

HDACs as regulators of T cell-mediated immunity in health and disease

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SFB F70 Seminar - ONLINE

Host-pathogen interactions in COVID-19: What we learn from genomics data

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Registration

Wednesday, 24th of March, 2021, 16:00-16:45 Uhr

Location: Online seminar - Zoom ([link](#))

Host: Michael Bonelli

Biosketch

Dr. Kazemian is trained in both computer science and molecular biology. His PhD training in the Department of Computer Science at University of Illinois at Urbana-Champaign was focused on locating gene enhancers and modeling their function, as enhancers are one of the main players of gene transcriptional regulation. His postdoctoral training in the Laboratory of Molecular Immunology at National Heart, Lung, and Blood Institute (NHLBI) was on utilizing the power of high-throughput sequencing data such as RNA-seq, ChIP-seq, ATAC-seq and Hi-C to characterize pathogen interactions with the host immune system. He is now an Assistant Professor at Purdue University with a joint appointment between the Department of Biochemistry, the Center for Cancer Research, and the Department of Computer Science. His lab primarily focuses on integrating computational and experimental approaches to study pathogen interactions with host cells and the immune system in infectious diseases and cancers. In particular his lab utilizes emerging genomics, transcriptomics, and epigenetics assays and computational models to identify novel pathways, new regulatory noncoding RNAs, and new host-pathogen interactions that could trigger disease.



Selected recent publications

- Kolev M et al., Diapedesis-Induced Integrin Signaling via LFA-1 Facilitates Tissue Immunity by Inducing Intrinsic Complement C3 Expression in Immune Cells. *Immunity* (2020) 52:513-527.e8
- Povoleri et al. Human retinoic acid-regulated CD161+ regulatory T cells support wound repair in intestinal mucosa. *Nat Immunol* (2018) 19:1403-1414
- McGregor et al. An Autocrine Vitamin D-driven Th1 shutdown program can be exploited for COVID-19. *bioRxiv* 2020.
- Yan et al. SARS-CoV2 drives JAK1/2-dependent local and systemic complement hyperactivation. *ResearchSquare* 2020.
- Yan et al. Host-virus chimeric events in SARS-CoV2 infected cells are infrequent and artifactual. *bioRxiv* 2021.