Abstract:
Germline gain-of-function (GOF) mutations in the transcriptional factor signal transducer and activator of transcription 3 (STAT3) promote early-onset multisystemic autoimmunity. To investigate how increased STAT3 promotes systemic inflammation, we generated a transgenic knock-in strain expressing a pathogenic human mutation Stat3^K392R within the endogenous murine locus. This talk will cover a brief introduction into the relevant immunology and pathology, (including the markers used by flow cytometry), as well as data that I generated for this project. Together, these data provide new insights into this complex syndrome and highlight the diverse cellular mechanisms by which dysregulated STAT3 activity promotes breaks in immune tolerance.