

Up to 3 relevant publications of the team:

## Master's degree in Biology – Chemistry-Biology Department

## Master 2 internship project Year 2025-2026

fear 2025-2026
Laboratory/Institute: TIMC UMR5525 CNRS/UGA Director: Pr. Alexandre Moreau-Gaudry Team: T-RAIG Head of the team: Prs. Bertrand Huard and Athan Baillet
Name and status of the scientist in charge of the project: Dr. Dalil Hannani, CNRS researcher / Dr. Frank Verhoeven, Rheumatologist
HDR: yes ■ no □  Address: Laboratoire TIMC – Faculté de Médecine de Grenoble – Bâtiment Jean Roget –  Domaine de la Merci 38700 La Tronche  Phone: - e-mail: Dalil.Hannani@univ-grenoble-alpes.fr
Program of the Master's degree in Biology:
<ul> <li>Microbiology, Infectious Diseases and Immunology □ Biochemistry &amp; Structure</li> <li>□ Physiology, Epigenetics, Differentiation, Cancer □ Neurosciences and Neurobiology</li> </ul>
<u>Title of the project</u> : Exploration of the Gut / Joint axis immunity in SpondylArthritis development
Objectives (up to 3 lines):
The aim of this internship is to dissect the immune mechanisms and cell populations that originate from the Gut and are involved in the Arthritis development
Abstract (up to 10 lines):
SpondylArthritis (SpA) is chronic rheumatic disease characterized by inflammation of the spine and sacroillar joints. The primary lesion is the enthesitis located in peripheral et axial entheses. The relationship between SpA and inflammatory bowel disease is closely linked to dysbiosis and gut inflammation. We hypothesized that immune cell activation originates from the gut and migrate toward joint to cause pathological lesions. We have recently shown, using our unique SKG mouse model that fully recapitulate SpA pathophysiology, that some immune cell populations are activated within the gut early after the injection of curdlan (arthritis trigger) before the onset of the arthritis. These cells are then detected in the periphery (Spleen) before accumulating within the joints at the onset and development of arthritis. We also observed a cytokine skewing toward IL-13 pathway. We know aim at dissecting the precise mechanism of action involved in these cell recruitment as well as study other very promising immune cell populations.
Methods (up to 3 lines):
Mouse experimentation. Ex vivo analyses. Flow Cytometry Analysis. In vitro co-culture. RT-qPCR.



## Master's degree in Biology – Chemistry-Biology Department

1. Increased gut permeability and intestinal inflammation precede arthritis onset in the adjuvant-induced model of arthritis.

Hecquet S, Totoson P, Martin H, Algros MP, Saas P, Pais-de-Barros JP, Atchon A, Valot B, Hocquet D, Tournier M, Prati C, Wendling D, Demougeot C, Verhoeven F. Arthritis Res Ther. 2023

2. Mediation of Interleukin-23 and Tumor Necrosis Factor-Driven Reactive Arthritis by Chlamydia-Infected Macrophages in SKG Mice.

Romand X, Liu X, Rahman MA, Bhuyan ZA, Douillard C, Kedia RA, Stone N, Roest D, Chew ZH, Cameron AJ, Rehaume LM, Bozon A, Habib M, Armitage CW, Nguyen MVC, Favier B, Beagley K, Maurin M, Gaudin P, Thomas R, Wells TJ, Baillet A. Arthritis Rheumatol. 2021

3. High Chlamydia Burden Promotes Tumor Necrosis Factor-Dependent Reactive Arthritis in SKG Mice. Baillet AC, Rehaume LM, Benham H, O'Meara CP, Armitage CW, Ruscher R, Brizard G, Harvie MC, Velasco J, Hansbro PM, Forrester JV, Degli-Esposti MA, Beagley KW, Thomas R. Arthritis Rheumatol. 2015

Requested domains of expertise (up to 5 keywords):

Immunology and Cell Biology.