

Department of Molecular and Cellular Biology
Graduate Seminar MCB*7500

Friday, Sept. 28, 2018 in SSC 1511 @ 12:45 p.m.

presented by:

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(Advisor: E. Allen-Vercoe)

“Characterizing the gut microbial ecosystem of infants genetically predisposed to developing Type 1 Diabetes”

Type 1 Diabetes (T1D) is a genetic autoimmune disease in which T lymphocytes destroy the insulin-producing β -cells of the pancreas. In recent decades, the incidence of T1D has drastically increased worldwide at a rate that cannot solely be explained by genetic susceptibility. This may suggest that environmental risk factors, such as changes in dietary habits and widespread use of antibiotics, which accompany life within the modern environment play a significant role in the pathogenesis of the disease. Evidence arising from both human and animal studies indicate that an altered gut microbiome is strongly associated with development of the disease. Environmental triggers that impact the gut microbiome may include early exposure to gluten, milk proteins and antibiotics. However, studies investigating these triggers have conflicting results and there is an overall lack of understanding of the underlying mechanisms at play. Thus, this study aims to characterize—using the unique ‘Robogut’ platform developed in the Allen-Vercoe Laboratory—the microbial abundance, functional complexity and metabolic output of fecal communities derived from infants that are genetically predisposed to developing T1D. Subsequently, defined communities will be derived from these fecal communities, which will then be exposed to gluten, casein and ampicillin. The resulting changes in species abundance and metabolic output will help to elucidate the role of these stressors in the development of T1D. Lastly, the inflammatory response of intestinal epithelial cells to the metabolites produced during the exposures will be determined. If new biomarkers of T1D progression are identified, these may be used as potential therapeutic targets to modulate or prevent islet autoimmunity.