Monday, June 3rd, 2019

**9:30 - 9:40 WELCOME ADDRESS**

**9:45 – 10:00: Avid Mohammadi**
Characterizing the impact of penile-vaginal sex on HIV-susceptible CD4+ T cell subsets in the female genital tract

**10:05 - 10:20: Erin O. Y. Wong**
Developing defined microbiota to model inflammation in the mouse gut

**10:25 - 10:40: Nora Mellouk**
An ATG16L1-dependent pathway promotes plasma membrane repair and limits Listeria monocytogenes cell-to-cell spread

**10:45 - 11:15: COFFEE BREAK**

**11:20 - 11:35: Jean-Paul R. Soucy**
Joint modelling of resistance to six antimicrobials in urinary *Escherichia coli* isolates in Quebec, Canada

**11:40 – 11:55: Sarah Birstonas**
EHEC utilizes two-component systems to modulate expression of major flagellar subunit protein, FliC, in response to host intestinal cues

**12:00 - 12:15: Nathaniel Winsor**
NLRP6 regulates the colonic mucus layer during Tritrichomonas infection

**12:35 – 1:30: LUNCH**

**1:35 - 12:50: Samuel Salamun**
Epstein-Barr Virus Protein BMRF1 Modulates Cellular SUMO and DNA Damage Response Pathways by Binding the Cellular NuRD Complex

**1:55 - 2:10: Nicola Case**
Elucidating the mechanism of Candida albicans morphogenesis in response to phagocytosis by macrophages

**2:15 - 2:30: Sarah Kronheim**
A small molecule anti-phage defense mechanism in *Streptomyces*

**3:05 - 3:20: Alexandra Willis**
Understanding inherited immunity using a *C. elegans* model of microsporidia infection

**3:25 - 3:40: Genevieve Mailhot**
Differentiating between protective and pathogenic neutrophil responses during *Neisseria gonorrhoeae* infection

**3:45 – 4:00: Tiffany Fitzpatrick**
Successes of anti-RSV prophylaxis among infants in Ontario: results from a multi-decade, population-based controlled interrupted time series analysis using health administrative data
Poster Presentations
Intercurrent Flaviviral Viremia and Plasmodium ovale Infection in Ill Returned Travelers to Ontario

Ruwandi Kariyawasam1, Katherine Tan2, Rachel Lau3, Filip Ralevski3, Aisha Khatib4,5, Andrea K. Boggild1,3,4,5

1Institute of Medical Sciences, University of Toronto, Toronto, ON, Canada 2Department of Human Biology, University of Toronto, Toronto, ON, Canada 3Public Health Ontario Laboratories, Toronto, ON, Canada 4Tropical Disease Unit, UHN-Toronto General Hospital, Toronto, ON, Canada 5Department of Medicine, University of Toronto, Toronto, ON, Canada.

Similar epidemiology and clinical presentations of arboviral infections and malaria coupled with the typically sequential approach to diagnostic testing, where malaria is confirmed or excluded urgently in febrile returned travelers, may mask the true epidemiology of co-infections. Flaviviruses are known to trigger relapsing forms of malaria, including Plasmodium ovale, long after primary malaria infection, and this may delay the diagnosis of malaria. We aim to understand the incidence of intercurrent flaviviral infection in confirmed Plasmodium ovale infection. DNA and RNA from biobanked isolates of P. ovale detected in whole blood at the Public Health Ontario Laboratory between 2006 and 2019 were extracted and screened for intercurrent flaviviral infections using previously validated real-time PCR (qPCR) assays targeting multiple flaviviruses (pan-FLAV) and, specifically, dengue virus types 1-4 (DEN1, DEN2, DEN3, DEN4). One-hundred seventeen unique isolates of P. ovale were identified, of which 68 had sufficient remaining specimen for further molecular analysis. Males accounted for 54.4% (n=37/68) of P. ovale cases, while females accounted for 44.1% (n=30/68), and sex was unassigned in 4.4% (3/68). Median age of P. ovale cases was 27.4 years (range 22 mos - 72 years; IQR 18.8 – 40.1 years). Median parasitemia was < 0.01% (range < 0.01% - 0.8%). Thirty-one (45.6%) P. ovale cases had documented travel history exclusively to Africa, with Nigeria as the most common source country (23/31 [74.1%]). Pan-FLAV assay yielded a 1.6% (1/68) positivity rate. DENV was not detected in any specimen. P. ovale infections are most commonly imported to Ontario from West Africa, and Nigeria, specifically. Intercurrent flaviviral viremia was noted in at least 1.6%, which may suggest that primary flaviviral infection triggered a relapse of P. ovale. Alternatively, such co-occurrence may suggest primary infection with both organisms known to cause fever in returning travelers. Consideration of flaviviral co-infection should be given to the P. ovale patient with deep thrombocytopenia, lymphopenia, and high-yield arboviral symptomatology such as rash and retro-orbital headache.