



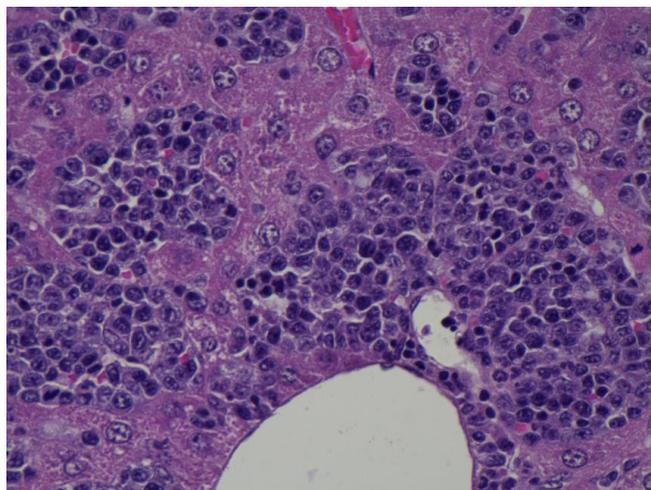
Chemical & Physical Sciences  
UNIVERSITY OF TORONTO  
MISSISSAUGA

COLLOQUIUM SEMINAR TALK  
WEDNESDAY, MARCH 21, 2018  
3:10PM  
**KN L1220**

## Dr. Richard Moriggl

*Director, Ludwig Boltzman Institute for Cancer Research  
Medical University of Vienna and the  
University of Veterinary Medicine in Vienna*

### **Gain of function mutations in the JAK-STAT core cancer pathway promote oncogene transcription and how it can be targeted**



The talk will describe how mutations of the JAK tyrosine kinases – STAT transcription factors form a core cancer pathway. This signaling cascade is downstream of cytokine and growth factor activation where it promotes cancer cell growth and survival through gene transcription reprogramming. We begin to understand how the JAK-STAT pathways controls also chromatin remodelling. We focus on innovative therapy approaches to block hyperactivity of the JAK-STAT pathway using small molecule weight inhibitors. Special attention is given to oligomeric STAT5 transcription factors to drive hematopoietic cancers since that promotes high oncogene transcription culminating into cancer progression. We

generated hyperactive STAT5 and JAK2 mouse models with graded activity levels as preclinical animal models to explore mechanistic cancer insights and how we can interfere with these malignant processes. One spotlight on conservation across species of the JAK-STAT core cancer pathway is given with comparative pathology examples. In summary, the talk will illuminate mechanistic insights into gene regulation through the JAK-STAT core cancer pathway and how it can be inhibited by chemical structures targeting either the JAK tyrosine kinases or their downstream STAT3/5 transcription factors to translate findings from basic research towards clinical application.

[Dr. Richard Moriggl](#) received his PhD from the University of Freiburg, Germany where he began his research in cancer immunology and the JAK-STAT pathway. During his post-doc at St. Jude Children's Research Hospital, he was among the first to use STAT-deficient models to establish the important role of this pathway in T cell biology and cancer development. Dr. Moriggl is internationally recognised for his contribution in the field with over 148 highly cited publications and numerous lectures as well as the generation of key JAK-STAT-specific tools including various mouse models (full and conditional knock-outs and transgenics for gain of function mutations, compound models of high tyrosine kinase signaling), genomics approaches and small molecule inhibitor design carried out in close collaboration with the lab of Patrick Gunning.

