

## Research Interests – Prof. Dr. Eicke Latz

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A key task of immune cells is to maintain tissue homeostasis, which is essential for unperturbed function of organs. Immune cells circulate within the vasculature and are resident cells in all tissues allowing them to quickly detect and respond to any disturbance of homeostasis. Immune cells of the innate arm of the immune system gather information about the functioning and health of the tissues through an array of germ-line encoded immune signaling receptors.

Our laboratory is interested in elucidating the molecular mechanisms by which innate immune signaling receptors allow cells to detect the presence of unusual or excess of molecules arising during situations of infection, tissue damage or metabolic derangements. We further study how the sensory input collected by immune cells during these challenges is processed and integrated and subsequently translated into an effector response. Cellular activation can further result in functional and epigenomic reprogramming of the responding cell allowing for an adjusted future response. We aim to decipher how this process, called “innate immune memory”, is regulated and how dysregulated innate immune memory influences inflammatory processes and can contribute to disease development.

The same innate immune signaling receptors that are critical for protective anti-infectious immune responses have been identified as the mediators of the heightened inflammatory state that is found in several non-communicable diseases. Among these innate immune signaling sensors, the so-called NLRP3 inflammasome mediates the activation of highly pro-inflammatory mediators, which are of particular relevance for the pathogenesis of many common diseases, including type II diabetes, atherosclerosis or Alzheimer’s disease. We study the mechanisms that explain how NLRP3 can respond to a range of substances occurring in tissues as a consequence of ageing, physical inactivity, over-nutrition or environmental factors. We further aim to elucidate the mechanisms that regulate and limit the NLRP3 pathway. We hope that these studies may form the basis for developing novel approaches for pharmacological intervention to fight cancers and infections and to limit inflammatory processes in common non-communicable diseases.