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 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

2.30 - 3:00: **COFFEE BREAK**

3:05 - 3:20: Alexandra Willis

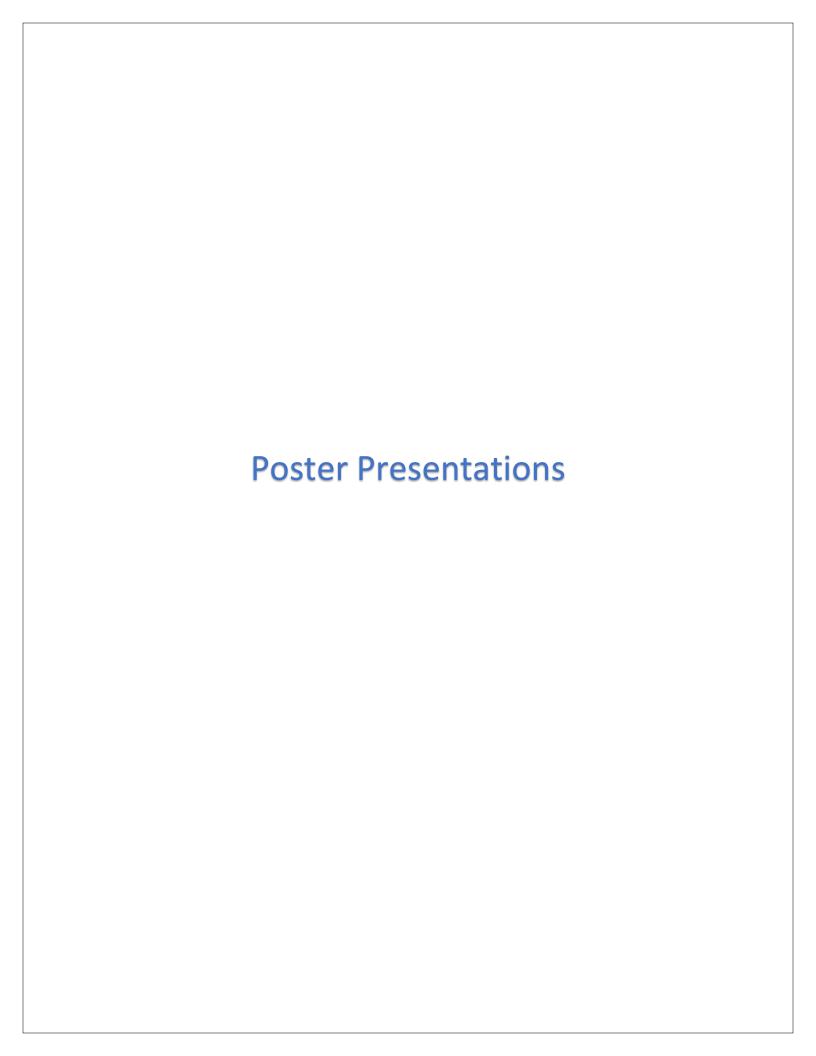
Understanding inherited immunity using a *C. elegans* model of microsporidia infection

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



49) A Systematic Review of Virulence Factors in the Leishmania Genus

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Parasite-determined factors play a complementary role in the pathogenesis of leishmaniasis, a disease caused by protozoans of the genus Leishmania with diverse and species-specific clinical manifestations. Virulence factors (VFs), or pathogen moieties facilitating disease, can potentiate host cell damage by Leishmania species via increased expression, host cell invasion, stress tolerance, and modulation of the host immune system. Due to large eukaryotic genomes in Leishmania species, there is a wide array of VFs which contribute to different aspects of pathogenesis. Here we conduct a comprehensive, systematized review of the literature around VFs in Leishmania spp. and construct a complete picture of parasite-determined contributors to the pathogenesis of various clinical forms of leishmaniasis. PubMed (NCBI), MEDLINE (OVID), EMBASE (OVID), Web of Science, and LILACS (VHL) were searched from inception to July 2018 using combinations of the search terms ""virulence factor*"", ""Leishmania"", and ""Leishmaniasis*"", while accounting for unique database syntax. Iterative inclusion and exclusion of search terms was employed to maximize relevant article extraction. For the systematic review, we will include primarily molecular and mechanistic pathogenesis studies in various model systems, observational studies, review studies, cohort studies, as well as clinical trials. Of 2620 articles remaining after title and abstract screening, some major VFs identified in the Leishmania genus are: heat shock proteins (HSP23, HSP70), cysteine peptidases (CPB), mannose phosphate isomerases (MPI), metalloproteases (GP63), and elongation factors (EF1alpha), among many others. Data will be grouped and summarized by species, geographic region of endemicity, and VFs. This systematic compilation of mechanistic VF data will add to the large body of work in molecular pathogenesis of kinetoplastids and enhance our understanding of species and regional variations in Leishmania pathogenesis.