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# Dynamic imaging of protective mast cells in living mice during severe contact hypersensitivity

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Contact hypersensitivity (CHS) is a common skin disease induced by epi-cutaneous sensitization to haptens. Conflicting results have been obtained regarding pathogenic versus protective roles of mast cells in CHS and this has been attributed in part to the limitations of certain models for studying mast cells functions in vivo. Here we describe a new fluorescent imaging approach that enables in vivo selective labeling and tracking of mast cell secretory granules by real-time intra-vital 2-photon microscopy in living mice and permits the identification of such mast cells as a potential source of cytokines in different disease models. We show using this method that dermal mast cells release their granules progressively into the surrounding microenvironment, but also represent an initial source of the anti-inflammatory cytokine IL-10, during the early phase of severe CHS reactions. Finally, using 3 different types of mast cell-deficient mice, as well as mice in which IL-10 is ablated specifically in mast cells, we show that IL-10 production by mast cells can significantly limit the inflammation and tissue pathology observed in severe CHS reactions.

## Biography

Nicolas Gaudenzio is an Assistant Professor in the field of Neuroimmunology and Allergic Skin Inflammation. After a productive experience as Post-doctorate, then as Instructor and as Intravital Imaging Specialist for the Department of Pathology at Stanford, he has now created a cross-disciplinary research group in Europe. His work has contributed substantially to identify molecular and cellular targets involved in allergic skin inflammation (such as atopic dermatitis) and to develop new intra-vital imaging methods to probe immune interactions in preclinical disease models. His expertise and creativity in these fields can contribute to apprehend the role of structural, neuronal and immune cells during the development of allergic inflammation and might help to identify new therapeutic options to modulate and prevent disease development.

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