

**Frank VERHOEVEN <fverhoeven@chu-besancon.fr> Master 2 internship
project
Year 2024-2025**

Laboratory/Institute: TIMC

Team: T-RAIG

Director:

Head of the team: A. Baillet/B. Huard

Name and status of the scientist in charge of the project:

Frank VERHOEVEN

HDR: yes

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Program of the Master's degree in Biology:

- Immunology, Microbiology, Infectious Diseases Structural Biology of Pathogens
 Physiology, Epigenetics, Differentiation, Cancer Neurosciences and Neurobiology

Title of the project:

Impact of IL33 on MAIT, ILC2 and intestinal permeability prior the onset of arthritis in SKG mice, a mouse model of reactive arthritis

Objectives (up to 3 lines):

This project aims to demonstrate the impact of IL 33 on ILC2 and MAIT activation and on the intestinal permeability prior the onset of the arthritis in the SKG model

Abstract (up to 10 lines):

An increase in intestinal permeability has been described in spondyloarthritis. A recent study showed, in a rat model of reactive arthritis, that this increase of intestinal permeability precedes the onset of arthritis with an important place of IL33. In order to investigate the role of IL 33 on intestinal permeability and immune cells in the arthritis development, we use an animal model, the ZAP-70(W163C) - mutant BALB/c (SKG) mice that develop arthritis after a subcutaneous injection of curdlan 3mg whereas the BALB/c mice do not. The present project will aim to compare the increased intestinal expression of IL33 and the behavior of innate immune cells from SKG and BALB/c mice. MAIT and ILC2 will be isolated from mouse ileon (SKG and BALB/c). Cytokine secretion (IL 17 and IL23) will be analyzed in presence or not with IL33. The presence of intestinal permeability will be assessed by FITC Dextran before the onset of arthritis (Day 5) and at the onset of arthritis (Day 15).

Methods (up to 3 lines):

Cell culture and cell isolation, cell stimulation, co-culture, flow cytometry, multiplex analysis, western blot, cell lysate, enzymatic activity, rt-PCR, FITC Dextran

Up to 3 relevant publications of the team:

1) Romand X *et al* - Mediation of Interleukin-23 and Tumor Necrosis Factor-Driven Reactive Arthritis by Chlamydia-Infected Macrophages in SKG Mice (2021) *Arthritis Rheumatol.* 73: 1200-1210.

2) Baillet A. *et al* - High Chlamydia Burden Promotes Tumor Necrosis Factor-Dependent Reactive Arthritis in SKG Mice. (2015) *Arthritis Rheumatol.* 67: 905-912.

Requested domains of expertise (up to 5 keywords):

Innate immunity, inflammation, immunology, biochemistry, cell biology