

Université Claude Bernard Lyon 1- Hosting offer for a MSCA Post-doctoral fellowship candidate in Cell Biology, Immunology

Host Organisation	Université Claude Bernard Lyon 1
Department	Biology
Laboratory	Centre International de Recherche en Infectiologie (CIRI)
Website (lab / research team)	https://ciri.ens-lyon.fr/teams/nlrp3
Supervisor Contact name	Bénédicte PY
Supervisor Contact email	Benedicte.py@inserm.fr

Host Organisation

With 62 laboratories and more than 7000 publications per year, and leading French university in terms of the number of patents filed in collaboration with industry, Lyon 1 contributes to scientific and innovation progress in numerous fields: health, mathematics, IT, physics, chemistry, earth and space sciences, life sciences, etc. Creator of emerging knowledge and new technologies, the University is consolidating its research excellence on a global and international level by developing inter- and multidisciplinary approaches targeting the major challenges facing today society.

Host research lab/team:

The *Centre International de Recherche en Infectiologie (CIRI)* (<https://ciri.ens-lyon.fr/>) is a leading research lab in Europe in the field of infectious diseases. The Center gathers Immunologists, Bacteriologists and Virologists (27 teams, >400 employees) to answer the new and older challenges in the fields of infection. It is part of the Bioscience Lyon-Gerland Campus (ENS-Lyon), and have access to top technology platforms (rodent SPF facility with A2/A3 and transgenesis services, microscopy, cytometry, protein preparation and analysis including mass spectrometry).

The “**NLRP3 inflammasome and Immune Response to Sepsis**” (<https://ciri.ens-lyon.fr/teams/nlrp3>) research team is highly international, with English as the working language, and has on-going collaborations with Germany, Ireland, US, China, Italy and Korea.

Their research focuses on the **regulation of inflammation and its impacts in diseases with special interest on sepsis, hereditary auto-inflammation (CAPS) and diabetes, using biochemistry, cell biology, mouse model and patient cohort analysis approaches**. The NLRP3 inflammasome pathway controls the release of major inflammatory cytokines (IL-1 β and IL-18) and a pro-inflammatory cell death responsible for the release of many alarmins. NLRP3-dependent inflammation contributes to many highly common metabolic diseases (diabetes, liver disease, obesity, gouty arthritis), neurodegenerations (Alzheimer’s and Parkinson’s diseases, age-associated decline), and vascular diseases (atherosclerosis). In addition, gain-of-function mutations in the NLRP3 gene cause the rare Cryopyrin-associated periodic syndrome (CAPS) hereditary autoinflammations. Better understanding of the molecular mechanisms of NLRP3 regulation and activation is critical in the development of specific inhibitors.

Using biochemistry and cell biology approaches, we decipher the molecular mechanisms of the inflammasome assembly.¹⁻³ Using transgenic mouse lines, we validated our results in vivo and investigate their impact in various diseases

(endotoxic choc, diabetes models).⁴ **Finally, we investigate the role of the inflammasome pathway in immune dysregulation associated with sepsis using mouse models and patients' cohorts.**^{5,6,8}

Hosting Offer

The “NLRP3 inflammasome and Immune Response to Sepsis” team offers to host a MSCA Postdoctoral Fellowship candidate (typically a post-doc of less than 8 years research experience since PhD defence), submitting an application to the next MSCA-2025 - PF call for proposals (deadline 9^h September 2026), interested to work on the following research topic:

- Regulation of the inflammasome through NLRP3 post-translational modifications (combining biochemistry, cell biology and mouse models)
- Impact of the inflammasome pathway in immune alterations following sepsis (mouse models and/or cohort studies)

The fellowship could last for 12 to 36 months, depending on the type of Postdoctoral Fellowship.

Supervision

The Post-doctoral fellow will be supervised by Dr Bénédicte F. Py and/or Pr Fabienne Venet.

Dr Bénédicte Py is head of the team. Her research interests include the molecular mechanisms of inflammasome NLRP3 regulation, and its implication in CAPS autoinflammation and metabolic diseases (diabetes model)

Pr Fabienne Venet is a full Professor of immunology and a clinician, with a strong expertise in the immunosuppression associated with sepsis. She developed both mouse models of sepsis induced immune alterations in the CIRI, and septic patient cohorts in the hospital.

Application process

Interested candidates are invited to contact us exclusively by email at benedicte.py@inserm.fr

Make sure that you include the reference to this offer in the title of your email. Please attach **a CV, a motivation letter**, and a 1 page research proposal (optional).

Professional grant application support:

Candidates will receive the support of the supervisors, as well as online training from a professional grant application company, and advices from successful applicants, to prepare and submit their application with the CIRI “NLRP3 inflammasome and immune response to sepsis team” as a host laboratory, to the next MSCA-PF call for proposals.

1. Cosson, C. *et al.* Functional diversity of NLRP3 gain-of-function mutants associated with CAPS autoinflammation. *J. Exp. Med.* **221**, e20231200 (2024).
2. Py, B. F. Functional analysis of NLRP3 variants provides insight into inflammasome regulation. *Nat. Immunol.* **26**, 337–339 (2025).
3. Py, B. F., Kim, M. S., Vakifahmetoglu-Norberg, H. & Yuan, J. Deubiquitination of NLRP3 by BRCC3 critically regulates inflammasome activity. *Mol Cell* **49**, 331–8 (2013).
4. Niu, T. *et al.* NLRP3 phosphorylation in its LRR domain critically regulates inflammasome assembly. *Nat. Commun.* **12**, 5862 (2021).
5. Coudereau, R. *et al.* Altered Ex Vivo NLRP3 Inflammasome Activation Is Associated with 28-Day Mortality in Septic Patients. *Viruses* **15**, (2023).
6. Coudereau, R. *et al.* Persistent NLRP3 inflammasome activation is associated with delayed immunosuppression in septic patients. *J. Leukoc. Biol.* **115**, 706–713 (2024).
7. Gustave, C.-A. *et al.* Septic Shock Shapes B Cell Response toward an Exhausted-like/Immunoregulatory Profile in Patients. *J. Immunol.* **1950** **200**, 2418–2425 (2018).
8. Gossez, M. *et al.* PD-L1+ plasma cells suppress T lymphocyte responses in patients with sepsis and mouse sepsis models. *Nat. Commun.* **16**, 3030 (2025).