

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

26) Low Sequence Heterogeneity of *Plasmodium falciparum* Isolates Imported to Ontario, Canada from West Africa over a 10-year Period with Increased Molecular Markers of Resistance to Proguanil

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Approximately 200 cases of malaria are imported to the province of Ontario annually, with the majority due to *Plasmodium falciparum* (Pf) originating from West Africa. We performed sequence analyses of Pf isolates returning from Ghana and Cameroon over a 10-year period to understand patterns of genetic heterogeneity and molecular drug resistance markers over time. We identified 36 Pf isolates from Ghana (18 from 2006-2008 and 18 from 2014-2016); and 16 from Cameroon throughout 2006-2016. DNA was extracted and regions commonly used for strain typing were analyzed including: merozoite surface protein (msp) 1 and 2; erythrocyte binding antigen (eba) 175; and glutamate-rich protein (glurp) regions. Molecular resistance markers including: cytochrome B (cytB) and dihydrofolate reductase (dhfr) for resistance to atovaquone-proguanil (Malarone®); atpase6 and kelch13 for artemisinin and derivatives; and chloroquine resistance transporter (PfCRT) for chloroquine were analyzed. Phylogenetic tree analysis revealed some sequence heterogeneity within Ghanaian and Cameroonian isolates, however, there was no clustering of isolates over time. All isolates were wild type on cytB codon 268. Isolates from Cameroon all had triple codon 51, 59, and 108 mutations at dhfr conferring resistance to proguanil, whereas isolates from Ghana had an increase of such mutations from 39% (7/18) in 2006-2008 to 83% (15/18) in 2014-2016 ($p=0.0153$). Eight percent (3/36) of Ghanaian isolates had a mutation in codon 623 of atpase6, while all Cameroonian isolates were wild type. No mutations were observed at atpase6 codon 769 or kelch13 codons >440. In PfCRT codon 76, 27% (7/26) of Ghanaian isolates were mutant compared to 50% (6/12) of those from Cameroon. Pf isolates from Ghana demonstrated increasing molecular markers of resistance to proguanil, but remain wild type to the partner drug atovaquone in Malarone. The relatively high percentage of molecular mutants to chloroquine resistance still predominates throughout West Africa. The low sequence heterogeneity suggest there was no major evolutionary genetic changes over the years.