

UNIVERSITY OF TORONTO

Microbiology & Infectious Diseases Research Days

Monday, June 3rd, 2019 – Trainee Day (Selected from Abstracts)

Tuesday, June 4th, 2019 – Invited Lectures & Poster Session

Talks in Medical Sciences Building, Room 2170

**Posters & Lunch in Medical Sciences Building,
Room 2171 (C. David Naylor Student Commons)**

Website: <http://microbeto.ca/mid-2019/>

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 *Trichomonas* 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*

2.30 - 3:00: COFFEE BREAK

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

81) Intercurrent Flaviviral Viremia and Plasmodium ovale Infection in Ill Returned Travelers to Ontario

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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.