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Research interests

The major research interest of my group is to unravel the immune evasion mechanisms used by human rhinoviruses (HRV) in order to identify molecular checkpoints of the immune system and to better understand the pathogenesis of the common cold, one of the most frequent infectious diseases in humans.

Thesis projects

Project 1: Molecular and functional characterization of IL-35 producing regulatory T cells (IL-35-Tregs) induced by human rhinoviruses (HRV)

Human rhinovirus (HRV) are the major cause of the common cold, one of the most frequent infectious disease in humans. We have recently demonstrated that dendritic cells (DCs) activated by HRV (R-DCs) induce IL-35-mediated suppressor function in T cells from human peripheral blood. Induction of IL-35⁺ Tregs by R-DCs was triggered by a combinatorial signal delivered from DCs to T cells via B7-H1 (CD274) and sialoadhesin (CD169). We now propose to analyze in this project the molecular mechanisms on the T cell side that cause the IL-35⁺ Treg differentiation program and to characterize this novel human Treg subset in HRV-infected patients.

Project 2: Analysis of the role of cysteamine dioxygenase in the antiviral state of DCs against HRV infection

DCs are known as specialists in sensing pathogens and inducing appropriate innate as well as adaptive immune responses. We have recently demonstrated that human DCs use an alternative activation program upon HRV entry and block HRV replication. We have now discovered that cysteamine (2-aminoethanethiol) dioxygenase (ADO) in DCs contributes to the resistance against HRV infection and transduction of epithelial Hela cells with the gene encoding ADO prevented HRV infection *in vitro*, ADO is a recently identified and unique enzyme that metabolizes cysteamine into taurine, an amino acid with known anti-inflammatory and also antiviral properties. We now propose to analyze in this project how ADO expression and function is regulated in DCs, and to examine if taurine is a potential novel reagent to treat HRV infection.

Selected publications

Kirchberger, S., O. Majdic, P. Steinberger, S. Bluml, K. Pfistershammer, G. Zlabinger, L. Deszcz, E. Kuechler, W. Knapp, and **J. Stockl**. 2005. Human rhinoviruses inhibit the accessory function of dendritic cells by inducing sialoadhesin and B7-H1 expression. **J Immunol** 175:1145.

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Schrauf, C., S. Kirchberger, O. Majdic, M. Seyerl, G. J. Zlabinger, K. M. Stuhlmeier, M. Sachet, J. Seipelt, and **J. Stockl**. 2009. The ssRNA genome of human rhinovirus induces a type I IFN response but fails to induce maturation in human monocyte-derived dendritic cells. **J Immunol** 183:4440.

Seyerl, M., S. Kirchberger, O. Majdic, J. Seipelt, C. Jindra, C. Schrauf, and **J. Stockl**. 2009. Human rhinoviruses induce IL-35-producing Treg via induction of B7-H1 (CD274) and sialoadhesin (CD169) on DC. **Eur J Immunol** 40:321.