Microbial and environmental factors that control gut-resident memory T cells in human health and disease: molecular signatures, function, and interaction partners

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The human intestine harbours a vast and diverse bacterial community that exerts several beneficial effects on the host such as a profound effect on immune responses. Maladaptation of this host-microbe dialogue can promote inflammatory responses and is implicated in various pathologies including inflammatory bowel disease (IBD). However, the microbial signals and molecular pathways that promote tissue-specific differentiation of gut-resident immune cells are still poorly characterized. Using cutting-edge technologies, a multidisciplinary approach, well-defined patient cohorts, and mouse models of colitis, this project aims at deciphering the complex host-microbiota relationship, i.e. the interactions between microbial, environmental, and inflammatory factors that promote intestinal inflammation. The overall goal is to utilize the acquired knowledge to identify targetable cytokine signals and pathogenic molecular pathways in microbiota-specific CD4+ T cell populations for therapeutic development in IBD.

Projektbeteiligte

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Open Access-Publikationen

Single-Cell Transcriptomics of Regulatory T Cells Reveals Trajectories of Tissue Adaptation
Th2 cells lacking T-bet suppress naive and memory T cell responses via IL-10.
Plasticity and lineage commitment of individual Th1 cells are determined by stable T-bet expression quantities.
Statistical Inference of Enhancer-Gene Networks Reveals Pivotal Role of T-bet Expression Intensity for T Helper Cell Fate.
Dissecting the dynamic transcriptional landscape of early T helper cell differentiation into Th1, Th2, and Th1/2 hybrid cells.