KEYNOTE:

Dr. Kirk Bergstrom

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Dr. Kirk Bergstrom obtained his BSc in Biology at University of Northern British Columbia (Prince George, BC), and graduated in May 2001. He then worked as a research assistant in the lab of biochemist Chow H. Lee at UNBC, where he purified and characterized a novel endoribonuclease that can degrade oncogene c-myc messenger RNA *in vitro*. In 2004, Dr. Bergstrom shifted his focus to studying innate immunity in the intestinal tract toward gut pathogens, as a graduate student in lab of innate immunologist Dr. Bruce Vallance at UBC-Vancouver; here he discovered novel roles of mucus producing goblet cells in host defense against *Enterpathogenic E. coli*-related pathogens in vivo. Following his graduation in 2011, Dr. Bergstrom went to Oklahoma Medical Research Foundation (Oklahoma City, USA) for his postdoctoral studies in the lab of glycobiology expert Dr. Lijun Xia, to study a key aspect of the gut mucus system – mucin-type O-glycosylation – in relation to homeostasis with our diverse microbiota. During this time, Dr. Bergstrom uncovered new insights into how mucin-type O-glycans regulate microbial communities to protect against spontaneous inflammation and cancer throughout the gastrointestinal tract in mouse models. In 2019, Dr. Bergstrom moved back up to BC to begin an independent position at UBC's Okanagan campus where he is exploring novel genetic systems that regulate the O-glycan microenvironment and microbial symbiosis in vivo to protect against microbiota-driven chronic diseases such as inflammatory bowel disease and colorectal cancer.

Holistic glycoscience: Combining the power of preclinical models of glycosylation deficiencies with glycomics to interrogate the biologic functions of glycans and their defects in chronic diseases

Many chronic diseases are associated with altered glycosylation of glycoconjugates (e.g. glycoproteins, glycolipids, proteoglycans, and perhaps the newly described glycoRNA) on and within tissue cells and their secretions. The challenge in these settings is to distinguish which glycan alterations are primary to the disease process, and which are secondary, i.e. a consequence of the disease itself with no bearing on pathogenesis. Addressing this challenge is crucial for understanding the biologic functions of glycans in normal physiology and in pathologic processes underlying chronic diseases. Using inflammatory bowel disease (IBD) and inflammation-associated colon cancer as "prototypes" for glycan defects in chronic diseases, in parallel with novel transgenic strategies in preclinical models (e.g. mice) that mimic these conditions, we have provided fascinating new insights into not only how glycans function in the healthy state, but also how glycosylation defects (specifically of O-glycans) predispose to chronic inflammation and carcinogenesis. Further, glycomic methods applied to these in vivo disease models reveal hidden information on how glycan defects modify the glycome—often in unexpected ways—and point to novel non-invasive biomarker strategies. These principles will be demonstrated herein this presentation. Ultimately we hope to make that case that combining glycomic approaches with preclinical models represents a particularly strategic way to advance the application of glycoscience in the identification of (i) novel underlying mechanisms; (ii) therapeutic targets; and (iii) biomarkers for chronic inflammatory diseases impacting Canadians and citizens around the world.