INFLAMMATION RESEARCH

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SUMMARY & MISSION STATEMENT

Inflammation Research: From an evolutionary perspective, the complex mammalian immune system developed to combat microbial threats. The flip side of this protective system is however that aberrant and deregulated immune responses can lead to **immune-mediated pathologies** as seen in **chronic inflammatory or autoimmune diseases**. Cancer represents largely the lack of 'attention' by the immune system

OVERVIEW

Fundamentally, deregulated communication between immune cells is the reason for unwanted immune responses. For the complex immune system to work, the individual cell types have not only specialized functions, but also a complex communication network. **Cytokines** are soluble factors with the capacity to serve as signals for the communication (*or words in the complex language*) between immune cells. Our goal is to uncover this communication network and to translate the ***language of the immune system*.**

Related to our studies of autoimmunity (an undesired process) we expanded our interest to apply our tool-set and expertise to study the impact of immunity to combat cancer (a desired process).

Our main research interests can be categorized as such:

- **Cytokine networks** in chronic inflammatory disease with a focus on in vivo modeling of *multiple sclerosis, psoriasis, graft-versus host disease*
- **Immune tolerance** and lymphoid development
- **Cancer-immunotherapy**: specifically the interaction of immune cells with cancer cells and therapeutic interventions to mount immune responses against tumors

SELECTED CANCER RELATED PUBLICATIONS

High dimensional single cell analysis predicts response to anti-PD-1 immunotherapy. Krieg C, Nowicka M, Guglietta S, Schindler S, Hartmann FJ, Weber LM, Dummer R, Robinson MD, Levesque MP, and Becher B. **Nat. Med.** 2018;24(2):144-153

High-Dimensional Single-Cell Mapping of Central Nervous System Immune Cells Reveals Distinct Myeloid Subsets in Health, Aging, and Disease. Mrdjen D, Pavlovic A, Hartmann FJ, Schreiner B, Utz SG, Leung BP, Lelios I, Heppner FL, Kipnis J, Merkler D, Greter M and Becher B. **Immunity.** 2018;48(2):380-95 e6

Tissue microenvironment dictates the fate and tumor-suppressive function of type 3 ILCs. Nussbaum K, Burkhard SH, Ohs I, Mair F, Klose CSN, Arnold SJ, Diefenbach A, Tugues S and Becher B. **J Exp Med.** 2017;214(8):2331-47.

Interleukin-12 bypasses common gamma-chain signalling in emergency natural killer cell lymphopoiesis. Ohs I, van den Broek M, Nussbaum K, Munz C, Arnold SJ, Quezada SA, Tugues S and Becher B. **Nat Commun.** 2016;7(13708.

IL17A-Mediated Endothelial Breach Promotes Metastasis Formation. Kulig P, Burkhard S, Mikita-Geoffroy J, Croxford AL, Hovelmeyer N, Gyulveszi G, Gorzelanny C, Waisman A, Borsig L and Becher B. **Cancer immunology research.** 2016;4(1):26-32.

High-dimensional analysis of the murine myeloid cell system. Becher B, Schlitzer A, Chen J, Mair F, Sumatoh HR, Teng KW, Low D, Ruedl C, Riccardi-Castagnoli P, Poidinger M, Greter M, Ginhoux F and Newell EW. **Nat Immunol.** 2014.

Intratumoral IL-12 combined with CTLA-4 blockade elicits T cell-mediated glioma rejection.Vom Berg J, Vrohlings M, Haller S, Haimovici A, Kulig P, Sledzinska A, Weller M and Becher B. **J Exp Med**. 2013;210(13):2803-11.

IL-12 initiates tumor rejection via lymphoid tissue-inducer cells bearing the natural cytotoxicity receptor NKp46. Eisenring M, vom Berg J, Kristiansen G, Saller E and Becher B. **Nat Immunol** 2010;11: 1030-1038.