximately 10% of the actual pathogen load in a subject thus potentially grossly underestimating the incidence and prevalence of individuals infected with emerging and re-emerging tick-borne diseases. In countries such as the United States the increased use of rapid molecular tests such as Polymerase chain reaction (PCR) and enzyme-linked immunosorbent assay (ELISA) have increased the ability to detect pathogens that would otherwise go unnoticed using microscopy. however these rapid tests are utilized less in more austere and remote regions of the world such as Ethiopia and India. Therefore by comparing the ticks and tick-borne diseases in the United States Ethiopia and India this presentation seeks to highlight the need for novel rapid detection methods particularly in remote regions of the world. Through the comparison of scientific literature regarding tick -borne diseases from the United States Ethiopia and India coupled with data from the Center for Disease Control and the World Health Organization e identified similarities and differences in the species of ticks and potential pathogens transmitted for each country. We further compared the methods employed to detect pathogens in each country and evaluated their effectiveness highlighting the need for novel diagnostic tools in order to decrease the potential for a novel global pathogen.

**Aaron C. Miller**

University of Iowa

Background: Improved forecasting of seasonal influenza outbreaks may help limit adverse effects. Although novel data sources, such as internet search volume, have been used to forecast influenza activity, such data represent proxy measures of influenza-related symptoms. However, internet-connected smart thermometers and mobile devices can be used to directly record symptoms in real time. These devices may capture specific features of influenza-like illness (ILI) including fever duration, possible household transmission, secondary infections or other user-reported symptoms.

Methods: We analyze time-stamped, geo-located temperature readings and user-reported symptoms from Kinsa Smart Thermometers. We consider ILI-related measures including: weekly counts of fevers, in-household transmission events, biphasic fever episodes, fever episodes lasting for influenza-specific durations (e.g., 3-7 days), and user reported symptoms such as cough. We develop dynamic forecasting models to predict CDC reported outpatient ILI activity, and evaluate out-of-sample performance using CDC reports available at the time of forecast.

Results: From September 1st, 2015 through January 16th, 2018 there were over 8.6 million temperature readings and 1.4 million other symptoms recorded. Forecasts improved up to 50% when symptom information was incorporated. The strongest predictors of influenza activity were distinct fever episodes and in-household transmission events. In particular, transmission events that spread from adult to adult or child to adult were most predictive of ILI.

Conclusions: Mobile devices can capture multiple influenza-related symptoms and can be used to generate real-time forecasts. Additional efforts to capture influenza related events, such as household transmission activity, may help better characterize or predict the severity of an outbreak.

**Marie Ozanne**

University of Iowa

Statistical modeling can provide valuable insight into the dynamics of infectious disease spread. One important question that can be addressed through statistical modeling concerns the proportions of individuals in the population that fall at different points in the spectrum from asymptomatic infection to severe disease. As these proportions are often difficult to observe estimation of these quantities can provide information about the nature and severity of the disease in a particular population. Logistic and multinomial regression techniques are often applied to infectious disease modeling of large populations. These methods are suited to identifying variables associated with a particular disease or disease state. However they are less appropriate for estimating infection state prevalence over time because they do not naturally accommodate known disease dynamics such as duration of time an individual is infectious heterogeneity in the risk of acquiring infection and patterns of seasonality. We propose a compartmental modeling approach for estimating latent infection state prevalence over time that easily incorporates known disease dynamics. We introduce a novel method for estimating Bayes factors and for estimating the empirically-adjusted reproductive number in the *L. infantum* endemic region. Through simulation we demonstrate that the stochastic compartmental modeling approach provides better estimates of latent infectious state proportions than a logistic regression approach does for dynamic diseases. We also provide a real data example for visceral leishmaniasis in Brazil for which we compare models and calculate the empirically-adjusted reproductive number.

**Corey Parlet**; Alexander Horswill; Mary Wilson

University of Iowa

*Staphylococcus aureus* (*S. aureus*) is the single greatest cause of skin and soft tissue infections, which are often a prelude to invasive, life-threatening disease. The development of both drug-resistance and hyper-virulence are typifying features of methicillin-resistant *S. aureus* (MRSA) strains that have inflicted pandemic disease in healthy populations. Both clinical and experimental evidence suggest that neutrophils (PMNs) are essential mediators of anti-*S. aureus* host-defense and that the enhanced capacity of emerging MRSA strains to unleash PMN killing toxins accounts for their ability to overwhelm host-defense responses and transmit disease among immune-competent populations. Using a MRSA challenge model that elicits protective immunity against recurrent MRSA skin infection, we show that the protection afforded to previously infected “immunized” mice, corresponds with a ten-fold increase in the accumulation of phagocytic PMNs at infectious foci. Suggesting that skin T cells contribute to enhanced PMN responses in immune mice, primary *S. aureus* infection prompts TH17 cell influx into infected skin and durably elevates total CD4+ T cells numbers throughout the tissue well into memory time points (>8wks). Supporting a role for B cell participation in anti-MRSA cutaneous immunity, primary infection robustly induces germinal center B cells in the skin draining lymph nodes and affords protection from dermonecrotic injury after sterile challenge with MRSA toxins. These findings provide new information about the adaptive T and B cell correlates of anti-MRSA protective immunity and suggest that the appropriate induction of humoral and cellular immune responses can separately but synergistically support anti-MRSA PMN activity and bolster host-defense.

**Avery Peace**; T. Goldberg; S. Paskewitz; & L. Bartholomay

University of Wisconsin-Madison

The black-legged tick (*Ixodes scapularis*) transmits a diverse array of human pathogens including many associated with emerging diseases in the Upper Midwest. In Wisconsin we have noted clinical cases of encephalitis in patients with a history of contact with ticks and symptoms consistent with tick-borne disease who tested negative for known tick-borne pathogens. To explore the possibility that black-legged ticks in WI could be involved in transmission of thus far undescribed pathogens RNA from pools of nymph stage *I. scapularis* collected in summer of 2016 was subjected to unbiased next-generation sequencing. This effort yielded evidence of two putative novel Rhabdoviruses as well as viruses closely related to black-legged tick viruses reported on the East Coast (Southbay virus and Black-legged Tick Phlebovirus). Additional specimens were collected in the summer and fall of 2017 and were subjected to next-generation sequencing of adult tick salivary gland tissue and whole bodies of nymphs. Results of this analysis and their potential significance to human health will be presented.

**Austin Rau**

University of Iowa

Influenza causes thousands of illnesses and deaths each year in the United States. In part this is due to rapid changes in influenza genetics resulting in different variants than the previous season. Seasonal influenza viruses travel across landscapes by infecting susceptible hosts thus allowing it to move great distances due to the mobility of humans who occupy diverse environments. Using H3N2 influenza viral sequences from Minnesota in the 2012-2013 season landscape genetic methods were used to test for correlations between genetic distances of influenza viruses with spatial and non-spatial distances separating the viruses at the zip code tabulation areas (ZCTA) scale where those viruses were isolated. Genetic distances between viruses had significant correlations with Euclidean and temporal distances. Overall influenza genetics had complex relationships with the distances used to measure separation between viruses. The information from these analyses can be used to inform our overall understanding of influenza diffusion and evolution across a landscape and adds to the growing body of landscape genetics research on infectious diseases.

**Kai Rogers;** Rahul Vijay; Chester Joyner; Mary Galinski; Noah Butler; Wendy Maury

University of Iowa

Ebola virus (EBOV) outbreaks occur sporadically in Central and West Africa with case fatality rates as high as 90%. Individuals are often infected with other pathogens endemic to these regions but consequences of such co-infections are understudied. Epidemiological studies from the 2014-2016 epidemic indicate that a significant number of EBOV patients were co-infected with *P. falciparum* when admitted to Ebola treatment units. Currently there is no consensus regarding how or whether malaria impacts EBOV infection with different epidemiological studies suggesting better or worse outcomes associated with co-infection. Here we investigated the effect of pre-existing malaria on EBOV challenge. C57BL/6 interferon alpha/beta receptor knock out mice were infected with *Plasmodium yoelii* and subsequently challenged intraperitoneally with a BSL-2 model of Ebola virus recombinant VSV encoding Ebola GP (EBOV/rVSV). Acute infection with *P. yoelii* protected mice from a lethal challenge with EBOV/rVSV and facilitated long-lived immunity against EBOV. Mice remained protected against EBOV challenge weeks after resolution of malarial disease suggesting the host response to *P. yoelii* rendered mice resistant to EBOV. Mechanistically we identified that protection against EBOV was linked to IFN?-mediated M1 polarization of peritoneal macrophages (pmacs) in *P. yoelii*-infected mice. Serum from mice acutely infected with *P. yoelii* induced M1 polarization of pmacs and reduced EBOV/rVSV infection ex vivo. Similarly human macrophages treated with serum from rhesus macaques acutely infected with *P. cynomolgi* were protected against EBOV/rVSV challenge. Protection in these assays was abolished by neutralizing anti-IFN?. Finally *P. yoelii*-infected mice lacking the IFN? receptor were not protected from EBOV/rVSV yet their serum containing IFN? induced an M1 phenotype and protected wild-type pmacs. These experiments support the hypothesis that acute malaria infection protects against EBOV

**Breanna M. Scorza**; Angela Toepp; Kurayi Mahachi; and Christine Petersen

University of Iowa

Tick-borne diseases are are the most common vector-borne disease diagnosed in people in the US and the incidence of tick-borne diseases such as Lyme disease Ehrlichiosis and Anaplasmosis is rising. Many of these tick-borne diseases are zoonotic. Dogs have been shown to serve as a sentinel species for human Lyme disease. Studies of the inflammatory responses induced during Lyme disease in people have found a significant role for Natural Killer (NK) immune cells during the acute disease response but whether these cells are invoked during canine Lyme disease is unknown. We identified Foxhounds with natural exposure to tick borne infections using the 4Dx snap-test (IDEXX) which detects serum antibodies to *Borrelia Ehrlichia* and *Anaplasma* species antigens. We performed physical exams to classify dogs as healthy controls having asymptomatic Lyme infection or clinical Lyme disease (apparent clinical arthritis or other signs of disease). NK cells from peripheral blood were compared in dogs on the basis of their proportion of circulating lymphocytes CD94 expression and Granzyme B expression. Identification of immune cell types and phenotypes associated with disease in canine Lyme disease is the first step to understanding the overall immunopathogenicity of this disease in dogs.

**Patricia Storlie;** Brittney Fuller; Alicia Urbain

Wartburg College

The *Leishmania* species protozoa are dimorphic parasites that are transmitted to susceptible animals through the bite of a sand fly. There are more than 30 species of *Leishmania* most of which infect humans and cause disease. *Leishmania tarentolae* is non-pathogenic to humans and is instead found in lizard species where it has been observed inhabiting the lumen of the intestine as the flagellated promastigote form as well as within the hostâ€™s macrophages as an intracellular non-flagellated amastigote form. This study focuses on a prominent surface protein called the major surface protease (MSP gp63) found in all *Leishmania* species. MSP has been shown to aid in parasite invasion into human macrophages and promote pathogenicity in mammals but its roles in other host species remains unclear. Studying MSP can afford knowledge on the difference in host specificity of *L. tarentolae* especially since *L. tarentolae* very high genetic similarity to other *Leishmania* species. The *Leishmania* genome has proven to be challenging to manipulate when deleting large gene families including gp63. Recently however CRISPR-Cas9 techniques have been successfully used in knocking out large-scale loss-of-function phenotypes in various *Leishmania* species. A CRISPR-Cas9 knock-out has not been completed on gp63 in any species of *Leishmania*. We are currently constructing CRISPR-Cas9 plasmids that specifically target multiple gp63 genes in *L. tarentolae*. Our hypothesis is that guide RNA (gRNA) vectors and Cas9 coexpression vectors can be used to delete MSP in *L. tarentolae* similar to the targeting of other multi-loci gene families in pathogenic *Leishmania* species.

**Angela J. Toepp M.S.**; Carolyne Bennett MPH; Benjamin Scott MPH; Reid Senesac MPH; Christine A. Petersen D.V.M PhD

University of Iowa

Canine leishmaniosis (CanL) a deadly disease caused by *Leishmania infantum* parasite it is endemic in animal populations in more than 98 countries across the globe. Within the U.S. hunting dog population CanL is enzootic. CanL was first identified within the US in 1980 and then again in 1999 when a large outbreak in a kennel in New York occurred. Within the U.S. hunting dog population CanL is transmitted vertically with no reported vector transmission in the population despite multiple attempts to find infected sand flies associated with these dogs. While vertical transmission has been reported in case reports around the globe risk factors associated with this unique means of *Leishmania* transmission have not been identified. Furthermore the basic reproductive number (R0) or number of new infections that one infected animal can cause has not been reported for vertical transmission of *L. infantum* within a population where large amount of longitudinal data is available. A cohort of 138 dogs from 19 dams was analyzed from 1999 to 2016. Puppies born to dams that were ever diagnostically positive for infection with *L. infantum* were 5x more likely to become positive for *L. infantum* themselves within their lifetime (RR: 5.06 95% CI: 2.43-10.51 p-value: <0.0001). The basic reproductive number for vertically transmitted *L. infantum* within this cohort was 6. These results underscore the need for any public health prevention and control efforts to address vertical as well as vector transmission of canine leishmaniosis in endemic countries.

**Kevin Tsai**

University of Iowa

Measuring infant food contamination in low and middle-income settings can be difficult as fecal indicators are nonspecific and often do not correlate well with viral bacterial and protozoan pathogens. Therefore it is crucial to develop an effective microbial testing method that enables quantitative and specific measuring of infant food contaminations in low and middle-income countries. In this study, we used a nucleic acid extraction method (ZymoBIOMICS DNA/RNA extraction mini-kit) ,which allows the samples to be collected and transported at ambient temperature before nucleic acid extraction and refrigeration. Bacterial, viral and protozoa enteric pathogens were detected in 62% of the infant weaning food samples collected. Ninety-five percent of the milk samples collected contained target enteric pathogen. Multivariate analysis indicated food type and month of sampling are associated with presence and absence of enteric pathogens and enteric pathogen diversity.

**Diogo G. Valadares**; Yani Chen; Rich Davis; Ellen Kiser; Mary E Wilson

University of Iowa

Leishmaniasis is caused by infection with the intracellular protozoan *Leishmania* causing a chronic progressive disease that usually is controlled by TH1 IFN-? producing cells. However despite the increasing IFN-? amount in skin of cutaneous or in spleens of VL patients these patients donâ€™t control the disease. T cell exhaustion is defined by poor effector function and prevents optimal control of infection. We hypothesized that during the chronic *Leishmania* infection an increased exhausted T cell population arises compromising the host healing. To address T cell exhaustion during cutaneous and visceral leishmaniasis Balb/C mice were infected with *L. major* or *L. donovani* and various anatomical compartments were analyzed by flow cytometry in different time points of infection. With *L. major* infection around 1 week post infection the main myeloid population expressing PDL-1 were CD11c+ cells in circulation and ear tissue. After 3 weeks post infection neutrophils in blood showed an increased PDL-1 expression (comparing with PBS group 2500 cells and 1300 cells respectively) and mostly of the myeloid populations in ear (site of infection) and draining LN revealed an increased PDL-1 surface expression. These same tissues showed an increased CD4 T and CD8 T cell populations expressing PD-1 comparing with healthy controls. For *L. donovani* infection the first myeloid population to reveal an increased PDL-1 expression was also CD11c+ cells in blood than PMNs and DCs subsets in spleens at week 4 post infections. Also during this time in spleens CD4 T cells showed an increased PD-1 expression comparing with healthy controls (21% and 12% respectively) but not CD8 T cells (4.8% and 4.3% respectively). In conclusion both forms of Leishmaniasis elicited T cell exhaustion markers having neutrophils and DCs subsets as main cells related to this phenomenon. Although CD4 T and CD8 T cells seem to be exhausted in cutaneous form the T cell exhaustion in visceral form seems to

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Protection from malaria depends on the differentiation of T follicular helper (Tfh) cells and their orchestration of parasite-specific antibody responses. However the acquisition of antibody-mediated anti-malarial immunity is often delayed and seldom protect individuals from repeated exposures. We and others have linked deficiencies in anti-malarial humoral immunity to the upregulation of multiple co-inhibitory receptors (immune checkpoints) on parasite-specific helper CD4 T cells. Blocking co-inhibitory receptor signaling can restore Tfh function elevate parasite-specific antibody titers and improve parasite clearance during experimental malaria. We have also shown that agonizing a single co-stimulatory receptor OX40 a tumor necrosis factor receptor superfamily (TNFRSF) member early during experimental malaria restores the immune response. We recently observed that a second costimulatory molecule 4-1BB (CD137) which also belongs to the TNFRSF is relatively highly expressed on CD4 responding to Plasmodium infection. Agonizing 4-1BB in models of cancer and viral infections potently stimulates T cell proliferation and accelerates the acquisition of a memory-like phenotype. Thus we hypothesized that agonizing 4-1BB would expand Tfh responses stimulate higher quality germinal center reactions and enhance parasite clearance during malaria. Contrarily CD4 T cells in mice treated with anti-4-1BB upregulated Eomesodermin (Eomes) and did not express Bcl-6 a Tfh specific transcription factor. Consistent with this we observed delayed clearance of the parasite that was associated with CD4 T cells acquiring a more cytotoxic phenotype and impaired germinal center reactions in the treated group. Experiments are underway to determine the role of 4-1BB and the relationships between Eomes induction Bcl-6 repression and the fate of Plasmodium-specific CD4 T cells.

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*Clostridium difficile* (*C. difficile*) is a gram positive anaerobic bacterium. The spore forming nature of *C. difficile* gives it the ability to persist in the environment for long periods of time and makes it impervious to many commonly-used hospital disinfectants. Previous studies have shown that toilets seeded with *C. difficile* spores are able to produce bioaerosols from the toilet plume. Toilet flushing could be a source of contamination for healthcare workers (HCW) who are exposed to the plume while preforming routine care procedures such as emptying the bed pan of *C. difficile* infected patients. Other routine activities conducted by healthcare workers in an infected patient’s room such as bathing the patient and performing bedside medical procedures could also lead to *C. difficile* exposure via aerosolized spores produced during these activities. The purpose of this study is twofold: first to establish how the aerosol produced during toilet flushing contributes to CDI contamination in the bathrooms of *C. diff* positive patients within the healthcare setting. Second to observe the frequency of eleven routine patient care activities performed by healthcare workers and determine if those activities are correlated to an increase in *C. difficile* bioaerosol concentration. The study hypothesis is that activities such as bed pan use toilet flushing bathing patients performing routine medical procedures changing soiled linens and moving the privacy curtains will be associated with an increased concentration of aerosolized *C. difficile* spores as compared to when there is minimal patient or HCW activity.

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