# Want to apply for a MSCA-PF at VHIR?

Check our list of proposed topics

**The Vall d'Hebron Institut de Recerca is looking for applicants to apply for the Marie Skłodowska-Curie Actions Postdoctoral Fellowship 2026 call**

The Vall d'Hebron Research Institute (VHIR) is inviting postdoctoral researchers to apply to the **Marie Skłodowska-Curie Actions Postdoctoral Fellowships (MSCA-PF) 2026** together with our research groups.
This catalogue brings together a selection of **topics proposed by VHIR research groups currently interested in hosting outstanding postdoctoral candidates**. If you are looking for a host institution in Spain and are interested in developing your project in a leading biomedical research environment, we encourage you to explore the opportunities listed here.
VHIR is a leading biomedical research centre promoting research, innovation and training within the Vall d'Hebron Barcelona Hospital Campus, the largest hospital complex in Barcelona and one of the main centres within the Catalan Institute of Health (ICS).
Since its foundation in 1994, VHIR has been committed to addressing major health challenges through excellence in clinical and translational research. Its close integration with Vall d'Hebron University Hospital (HUVH) enables a strong bench-to-bedside approach, ensuring that scientific advances are rapidly translated into improved diagnostics, therapies and patient outcomes.
VHIR is internationally recognised as a highly competitive European research institute and a preferred partner for industry, hosting over 1,000 active clinical studies in 2026 and positioning the institution as an international benchmark in biomedical and clinical research.

## A highly collaborative environment for MSCA fellows

VHIR offers a **structured and highly interconnected scientific environment**, currently comprising **63 research groups,** designed to foster interdisciplinary research and collaboration. Its scientific organisation is based on four complementary building blocks (*Figure 1*):

- **5 Research Areas**, which constitute the main structural axis according to major biomedical disciplines and clinical specialities

- **1 Research Hub**, currently focused on paediatric and perinatal research, acting as a strategic platform to accelerate translational research and clinical implementation.

- **6 COREs (Challenge-Oriented Research)**, which are transversal, flexible and multidisciplinary spaces addressing strategic and emerging scientific challenges, bringing together expertise to generate new ideas and build competitive high-impact research lines aligned with European priorities.

- **2 Programs**, promoting interdisciplinary approaches in areas such as gender and diversity in research or advanced technologies.

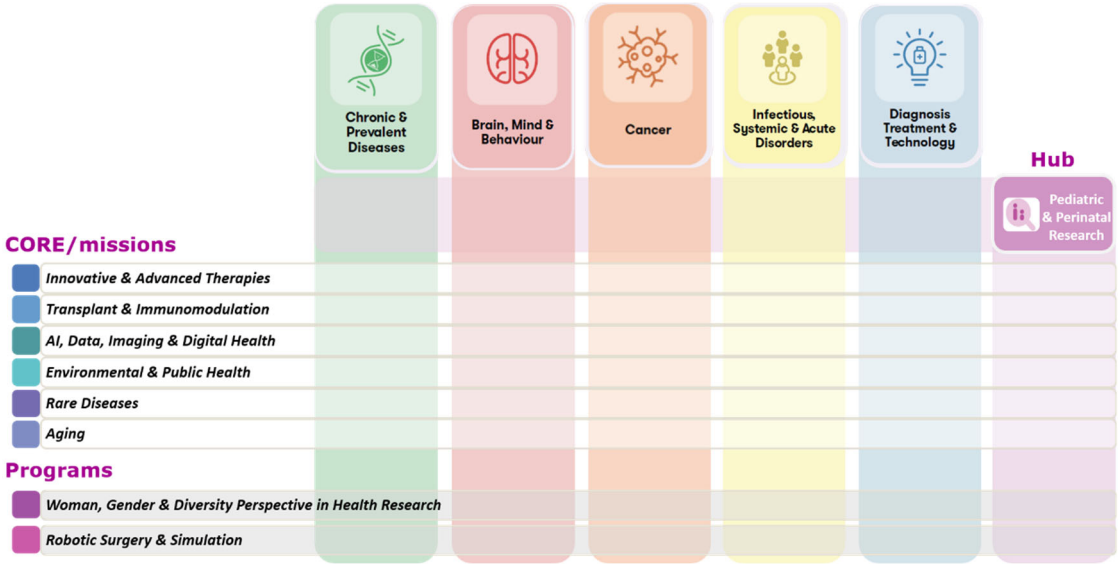


***Figure 1:*** *Scientific Structure at VHIR: Research Areas, Hubs, COREs and Programs*

Within this framework, research groups are anchored in a specific **Research Area**, while principal investigators may simultaneously contribute to the **Hub, COREs and programs.** This structure creates a **coherent and highly collaborative ecosystem**, facilitating cross-disciplinary interaction and enabling **MSCA fellows** to engage with multiple groups, access complementary expertise and develop ambitious research projects.

## Research themes and icons in this catalogue

Each research proposal in this catalogue is labelled with a set of **icons** to help candidates quickly identify its scientific scope and collaborative potential:

| Research Area | Description | Nº of Research Groups |
|---|---|---|
| **Chronics & Prevalent Diseases** | Understanding and management of high-burden chronic diseases to improve patient outcomes and quality of life | 13 |
| **Brain, Mind & Behaviour** | Research on the mechanisms, diagnosis and treatment of neurological and mental health disorders, promoting integrated and precision approaches. | 15 |
| **Cancer** | Oncology research aimed at improving detection, biological characterisation and treatment of cancer, fostering innovative therapies and efficient clinical translation. | 10 |
| **Infectious, Systemic & Acute Disorders** | Research addressing infectious, systemic and acute processes, with emphasis on improving diagnosis, clinical response and prevention strategies. | 10 |
| **Diagnosis, Treatment & Technology** | Research focused on the development and validation of diagnostic and therapeutic technologies that facilitate innovation and clinical implementation. | 15 |

| HUB | Description | Nº of Research Groups |
|---|---|---|
| **Paediatric & Perinatal Research Hub** | Strategic and integrated platform aimed at accelerating translational research from prenatal stages to adolescence, fostering clinical implementation and innovation in paediatric health. | 30 |

| COREs | Description | Nº of Principal Investigators |
|---|---|---|
| **Innovative & Advanced Therapies** | Research on the development of advanced and emerging therapies (cellular, gene and biological therapies), promoting their translation into clinical practice. | 68 |
| **Transplant & Immunomodulation** | Research focused on transplantation and the modulation of the immune response, aiming to improve tolerance and clinical outcomes. | 30 |
| **AI, Data, Imaging & Digital Health** | Research in artificial intelligence, big data and digital health to enable precision diagnosis, prediction and patient stratification. | 42 |
| **Environmental & Public Health** | Research on environmental, social and global determinants of health, including climate change and public health challenges. | 20 |
| **Rare Diseases** | Translational research aimed at improving the understanding, diagnosis and management of rare diseases across the life course. | 31 |
| **Ageing** | Research on biological mechanisms of ageing, frailty and associated diseases, with impact on health systems sustainability. | 21 |

## How to proceed?

While there is no hard deadline other than the official call deadline (**9 September 2026, 17:00 CET**), **early expressions of interest are strongly encouraged**.

Researchers who submit their expression of interest early will benefit from earlier support.

### 1. Check eligibility

Please make sure you fulfil the general MSCA-PF eligibility criteria:

- PhD defended by **9 September 2026.**
- Maximum **8 years of research experience** from PhD award (career breaks excluded).
- Compliance with the **MSCA mobility rule**: applicants must not have resided or carried out their main activity in Spain for more than 12 months between **9 September 2023 and 9 September 2026.**

Full eligibility details: MSCA Postdoctoral Fellowships 2026

### 2. Explore the proposed research topics

Review the list of available host laboratories here and identify the topics that best match your profile and research interests.

### 3. How to apply?

You can submit your application via our website to the position corresponding to your project of interest in the following link: apply

## What can you expect?

- If selected, you will be invited for an interview with the host group.
- If both parties agree to proceed, proposal development will begin.
- VHIR will provide support throughout the preparation process, including:
  - Proposal structuring
  - Internal scientific review
  - Administrative review
  - Submission support

As MSCA-PF proposals are highly competitive, applicants will be asked to **submit an advanced draft in early July** in order to benefit from the full internal review process.

## Key dates

**1 August**
Deadline to express your interest

**24 August**
Final administrative review

**8 September**
Internal submission deadline

**9 September 2026 (17:00 CET)**
Official call deadline

*Project funded by Instituto de Salud Carlos III (ISCIII), "FORT23/00034".*

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**PROPOSED TOPICS** – Click on them to read the full description

| REF. | TOPICS | IPs |
|---|---|---|
| VHIR-MSCA-PF-2026.001 | **Targeting cardiac amino acid transport and catabolic reprogramming in heart failure** | Javier Inserte |
| VHIR-MSCA-PF-2026.002 | **Inhibition of mitochondrial sodium/calcium exchanger, NCLX, as a therapeutic strategy to reduce myocardial ischemia-reperfusion injury during myocardial infarction** | Antonio Rodríguez Sinovas |
| VHIR-MSCA-PF-2026.003 | **Precision modelling of FHHNC using patient-derived kidney organoids for mechanistic and therapeutic discovery** | Cristina Martínez<br>Gema Ariceta |
| VHIR-MSCA-PF-2026.004 | **Deciphering the neurotoxic mechanisms of chitinase 3-like 1 (CHI3L1) in progressive multiple sclerosis and identification of therapeutic compounds blocking its pathogenic effects** | Manuel Comabella |
| VHIR-MSCA-PF-2026.005 | **Nucleotide homeostasis and mitochondrial function: physiological and therapeutic implications** | Ramón Martí |
| VHIR-MSCA-PF-2026.006 | **NEURO-immune and GATEway-mediated mechanisms to counteract Parkinson's Disease (NEUROGATE-PD)** | Ariadna Laguna |
| VHIR-MSCA-PF-2026.007 | **Immune and molecular biomarkers in a clinical trial of probiotic therapy in multiple sclerosis: a translational approach** | Carmen Espejo |
| VHIR-MSCA-PF-2026.008 | **Cytotoxic CD8+ T Cells as Early Drivers of Dopaminergic Neuronal Death in Parkinson's Disease** | Jordi Bové |
| VHIR-MSCA-PF-2026.009 | **Development of an *in vitro* diagnostic triage test for patients at risk of endometrial hyperplasia and endometrial cancer based on protein biomarkers in cervical fluids** | Eva Colás |
| VHIR-MSCA-PF-2026.010 | **Beyond Gap Junctions: Connexins as Orchestrators of Tumor–Stroma Communication** | Trond Aasen |
| VHIR-MSCA-PF-2026.011 | **Understanding primary and adaptive tumor immune resistance** | Juan Ángel Recio |

| | | |
|---|---|---|
| VHIR-MSCA-PF-2026.012 | **Exploring the Interplay of Chronic Stress and Antiphospholipid Autoantibodies on Trophoblast Function and the Induction of Preeclampsia** | Francesc Miró Mur |
| VHIR-MSCA-PF-2026.013 | **Inducing CD8+T cell Tissue Resident Phenotypes** | Meritxell Genescà |
| VHIR-MSCA-PF-2026.014 | **Identification and Genetic Engineering of Cytotoxic Tissue NK Cells for Cellular Therapy** | Maria J. Buzón |
| VHIR-MSCA-PF-2026.015 | **Federated and interpretable clinical AI for earlier diagnosis of inborn errors of immunity** | Pere Soler Palacín<br>Jaques Rivière |
| VHIR-MSCA-PF-2026.016 | **Functional validation of genetic variants in pediatric inborn errors of immunity** | Pere Soler Palacín<br>Jaques Rivière |

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Vall d'Hebron Research Institute

Aging

Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.001

# Targeting cardiac amino acid transport and catabolic reprogramming in heart failure

## BACKGROUND

Heart failure (HF) is a leading cause of hospitalization and mortality worldwide, and current therapies only partially slow its progression. The failing heart undergoes maladaptive metabolic remodeling, with reduced substrate flexibility and a striking accumulation of branched-chain amino acids (BCAAs: leucine, isoleucine, valine) in both plasma and myocardium. Elevated BCAAs drive insulin resistance and chronic mTORC1 activation, fueling pathological hypertrophy, fibrosis and contractile dysfunction. Recent work shows that boosting cardiac BCAA catabolism alone is insufficient to normalize myocardial BCAA content, pointing to amino acid transport, rather than intrinsic oxidation, as the key control point. Our preliminary data identify the neutral amino acid transporter Slc7a5 (LAT1) as a critical determinant of cardiac BCAA uptake: it is upregulated in failing cardiomyocytes, and cardiomyocyte-specific deletion reduces myocardial BCAA accumulation and attenuates adverse remodeling in pressure-overload models. This opens an unexplored therapeutic axis at the intersection of cardiac metabolism, insulin signaling and HF.

## OBJECTIVE

The candidate will investigate how alterations in cardiac BCAA transport contributes to insulin resistance, adverse remodeling and HF progression, and will evaluate pharmacological and dietary strategies to correct cardiac BCAA overload. Specific aims include: (i) characterize cardiac amino acid metabolic rewiring in murine HF models and in endomyocardial biopsies and serum from patients with aortic stenosis, combining UPLC-MS/MS metabolomics and RNA-seq; (ii) dissect the role of cardiomyocyte and fibroblast Slc7a5 using inducible tissue-specific knockout mice, with echocardiographic, histological and stable-isotope tracing readouts ([U-13C,15N]-leucine, [U-13C,15N]-glutamine, [U-13C]-glucose); (iii) test pharmacological inhibition of LAT1 and BCAA-modulating diets in TAC mice, including ovariectomized females, to capture sex-specific effects. The project is expected to yield mechanistic insight and to seed a translational program for a new class of metabolic HF therapies.

## THE POSITION

The fellow will join the Cardiovascular Diseases group at Vall d'Hebron Research Institute (VHIR), embedded in the Cardiology Department of Hospital Universitari Vall d'Hebron — one of Spain's largest tertiary cardiology centers and a CIBER-CV reference site. VHIR offers state-of-the-art core facilities and direct access to a structured Biobank. The fellow will work alongside basic researchers and clinicians, with active collaborations with IRB Barcelona (Prof. A. Zorzano, amino acid transporters and insulin resistance), the CIBERDEM Metabolomics Platform (URV) for stable-isotope flux analysis, CIMA (A González, amino acids and diffuse cardiac fibrosis) and the EU-METAHEART COST Action, which provides a networking for cardiovascular metabolism researchers. The position includes a personalized career development plan with training in preclinical experimental models, dynamic metabolomics, transcriptomics, scientific writing and grant preparation, with a strong emphasis on translational relevance and the potential clinical application of the findings.

## THE GROUP

The Cardiovascular Diseases group at VHIR is a multidisciplinary team integrating basic and clinical researchers, with a long-standing track record in translational cardiovascular research, with seminal contributions to ischemia/reperfusion injury, cardioprotection and the metabolic basis of adverse cardiac remodeling and HF. The group integrates a wide range of preclinical HF models in mice, rats and pigs, cardiomyocyte and fibroblast isolation, ex vivo Langendorff perfusion, knockout mouse lines and has privileged access to ventricular biopsies and serum from cardiology patients. A core current line is supported by several competitive grants. The team collaborates with international academic and biotech partners, providing a vibrant, multidisciplinary environment for an MSCA postdoctoral fellow.

## GROUP'S RELATED PUBLICATIONS


- Mericskay M. Cardiac intermediary metabolism in heart failure: substrate use, signalling roles and therapeutic targets. Nat Rev Cardiol. 2025.
- Inserte J. Implications of Iron Deficiency in STEMI Patients and in a Murine Model of Myocardial Infarction. JACC Basic Transl Sci. 2021;6(7):567-580.
- Aluja D.Efficacy of a cysteine protease inhibitor compared with enalapril in murine heart failure models. iScience. 2024


## WHAT DO WE LOOK FOR?

- **Education and qualifications**

**Required:**

- PhD in Biomedicine, Biochemistry, Molecular/Cell Biology, Pharmacology or related discipline. Candidate must comply with MSCA Postdoctoral Fellowship eligibility.

**Desirable:**

- Prior postdoctoral experience in cardiovascular research or metabolism. Official accreditation for work with laboratory animals.

- **Experience and Knowledge**
  **Required:**

  - Solid molecular biology toolkit (qPCR, Western blot, immunofluorescence, cloning).
  - Hands-on experience with in vivo rodent.
  - Strong scientific writing in English and the ability to work independently in a translational, multidisciplinary team.

  **Desirable:**

  - Experience with stable-isotope-resolved metabolomics (13C/15N tracing) and transgenic mouse models.

## CONTACT INFORMATION

**Dr. Javier Inserte:** apply

Aging

Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.002

# Inhibition of mitochondrial sodium/calcium exchanger, NCLX, as a therapeutic strategy to reduce myocardial ischemia-reperfusion injury during myocardial infarction

## BACKGROUND

Myocardial ischemia-reperfusion (IR) injury remains a major challenge in cardiovascular medicine. While rapid reperfusion improves survival after myocardial infarction, it paradoxically triggers additional myocardial damage, promoting infarct expansion and heart failure. Mitochondria play a central role via disruptions in energetics, calcium handling, and excessive reactive oxygen species (ROS) production. Recent studies identify the mitochondrial sodium/calcium exchanger NCLX (SLC8B1) as a key regulator of mitochondrial ROS signaling, linking sodium influx to electron transport chain dysfunction during hypoxia/reoxygenation. Notably, NCLX inhibition reduces ROS and tissue damage in cerebral ischemia models, suggesting a novel role for mitochondrial sodium in ischemic injury, still unexplored in the heart. Our preliminary data show that NCLX inhibition significantly reduces infarct size in cardiac IR models. This project combines cutting-edge mitochondrial biology with translational cardiovascular research to uncover how NCLX controls ROS production, mitochondrial function, and cardiomyocyte survival using advanced in vitro, ex vivo, in vivo, and human models. These findings may redefine reperfusion injury mechanisms and open new therapeutic strategies for ischemic heart disease.

## OBJECTIVE

The goal of this project is to determine whether the mitochondrial sodium/calcium exchanger NCLX is a key regulator of reactive oxygen species (ROS) production and myocardial injury during ischemia-reperfusion (IR), and to assess the therapeutic potential of its inhibition in acute myocardial infarction. The project will examine how NCLX controls mitochondrial sodium and calcium balance, respiratory chain function, and ROS generation during ischemia and reperfusion, as well as the molecular mechanisms linking NCLX activity to mitochondrial dysfunction and cardiomyocyte death. In parallel, the cardioprotective efficacy of novel NCLX inhibitors will be tested in isolated hearts, and in vivo rodent and pig models. By integrating mechanistic and translational approaches, this work aims to establish mitochondrial $Na^+/Ca^{2+}$ exchange as a novel therapeutic target in ischemic heart disease.

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## THE POSITION

The Cardiovascular Diseases Research Group at the Vall d'Hebron Research Institute (VHIR, Barcelona, Spain) has extensive expertise in cardiovascular pathophysiology, with a particular focus on myocardial ischemia-reperfusion injury, adverse ventricular remodeling and cardiac arrhythmias. The group is embedded in a highly collaborative environment within VHIR and maintains active national and international collaborations with leading institutions in cardiovascular research, including IRB Barcelona, IQAC-CSIC, IR Sant Pau, IIS Princesa, IBEC, Universidad Autónoma de Madrid, Lund University and King's College London, as well as participation in networks such as CIBERCV and IMPACT. This highly interconnected environment offers excellent training, networking and career development opportunities for MSCA fellows.

## THE GROUP

The research group focuses on the molecular and translational mechanisms underlying cardiovascular disease, with particular expertise in myocardial ischemia-reperfusion injury, heart failure, adverse ventricular remodeling and cardiac arrhythmias. Its main research lines include the study of mitochondrial and cellular mechanisms of ischemia-reperfusion injury, heart failure progression, inflammatory signaling, cardiovascular aging and metabolic stress, as well as the impact of environmental factors such as air pollution on cardiac function. The group integrates mechanistic research in mitochondrial biology, redox signaling, calcium handling and inflammation with translational approaches using cellular systems, isolated organs, rodent and porcine models, and human cardiac samples.

## GROUP'S RELATED PUBLICATIONS


- **Ganse et al.** *Part Fibre Toxicol* 2025;22(1):36. (2) **Consegal et al.** *Basic Res Cardiol* 2024;119(4):673-689. (3) **Valls-Lacalle et al.** *Antioxidants* 2021;11(1):75.


## WHAT DO WE LOOK FOR?

We are seeking an outstanding and highly motivated postdoctoral researcher to apply for a Marie Sklodowska-Curie Postdoctoral Fellowship in the field of mitochondrial biology, cardiovascular pathophysiology and translational cardioprotection.

- **Education and qualifications**
  **Required:**

- Bachelor's degree in Health Sciences (Biology, Biomedicine, Pharmacy, Biomedicine, Veterinary Medicine or a closely related biomedical discipline).
- Recent PhD degree.
- Good publication track record, and written and oral communication skills in english.

**Desirable:**

- Previous experience in cardiovascular research, mitochondrial biology, ischemia-reperfusion injury, redox signaling or calcium handling.
- Expertise in cellular and molecular biology, live-cell imaging, or animals models.
- Ability to work independently while contributing to a collaborative and interdisciplinary research environment.

- **Experience and Knowledge**
  **Required:**

  - Solid background in cardiovascular physiology, cell biology or related fields.
  - Previous hand-on research experience in molecular and cellular biology techniques, including Western blotting, RT-qPCR, immunofluorescence and biochemical assays.

  **Desirable:**

  - Knowledge of mitochondrial physiology and live-cell imaging techniques.
  - Basic skills in statistical analysis and scientific programming/data analysis.

## CONTACT INFORMATION

**Dr. Antonio Rodríguez Sinovas**: apply

Rare Diseases

Ref: VHIR-MSCA-PF-2026.003

# Precision modelling of FHHNC using patient-derived kidney organoids for mechanistic and therapeutic discovery

## BACKGROUND

Familial hypomagnesemia with hypercalciuria and nephrocalcinosis (FHHNC) is a devastating ultra-rare renal tubulopathy caused by loss-of-function mutations in CLDN16 and CLDN19. The disease leads to severe magnesium and calcium wasting, nephrocalcinosis, and progressive chronic kidney disease, often culminating in renal failure at a young age. Additionally, patients carrying *CLDN19* mutations develop early-onset ocular defects, causing lifelong visual impairment. Despite its severity, no disease-modifying therapies or specific prognostic biomarkers are currently available. A striking feature of FHHNC is its marked phenotypic variability, even among siblings carrying identical mutations, particularly in Southern European patients harbouring the prevalent *CLDN19* founder mutation p.G20D. This suggests the involvement of additional molecular mechanisms, including modifier genes and dysregulated pathways, which remain poorly understood.

Our group has established one of the largest and best-characterized FHHNC cohorts worldwide and pioneered an interdisciplinary strategy combining clinical research, molecular profiling, and artificial intelligence to uncover mechanisms and therapeutic opportunities in FHHNC. Using patient-derived molecular data integrated with AI-based network medicine approaches, we identified novel phenotype modifier genes, disease-associated urinary exosomal miRNA signatures, and multiple candidate drugs and synergistic drug combinations with therapeutic potential. Building on these findings, we recently generated induced pluripotent stem cells (iPSC) from two siblings carrying the homozygous *CLDN19* p.G20D mutation but displaying markedly divergent renal and ocular phenotypes. Together with our unique *CLDN19* p.G20D knock-in mouse model, these resources provide an unprecedented platform to investigate FHHNC pathophysiology. In this project, patient-derived genetically engineered kidney organoids will be used as advanced human 3D models to dissect disease mechanisms, understand phenotypic variability, and evaluate therapeutic candidates in a precision medicine framework. By integrating stem cell technology, genome engineering, and translational nephrology, this project aims to deliver transformative insights and accelerate therapeutic innovation for this unmet rare disease.

## OBJECTIVE

The main goal of this project is to develop and exploit genetically engineered patient-derived kidney organoids to unravel the molecular mechanisms driving FHHNC, with a particular focus on phenotypic variability and sex-specific disease mechanisms, and to accelerate the identification of novel precision medicine strategies through drug repurposing and advanced human disease modelling.

## THE POSITION

We are seeking a highly motivated postdoctoral researcher to join the Kidney Pathophysiology Research Group at VHIR, Barcelona, and develop a competitive application for the MSCA Postdoctoral Fellowship. The selected candidate will take a leading role in the preparation of an MSCA proposal focused on translational research in rare renal diseases using advanced *in vitro*, *ex vivo*, and *in vivo* models. The project will combine stem cell technology, functional genomics, disease modelling, and therapeutic evaluation in the context of inherited renal tubulopathies and epithelial dysfunction. The candidate will work within a multidisciplinary and highly translational environment integrated into the Paediatric Nephrology Service of the Vall d'Hebron University Hospital. The position offers close interaction between clinicians and basic researchers, providing a unique framework to translate experimental discoveries into clinically relevant applications.

The successful applicant will have access to state-of-the-art technologies and unique research resources, including patient-derived induced pluripotent stem cells (iPSC), kidney organoids, genetically engineered cellular systems, and innovative mouse models. The fellowship will provide excellent opportunities for scientific training, international networking, career development, and the establishment of an independent research profile in the field of rare kidney diseases and precision medicine.

## THE GROUP

The Kidney Pathophysiology Research Group at VHIR focuses on understanding the molecular and cellular mechanisms underlying rare and chronic kidney diseases, with a special emphasis on inherited tubulopathies, epithelial dysfunction, fibrosis, and the progression of chronic kidney disease. The group develops highly translational research programs integrating clinical data, experimental biology, and computational approaches to identify disease mechanisms and novel therapeutic opportunities. One of the group's main research lines focuses on Familial Hypomagnesemia with Hypercalciuria and Nephrocalcinosis (FHHNC), an ultra-rare inherited renal disease caused by mutations in *CLDN16* and *CLDN19*. Over the last decade, the group has established one of the largest and best-characterized FHHNC patient cohorts worldwide and pioneered interdisciplinary approaches combining molecular biology, artificial intelligence, and

translational nephrology to investigate disease progression and therapeutic strategies. The group has generated unique experimental platforms, including patient-derived iPSC lines and the first *CLDN19* p.G20D knock-in mouse model available for FHHNC research. In parallel, strong collaborations with clinicians, computational scientists, and patient advocacy organizations ensure a highly collaborative, multidisciplinary, and patient-oriented research environment with direct translational impact.

## GROUP'S RELATED PUBLICATIONS


- Vall-Palomar M, Burballa C, Claverie-Martín F, Meseguer A, Ariceta G. Heterogeneity is a common ground in familial hypomagnesemia with hypercalciuria and nephrocalcinosis caused by *CLDN19* gene mutations. J Nephrol. 2021 Dec;34(6):2053-2062. doi: 10.1007/s40620-021-01054-6.
- Vall-Palomar M, Arévalo J, Ariceta G, Meseguer A. Establishment of urinary exosome-like vesicles isolation protocol for FHHNC patients and evaluation of different exosomal RNA extraction methods. J Transl Med. 2018 Oct 11;16(1):278. doi: 10.1186/s12967-018-1651-z.
- Vall-Palomar M, Torchia J, Morata J, Durán M, Tonda R, Ferrer M, Sánchez A, Cantero-Recasens G, Ariceta G, Meseguer A, Martinez C. Identification of modifier gene variants overrepresented in familial hypomagnesemia with hypercalciuria and nephrocalcinosis patients with a more aggressive renal phenotype. PLoS Genet. 2025 Apr 2;21(4):e1011568. doi: 10.1371/journal.pgen.1011568.


## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**
  - PhD in Biomedical Sciences or related discipline.
  - Formal training in animal experimentation (EU Directive 2010/63/EU or equivalent certification).
  - Have a maximum of 8 years of full-time equivalent research experience after the award of their PhD (extensions apply for eligible career breaks such as parental leave, illness, or non-research periods, in accordance with MSCA rules).
  - Comply with the MSCA mobility rule: candidates must not have resided or conducted their main activity (work, studies, or research) in Spain for more than 12 months in the 36 months immediately before the call deadline.
  - Fluency in English (business level).

**Desirable:**

- Specific training in organoid technology and *in vivo* disease modelling.
- Training in data analysis and/or bioinformatics would be highly desirable, particularly experience with omics data, systems biology approaches, or computational analysis of biological datasets.
- Strong scientific background suitable for the preparation of a Marie Skłodowska-Curie Postdoctoral Fellowship application.

- **Experience and Knowledge**
  **Required:**

  - Experience with advanced experimental techniques (e.g. imaging, molecular biology, omics analysis, or functional assays).
  - Knowledge of epithelial cell biology and/or experimental models of disease.
  - Hands-on experience with animal models of disease.
  - Ability to design, perform, and analyse experimental research independently.
  - Previous exposure to translational research environments or clinical collaboration frameworks.
  - Ability to work in multidisciplinary and collaborative research environments.

  **Desirable:**

  - Experience with organoid systems and/or human induced pluripotent stem cells (hiPSC).
  - Knowledge of kidney physiology, tubular epithelial biology, or fibrosis-related mechanisms.
  - Experience with organoids and/or hiPSC-derived systems.
  - Familiarity with translational research or drug evaluation pipelines.
  - Ability to integrate multi-level experimental data (cellular, molecular, and *in vivo*).

## CONTACT INFORMATION

**Dra. Cristina Martínez** and **Dra. Gema Ariceta:** apply

Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.004

# Deciphering the neurotoxic mechanisms of chitinase 3-like 1 (CHI3L1) in progressive multiple sclerosis and identification of therapeutic compounds blocking its pathogenic effects

## BACKGROUND

Progressive forms of multiple sclerosis (MS) are characterized by chronic neurodegeneration leading to irreversible neurological disability. Current therapeutic options for progressive MS remain limited and only partially effective, highlighting the urgent need for neuroprotective strategies targeting mechanisms of neuronal damage.

Our group identified chitinase 3-like 1 (CHI3L1) as one of the most important prognostic biomarkers associated with disease progression and long-term disability in MS patients. Additional studies performed by our group demonstrated that CHI3L1 exerts direct neurotoxic effects in both murine neurons and human neurons differentiated from induced pluripotent stem cells (iPSCs) derived from MS patients. These findings suggest that CHI3L1 is not only a biomarker of disease progression but also a potential therapeutic target.

The project aims to investigate the molecular mechanisms underlying CHI3L1-mediated neurotoxicity in human neuronal models and to identify compounds capable of neutralizing its pathogenic effects. The work will combine stem cell-derived neuronal cultures, molecular and cellular biology, advanced imaging, transcriptomic and proteomic approaches, and drug screening strategies.

This research has strong translational potential, as the identification of compounds blocking CHI3L1 activity may open the door to future clinical trials in patients with progressive MS.

## OBJECTIVE

Main objectives of the project:

1. To identify intracellular mediators and signaling pathways involved in CHI3L1-induced neuronal toxicity.
2. To identify neuronal receptors mediating the pathogenic effects of CHI3L1.

3. To generate and characterize human iPSC-derived neuronal models.
4. To identify approved drugs and/or small molecules capable of blocking CHI3L1 activity or preventing its binding to neuronal receptors.
5. To validate candidate compounds in human neuronal cultures as potential therapeutic strategies for progressive MS.

## THE POSITION

The project will be developed at the Vall d'Hebron Research Institute (VHIR) and the Centre d'Esclerosi Mútiple de Catalunya (Cemcat), within a highly collaborative and multidisciplinary environment integrating clinical neurology, stem cell biology, neuroimmunology, bioinformatics, and translational neuroscience.
The project includes collaborations with experts in stem cell biology and neuronal differentiation, proteomics and mass spectrometry, bioinformatics and systems biology, drug discovery and virtual screening, as well as neuroimmunology and progressive MS.

The research environment provides access to state-of-the-art facilities including advanced microscopy and high-throughput molecular technologies.

## THE GROUP

The Clinical Neuroimmunology group at VHIR is dedicated to translational clinical-basic research in MS, with a particular focus on neurodegeneration, biomarkers, neuroinflammation, and the development of innovative therapeutic strategies for progressive forms of the disease. The group has strong expertise in:
- Human iPSC generation and differentiation.
- Neuronal and glial cell cultures.
- Advanced molecular and imaging techniques.
- Biomarker discovery.
- Translational multiple sclerosis research.

## GROUP'S RELATED PUBLICATIONS


- Cantó E et al. Chitinase 3-like 1: prognostic biomarker in clinically isolated syndromes. Brain 2015; 138:918-931.
- Matute-Blanch C et al. Chitinase 3-like 1 is neurotoxic in primary cultured neurons. Sci Rep. 2020 Apr 28;10(1):7118.
- Pinteac R et al. Chitinase 3-like 1 is neurotoxic in multiple sclerosis patient-derived cortical neurons. Clin Transl Med. 2024 Dec;14(12):e70125.

## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - PhD in neuroscience, neuroimmunology, stem cell biology, molecular biology, biomedical sciences, or related disciplines.

  **Desirable:**

  - Previous experience in neurodegeneration and/or MS research. Experience in translational neuroscience research.

- **Experience and Knowledge**
  **Required:**

  - Strong hands-on experience in human stem cell culture, particularly iPSCs.
  - Experience differentiating iPSCs into neuronal lineages.
  - Experience in neuronal cell culture and cellular functional assays.
  - Experience in molecular and cellular biology techniques, including immunocytochemistry, qPCR, and western blot.
  - Experience in microscopy and image analysis.
  - Experience with transcriptomics, proteomics, bioinformatics, or single-cell approaches.
  - Ability to work independently and within multidisciplinary collaborative teams.

  **Desirable:**

  - Experience with drug screening or small molecule validation.
  - Scientific productivity demonstrated through peer-reviewed publications.

## CONTACT INFORMATION

**Dr. Manuel Comabella:** apply

Rare Diseases

Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.005

# Nucleotide homeostasis and mitochondrial function: physiological and therapeutic implications

## BACKGROUND

Mitochondrial DNA (mtDNA) depletion/deletions syndromes (MDDS) are severe conditions caused by mutations in nuclear genes involved in mtDNA replication. dNTP imbalancess are often associated to these disorders, either because the primary gene is involved in dNTP anabolic or catabolic pathways, or because the mtDNA aberrations secondarily alter dNTP metabolism.

**The dNTP homeostasis is a therapeutic target for some MDDS**. For instance, mitochondrial neurogastrointestinal encephalomyopathy (MNGIE) is caused by the toxic accumulation of thymidine and deoxyuridine, and the treatments of this disorder aim to clear the overload of these metabolites. Also, **the recent approval of thymidine and deoxycytidine to treat TK2 deficiency (Kigevvy™) constitutes the first therapy that changes the natural history of an MDDS form,** extending the life expectancy of patients that otherwise would die in months or few years.

Preclinical studies support the notion that **stimulating mtDNA replication by enhancing dNTP synthesis may be an effective treatment of other MDDS forms**. Moreover, our recent demonstration of **dGTP binding the OXPHOS complex I constitutes a novel finding with potential unexplored functional consequences**. Therefore, the mitochondrial dNTP metabolism and how it is linked to the OXPHOS function and the mitochondrial disorders is currently a hot topic. Improving our knowledge in this field may result in therapies for devastating disorders with no effective treatment so far.

## OBJECTIVE

The general objective of this action is **to investigate so far unexplored aspects of the dNTP metabolism in the context of the mitochondrial (dys)function, with especial focus on mtDNA replication and maintenance**, including the investigation and further development of potential therapies for mtDNA maintenance disorders.

Examples of some specific objectives:

- To identify genetic/protein/metabolic factors determining mitochondrial dNTP homeostasis and their relation with normal and dysfunctional mtDNA replication.
- To study dNTP homeostasis in several *in vitro* and *in vivo* models of mtDNA replication defects, and to design/test therapy strategies to modulate dNTP imbalances.

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vhir.vallhebron.com/ca

- To explore novel potential dNTP-related therapy targets, such as nucleoside/nucleotide transport or enzyme activation/inhibition.
- To contribute to preclinical/clinical development of ongoing therapies, such as AAV-based gene therapy for MNGIE and other dNTP-related therapies in their track to the regulatory approval.

Beyond these examples, the details of the project will be finally re-designed according to the expertise of the candidate and will be jointly re-shaped once he/she is integrated in the research group.

## THE POSITION

The candidate will be integrated in the VHIR, a research institute associated to a University Hospital in Barcelona, in the **research group of neuromuscular and mitochondrial disorders**. The investigator will work with cellular and mouse models (available mouse strains genetically modified in the following genes: *Polg, Tk2, Dguok, Opa1, Tymp/Upp1, Ndufa10, Pygm*). The methodology will comprise, among other techniques, general molecular biology methods, real-time qPCR, digital PCR, enzymology-linked methods, dNTP assessment, targeted metabolomics (HPLC-masss spectrometry). In addition, the VHIR core facilities offer well-equipped platforms: genomics, molecular diagnosis, cytometry, microscopy.

## THE GROUP

The **research group of neuromuscular and mitochondrial disorders** focuses its research activity on studying the pathophysiological mechanisms of mitochondrial diseases, especially those associated with defects in mtDNA maintenance. In addition, we have some research lines dedicated to the study of another group of metabolic disorders, the muscle glycogenoses. Some of our current research lines are:

- Study of the pathomechanisms of MDDS and development of therapeutic strategies for them.
- Pathophysiology of mitochondrial diseases due to defects in mitochondrial translation.
- AAV-based gene therapy (for MNGIE disease and for mutations in GFM1).
- Pathomechanisms of the McArdle's disease using a murine model of this disorder.
- Coordination of EUROMAC (European registry of patients with McArdle's disease and other glycogenosis).

## GROUP'S RELATED PUBLICATIONS

- Molina-Granada et al, **Most mitochondrial dGTP is tightly bound to respiratory complex I through the NDUFA10 subunit**. Commun Biol 2022;5:620.
- Vila-Julià et al, **Efficacy of adeno-associated virus gene therapy in a MNGIE murine model enhanced by chronic exposure to nucleosides**. EBioMedicine 2020;62:103133.
- Blázquez-Bermejo et al, **Age-related metabolic changes limit efficacy of deoxynucleoside-based therapy in thymidine kinase 2-deficient mice**. EBioMedicine 2019;46:342-355.
- Blázquez-Bermejo et al, **Increased dNTP pools rescue mtDNA depletion in human POLG-deficient fibroblasts**. FASEB J 2019;33:7168-79.

## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**
  - PhD in any area related to biology, biomedicine, biochemistry, chemistry, biotechnology, or similar.
  - At least basic communication skills in English.

  **Desirable:**
  - Official certificate to conduct experimental tasks with mice.

- **Experience and Knowledge**
  **Required:**
  - General laboratory biochemical and molecular biology methods.

  **Desirable:**
  - Any additional skills in the field of biomedical research methodology will be welcome.

## CONTACT INFORMATION

**Dr. Ramon Martí:** apply

Vall d'Hebron Research Institute

Aging | Environmental & Public Health | Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.006

# NEURO-immune and GATEway-mediated mechanisms to counteract Parkinson's Disease (NEUROGATE-PD)

## BACKGROUND

Parkinson's disease (PD) is the fastest-growing neurodegenerative disorder, affecting over 10 million people worldwide. Current treatments remain symptomatic and do not prevent disease progression. Although classically defined by dopaminergic neuron loss and α-synuclein aggregation, PD is now recognised as a multi-system disorder.
Non-motor symptoms such as gastrointestinal dysfunction, mood alterations, and sleep disturbances often precede motor symptoms, highlighting the gut–brain axis as a bidirectional communication network relevant for PD pathogenesis. Within this axis, microbiota composition, barrier integrity, and immune signalling contribute to neuroinflammation and neurodegeneration. Aging impairs both barrier function and immune homeostasis. In PD, these alterations are exacerbated, increasing permeability, immune cell infiltration, chronic inflammation, and neurodegeneration. However, the relative contribution of aging versus environmental factors (e.g., infections, pollutants), and the therapeutic opportunities derived from restoring these functions, remain unclear.
To address these gaps, this project will integrate clinical research, experimental models, and mechanistic studies in a multidisciplinary and international framework, aiming to uncover mechanisms linking barrier dysfunction to neurodegeneration and to develop regenerative strategies targeting these interfaces.
This approach will advance our understanding of brain–body communication, identify biomarkers, and open new therapeutic avenues for PD and other age-related diseases.

## OBJECTIVE

NEUROGATE-PD aims to understand the principles of barrier-mediated brain–body interactions in Parkinson's disease (PD). This project will address key knowledge gaps essential for future biomarker discovery and therapeutic development aimed at preventing neurodegeneration through restoration of barrier function. In addition, it will investigate the impact of lifestyle factors (e.g., nutrition, microbiota, and environmental pollutants) on barrier integrity and how these factors influence PD onset and progression using relevant clinical cohorts and humanized experimental models.

## THE POSITION

The candidate will investigate inter-organ communication mechanisms in PD to identify novel pathogenic pathways, biomarkers, and disease-modifying strategies.
PD remains incurable, lacking early diagnostic biomarkers and effective disease-modifying treatments. Diagnosis is currently based on motor symptoms appearing after significant neuronal loss, whereas non-motor symptoms and microbiota alterations arise earlier.The project focuses on how gut–brain, neuroimmune, neuroendocrine, and metabolic pathways contribute to disease onset and progression.
The role includes:

- Analysis of human samples and clinical data
- Mechanistic studies in rodent and cellular models

The candidate will join a multidisciplinary environment within the Neurodegenerative Diseases Research Group at VHIR, involving expertise in neurology, biomedicine, biotechnology, chemistry, and bioinformatics.
VHIR is a leading biomedical research institute integrated within Vall d'Hebron University Hospital, enabling strong translational (bench-to-bedside) research. It offers advanced training opportunities in both technical (omics, flow cytometry, statistics) and transversal skills.
The candidate will participate in seminars, international collaborations, conferences, and short research stays.

## THE GROUP

The PI is a junior group leader within the Neurodegenerative Diseases Research Group led by Dr. Miquel Vila (ICREA Professor, UAB). The group includes experts such as Dr. Marta Martínez-Vicente (autophagy research) and Dr. Jorge Hernández-Vara (clinical neurology). The team has established the Vall d'Hebron Parkinson's Initiative (VHIP), a key longitudinal cohort for this project. The group collaborates extensively with national and international experts and networks (CIBERNED, ASAP, GBA consortium). Their research addresses PD as a multifactorial disorder with significant societal impact, combining mechanistic research, clinical cohorts, and translational innovation.
**Main research lines:**

- **Clinical and Biomarker Research**

VHIP includes >200 participants (patients, carriers, controls), combining clinical, genetic, imaging, and biospecimen data. Objectives:

  - Biomarker discovery (risk, diagnosis, prognosis)
  - Disease stratification
  - Early pathophysiology understanding gender perspective is systematically integrated.

- **Translational Innovation and Therapeutics**
  - Focus on gut–brain communication and neuroimmune modulation
  - Studying enteric α-synuclein pathogenicity

- Dietary interventions
- Targeting bacterial neurotoxins

- **Social Innovation**
  - Development of participatory initiatives involving patients and stakeholders to improve quality of life and promote societal impact.

## GROUP'S RELATED PUBLICATIONS

- "Age-dependent brain pigmentation drives early neuroinflammatory molecular signatures linked to neurodegeneration". Núria Peñuelas *et al*.. **Journal of Neuroinflammation**, *under second revision*.
- "The Human Microglia Atlas (HuMicA) Unravels Changes in Homeostatic and Disease-Associated Microglia Subsets across Neurodegenerative Conditions". Ricardo Martins-Ferreira *et al*. **Nature Communications, 2025,** *16, 739. https://doi.org/10.1038/s41467-025-56124-1*
- "Modelling human brain-wide pigmentation in rodents recapitulates age-related multisystem neurodegenerative deficits". Ariadna Laguna* *et al.* **Nature Communications, 2024**; 15 (1), 8819. *(IF: 14,7; D1)* https://doi.org/10.1038/s41467-024-53168-7
- *"*Gene therapy with VMAT2 prevents Parkinson's disease phenotype in humanized neuromelanin-producing rats*".* M. Gonzalez-Sepulveda *et al.* **Brain,** 2023*, Mar 1;146(3):1040-1052. doi: 10.1093/brain/awac445*

## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - Bachelor's Degree in Health/Life Sciences.
  - Completed PhD in Neurosciences, Translational Biomedicine Research, Immunology, Microbiology or similar.
  - Good communication skills and fluency in spoken and written English.
  - Experience and knowledge of basic laboratory histological techniques (e.g. stereological cell analysis, IHC, IF, confocal microscopy) and molecular techniques (ELISA, RTqPCR, WB, Dot Blot, Flow Cytometry).
  - Certification and experience working with animal models and knowledge of animal experimentation techniques (stereotaxic injection of AdenoAssociatedViral Vectors (AAV), behavioral tests, oral gavage, ip/iv/sc administration).

  **Desirable:**

- Previous knowledge in the field of neurodegenerative diseases, specifically in Parkinson's Disease.
- Previous experience processing human and animal samples (brain tissue and biofluids).
- Experience in software for image and data analysis (OlyVIA, Image J, GraphPad, Stata, R).

- **Experience and Knowledge Required:**

  - At least 3 years of experience working with preclinical rodent models.
  - Proactive, dynamic and outstanding organizational skills.
  - Strong sense of responsibility, initiative, self-motivation and social skills as key personal abilities.
  - Ability to work independently as well as in a team environment.

  **Desirable:**

  - Previous experience with Neurosciences, Immunology or Microbiology.
  - Main author of scientific publications in indexed journals.
  - Mobility and research experience abroad.

## CONTACT INFORMATION

**Dra. Ariadna Laguna**: apply

Innovative & Advanced Therapies | Transplant & Immunomodulation

Ref: VHIR-MSCA-PF-2026.007

# Immune and molecular biomarkers in a clinical trial of probiotic therapy in multiple sclerosis: a translational approach

## BACKGROUND

Multiple sclerosis (MS) is a chronic inflammatory and neurodegenerative disease of the central nervous system, characterized by demyelination and immune dysregulation. Emerging evidence highlights the role of gut microbiota as a key environmental factor influencing MS pathogenesis, through its interaction with the immune system. Dysbiosis has been consistently described in MS patients and is associated with pro-inflammatory immune responses.

Preclinical studies, including those performed by our group, demonstrate that probiotic administration can modulate immune responses, reduce neuroinflammation, and improve disease severity in experimental models.

However, the mechanisms underlying these effects in humans remain insufficiently understood, particularly at the level of **immunological and molecular biomarkers**, which are critical for translating microbiota-based interventions into clinical practice.

## OBJECTIVE

The overall aim of this project is to characterize the immunological and molecular mechanisms underlying the therapeutic effects of probiotic administration in patients with MS.

Specific objectives include:

- To assess the impact of probiotic treatment on immune cell subsets.
- To quantify changes in serum biomarkers associated with neurodegeneration and inflammation.
- To evaluate metabolic biomarkers (short-chain fatty acids) linked to microbiota activity.
- To characterize changes in gut microbiota composition and explore its association with immune and molecular responses to treatment.
- To integrate these data with clinical and imaging outcomes to identify biomarkers of treatment response.

## THE POSITION

The postdoctoral researcher will lead the laboratory-based biomarker characterization within an ongoing randomized, double-blind, placebo-controlled clinical trial in MS.
The fellow will be responsible for:

- Immunophenotyping using multi-parameter flow cytometry.
- Quantification of molecular biomarkers (SIMOA, ELLA).
- Integration and interpretation of biomarker data.

Microbiota analysis will be performed through external collaboration, allowing the fellow to focus on high-impact translational immunology and biomarker discovery.
The project is embedded in a multidisciplinary environment combining clinical neurology, immunology, and advanced biomarker platforms at VHIR.

## THE GROUP

The researcher will join the Clinical Neuroimmunology Group at VHIR/Cemcat, led by Carmen Espejo Ruiz, an experienced investigator in MS pathogenesis, immunology, and biomarker discovery.
The group has strong expertise in:

- Experimental model of MS (experimental autoimmune encephalomyelitis [EAE]).
- Translational immunology and biomarker identification.
- Clinical trials in MS, with access to large patient cohorts and biobanked samples.

The project benefits from a highly collaborative ecosystem including:

- Clinical Trials Unit at Cemcat.
- VHIR technological platforms (flow cytometry, SIMOA, statistics unit, biobank).
- External collaborations for microbiota sequencing and metabolomics.

## GROUP'S RELATED PUBLICATIONS

Full publication list available at: https://orcid.org/0000-0001-9949-5901


- Calvo-Barreiro et al. Combined therapies to treat complex diseases: The role of the gut microbiota in multiple sclerosis. doi: 10.1016/j.autrev.2017.11.019.
- Calvo-Barreiro et al. A Commercial Probiotic Induces Tolerogenic and Reduces Pathogenic Responses in Experimental Autoimmune Encephalomyelitis. doi: 10.3390/cells9040906.


- Calvo-Barreiro et al. Selected Clostridia Strains from The Human Microbiota and their Metabolite, Butyrate, Improve Experimental Autoimmune Encephalomyelitis. doi: 10.1007/s13311-021-01016-7.


## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - PhD in Immunology, Neuroscience, Biomedical Sciences, or related field.
  - Strong background in immunology and translational research.
  - Excellent command of English (spoken and written).

  **Desirable:**

  - Experience in neuroimmunology or multiple sclerosis research.
  - Training in biomarker development.

- **Experience and Knowledge**
  **Required:**

  - Hands-on experience in flow cytometry and immune profiling.
  - Experience in handling human biological samples (PBMCs, serum).
  - Data analysis and interpretation skills.

  **Desirable:**

  - Experience with SIMOA/ELLA or similar biomarker platforms.
  - Experience integrating multi-modal datasets (clinical + molecular).

## CONTACT INFORMATION

**Dra. Carmen Espejo Ruiz**: apply

Vall d'Hebron Research Institute

Aging | AI, Data, Imaging & Digital Health | Innovative & Advanced Therapies | Transplant & Immunomodulation

Ref: VHIR-MSCA-PF-2026.008

# Cytotoxic CD8+ T Cells as Early Drivers of Dopaminergic Neuronal Death in Parkinson's Disease

## BACKGROUND

Parkinson's disease (PD) is characterized by the selective degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc), yet the events that initiate their death remain unresolved. The host laboratory has generated postmortem human evidence that places cytotoxic CD8+ T cells at the very beginning of this process. In a systematic analysis of SNpc tissue spanning the full neuropathological spectrum of PD, CD8+ T cells were found to infiltrate the substantia nigra before α-synuclein aggregation or neuronal loss is detectable. At the earliest presymptomatic Braak stages, when α-synuclein pathology is still confined to the olfactory bulb and brainstem, the SNpc already shows robust infiltration of CD8+ T cells—including granzyme B-expressing tissue-resident memory subsets—many of which make direct physical contacts with tyrosine hydroxylase-positive dopaminergic neurons. CD4+ T cells do not show a comparable pattern of nigral infiltration or neuronal contact. These findings, published in Brain (Galiano-Landeira et al., 2020), establish that a cytotoxic T cell response against dopaminergic neurons is one of the earliest detectable events in PD, raising the hypothesis that it may trigger, rather than merely accompany, the subsequent cascade of protein aggregation and neuronal death. What renders SNpc dopaminergic neurons specifically vulnerable to this CD8+ attack remains an open and fundamental question. These neurons operate under conditions of chronic and exceptionally high oxidative stress, arising from dopamine metabolism, mitochondrial complex I insufficiency, and sustained catecholamine auto-oxidation. This intrinsic oxidative burden generates modified intracellular proteins and potential neo-antigens that may be processed and presented via MHC class I, making stressed dopaminergic neurons immunologically visible to antigen-specific CD8+ T cells. Neuromelanin—the dark intraneuronal pigment that accumulates progressively with age as a byproduct of catecholamine oxidation, uniquely abundant in human SNpc neurons—is a histological indicator of this sustained oxidative state. The neurons that accumulate the most neuromelanin are precisely those that degenerate earliest and most severely in PD, and postmortem data from the host laboratory show that CD8+ T cells preferentially contact neuromelanin-laden neurons, pointing to oxidative stress-associated antigenicity as a potential determinant of selective vulnerability. To interrogate this hypothesis in a tractable experimental system, the host laboratory has developed unique rodent models in which dopaminergic neurons accumulate intraneuronal neuromelanin and replicate the oxidative microenvironment of human SNpc neurons. Preliminary data in these animals show that CD8+ T cells infiltrate

the SNpc and establish contacts with neuromelanin-accumulating neurons, closely mirroring the human postmortem
findings, and providing the first experimentally tractable platform to dissect the causal role of CD8+ T cells in dopaminergic neurodegeneration in vivo.

## OBJECTIVE

The overarching goal of this project is to establish whether cytotoxic CD8+ T cells initiate, amplify, or are required effectors of dopaminergic neuronal dysfunction and death in PD. The project builds directly on the host laboratory's postmortem human findings and uses unique rodent models that replicate the oxidative microenvironment of human SNpc neurons to interrogate CD8+ T cell-mediated pathology in vivo. Objective 1: Establish the causal and temporal relationship between CD8+ T cell infiltration and the earliest events of dopaminergic neuronal stress. High-resolution immunofluorescence will quantify markers of neuronal stress (tyrosine hydroxylase downregulation, mitochondrial and lysosomal dysfunction, protein aggregation), microglial activation (Iba1, CD68), and the extent, activation state, and spatial distribution of infiltrating CD8+ T cells. Spatial transcriptomics will be applied at the earliest time point at which CD8+ infiltration is detectable, enabling in situ reconstruction of the neuron–immune transcriptional interactome at the onset of the cytotoxic response. Objective 2: Determine whether CD8+ T cells are necessary effectors of dopaminergic neurodegeneration through antibody-mediated depletion at two critical stages: (a) the earliest time point showing tyrosine hydroxylase downregulation without overt neuronal loss, to test whether CD8+ T cells drive the transition from dysfunction to death; and (b) at the stage of maximal infiltration and neurodegeneration, to assess whether their removal can halt ongoing injury. Outcomes will include dopaminergic neuron counts, neuronal stress markers, and microglial activation state. Objective 3: Characterise the phenotype, clonal architecture, and antigen specificity of SNpc-infiltrating and CSF-derived CD8+ T cells. Full-length single-cell immunogenomics (scRNA-seq + scTCR-seq) will be performed on CD8+ T cells isolated from the SNpc and CSF at the most informative time points. This will define cytotoxic, exhausted, and tissue-resident memory states; reveal clonally expanded TCR repertoires indicative of antigen-driven selection; and generate convergent CDR3 motifs as candidates for computational antigen prediction. Findings will be interpreted in light of the human postmortem TCR data generated in parallel by the host laboratory.

## THE POSITION

The successful candidate will join the Neurodegenerative Diseases Research Group at the Vall d'Hebron Institut de Recerca (VHIR), hosted within the Vall d'Hebron University Hospital campus in Barcelona, under the direct supervision of Dr. Jordi Bové (Principal Investigator). The group is led by Dr. Miquel Vila and focuses on understanding the earliest mechanisms of dopaminergic neuronal

death in PD. The fellow will have full access to the group's and their collaborators' unique experimental platforms, including neuromelanin-accumulating rodent models, human postmortem SNpc tissue spanning the full neuropathological spectrum of PD, multiplex immunofluorescence, spatial transcriptomics, scRNA-seq, scTCR-seq, and metatranscriptomics. The position offers advanced training in experimental neuroimmunology, translational PD research, and single-cell genomics within a highly collaborative international environment. VHIR holds the HR Excellence in Research award and is fully committed to the principles of the European Charter for Researchers.

## THE GROUP

The Neurodegenerative Diseases Research Group at VHIR is led by Dr. Miquel Vila (ICREA Professor) and is part of CIBERNED (Centro de Investigación Biomédica en Red de Enfermedades Neurodegenerativas) and the Aligning Science Across Parkinson's Collaborative Research Network (ASAP-CRN), providing access to a broad national and international collaborative framework in basic and clinical PD research. The host institution is the Vall d'Hebron Institut de Recerca (VHIR), the research arm of Vall d'Hebron University Hospital — the largest hospital of the Catalan Institute of Health and the main hospital of Barcelona — and a recognized leader in clinical and translational research at the European level, part of the Vall d'Hebron Barcelona Hospital Campus, a global-leading health park where research, clinical care, teaching, and innovation go hand in hand. VHIR is a CERCA center, accredited by the Instituto de Salud Carlos III (ISCIII), and affiliated to the Autonomous University of Barcelona (UAB). Core research facilities available to fellows include a High Technology Unit, Statistics and Bioinformatics Unit, Lab Animal Service, Biobank, and Clinical Research Support Unit, and VHIR is a member of EATRIS and ECRIN, providing access to pan-European research infrastructures. The group's methodological portfolio spans in vivo rodent neurobiology, human postmortem and patient tissue analysis, multiplex immunofluorescence, spatial transcriptomics, bulk and single-cell RNA sequencing, single-cell TCR sequencing, and metatranscriptomics, with active international collaborations in neuropathology, neuroimmunology, and computational biology.

## GROUP'S RELATED PUBLICATIONS


- Galiano-Landeira J, Torra A, Vila M, Bové J. CD8 T cell nigral infiltrationprecedes synucleinopathy in early stages of Parkinson's disease. Brain.2020;143(12):3717–3733. DOI: 10.1093/brain/awaa269
- de Fàbregues O, Sellés M, Ramos-Vicente D, Roch G, Vila M, Bové J.Relevance of tissue-resident memory CD8 T cells in the onset of Parkinson's
- disease and examination of its possible etiologies: infectious or autoimmune? Neurobiol Dis. 2023;187:106308. DOI: 10.1016/j.nbd.2023.106308
- Carballo-Carbajal I, Laguna A, Romero-Giménez J, Cuadros T, Bové J,Martinez-Vicente M, Parent A, Gonzalez-Sepulveda M, Peñuelas N, Torra A,Rodríguez-Galván

B, Ballabio A, Hasegawa T, Bortolozzi A, Gelpi E, Vila M. Braintyrosinase overexpression implicates age-dependent neuromelanin production inParkinson's disease pathogenesis. Nat Commun. 2019;10(1):973. DOI:10.1038/s41467-019-08858-y

## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - PhD in neuroscience, immunology, cell biology, or a closely relateddiscipline

  **Desirable:**

  - Formal training in bioinformatics or computational biology, evidenced by apostgraduate degree, specialization diploma, or completion of a recognized course program.

- **Experience and Knowledge**
  **Required:**

  - Demonstrable hands-on experience in standard neuroimmunology techniques, including immunohistochemistry or immunofluorescence on brain tissue, flow cytometry, or immune cell isolation and culture from CNS or peripheral compartments.
  - Proficiency in English (written and spoken).
  - Strong analytical and problem-solving skills with capacity for independent scientific thinking.

  **Desirable:**

  - Experience with spatial transcriptomics or protein multiplexing platforms.
  - Familiarity with bioinformatics pipelines for single-cell and bulk data analysis.
  - Experience with animal models.

## CONTACT INFORMATION

**Dr. Jordi Bové:** apply

Vall d'Hebron Research Institute

Aging | AI, Data, Imaging & Digital Health | Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.009

# Development of an *in vitro* diagnostic triage test for patients at risk of endometrial hyperplasia and endometrial cancer based on protein biomarkers in cervical fluids

## BACKGROUND

Endometrial cancer (EC) is the fourth most common cancer in women, and its incidence is increasing. Despite the importance of early diagnosis for patient survival, there is no accurate and non-invasive strategy for early triage or screening of EC. Diagnosis relies on imaging and invasive biopsies, affecting more than 35M women presenting symptoms -86% postmenopausal and 14% premenopausal- and patients with hereditary risk -Lynch Syndrome- who have annual surveillance. Only 10% of those will have EC.

## OBJECTIVE

This project builds on our previous projects, and aims to **advance the development of a non-invasive triage test based on protein biomarkers in cervical fluids for the accurate detection of atypical endometrial hyperplasia (AEH) and EC across high-risk populations.** We will advance the development, preanalytical and analytical validation of the test using the recently developed key reagents, and assess its performance in symptomatic pre- and postmenopausal women prior and after imaging techniques, and Lynch Syndrome carriers. In parallel, we will investigate the proteomic landscape of EC progression using a transgenic mouse model, and cervical fluids from AEH patients, to elucidate tumor progression mechanisms and AEH biomarkers, with the ultimate goal to advance our triage test for EC prevention. This project is expected to transform EC diagnosis, improving patient care, quality of life, EC survival, and healthcare efficiency.

## THE POSITION

The Vall d'Hebron Research Institute (VHIR) is seeking a Postdoctoral Researcher to join the Group of Biomedical Research in Gynaecology. The position is integrated into a scientific environment of excellence and is highly dynamic, focusing on high-end

biomedical projects. The successful candidate will lead translational research focused on the diagnosis of endometrial cancer and endometriosis using proteomics in complex human fluids. This full-time role includes the following responsibilities and duties:

- Participate in translational research projects to improve endometrial cancer and endometriosis diagnosis through biomarker research.
- Collaborate with other projects of the group, by promoting fruitful discussions or by helping in experimental work.
- Participate in general activities of the group.
- Support the principal researcher in managerial activities.

## THE GROUP

The **Group of Biomedical Research in Gynaecology (GineLab)**, integrated into the Vall d'Hebron Research Institute (VHIR), is a consolidated, multidisciplinary core consisting of 37 professionals who operate under a "two-way translational research" philosophy: from the patient to the laboratory and back to the clinic. The group is co-directed by Dr. Antonio Gil-Moreno, who leads the clinical strategy, and Dr. Eva Colás, who is responsible for translational research and innovation.
Their activity is centered on resolving clinical needs in women's health through two main pillars: early non-invasive diagnosis and precision oncology. The research is structured into three primary vertical work lines:

- **Endometrial Cancer:** This research line is a pioneer in molecular diagnosis using gynecological fluids (such as cervical fluid), employing proteomics to develop non-invasive triage tests aimed at avoiding unnecessary biopsies.
- **Ovarian Cancer:** This research line focuses on precision oncology and therapy optimization. To this end, they utilize the GynePDX platform, a biobank featuring over 174 patient-derived xenograft (PDX) models and organoids, which is used for preclinical "clinical trials" of targeted therapies.
- **Endometriosis:** This research line integrates molecular classification and tissue proteomic characterization with the development of a non-invasive diagnostic test based on protein biomarkers in cervical fluids, while simultaneously investigating angiogenic biomarkers to improve recurrence risk prediction.

**Innovation and Clinical Translation** GineLab stands at the forefront of clinical innovation through a robust portfolio of active patents and the creation of its own femtech spin-off, **MiMARK Diagnostics S.L.** (founded in 2021). This spin-off has secured approximately €8M in funding, including a prestigious €2.5M EIC Accelerator grant, to bring non-invasive in vitro diagnostic (IVD) tests to the global healthcare market.

**International Leadership and Strategic Collaborations:** The group holds a prominent position in the international scientific community:

- **Consortia Coordination:** GineLab coordinates the European CytoMARK consortium (ERA PerMed), integrating proteomic markers and clinical data across five countries.
- **Accreditations:** The clinical headquarters at VHH maintains ESGO Accreditation as a Centre of Excellence for the surgical management of advanced ovarian cancer.
- **Strategic Alliances:** The group maintains high-level collaborations with institutions such as the Broad Institute of MIT and Harvard, the Luxembourg Institute of Health, and numerous European and US universities.
- **Scientific Governance:** Members hold leadership roles in the European Network for Individualized Treatment of Endometrial Cancer (ENITEC), the European Society of Gynaecological Oncology (ESGO), and the national Spain-GOG network.

**Social Impact and Talent Development**: Beyond technical excellence, the group drives social change through the ENDO-HIT program, a pioneer citizen-science initiative that raises endometriosis awareness in schools and empowers women regarding their reproductive health. Furthermore, GineLab is a proven engine for academic development, having supervised 6 PhD theses with honors since 2022 and maintaining a robust internal training program that attracts international talent from across Europe.

## GROUP'S RELATED PUBLICATIONS


- Martinez-Garcia, E. *et al.* (2017). Targeted Proteomics Identifies Proteomic Signatures in Liquid Biopsies of the Endometrium to Diagnose Endometrial Cancer and Assist in the Prediction of the Optimal Surgical Treatment. *Clinical cancer research : an official journal of the American Association for Cancer Research*, *23*(21), 6458–6467. https://doi.org/10.1158/1078-0432.CCR-17-0474
- Martinez-Garcia, E. *et al.* (2023). Cervical Fluids Are a Source of Protein Biomarkers for Early, Non-Invasive Endometrial Cancer Diagnosis. Cancers, 15(3), 911. https://doi.org/10.3390/cancers15030911
- Luzarraga Aznar, A. *et al.* (2025). Current challenges and emerging tools in endometrial cancer diagnosis. *International journal of gynecological cancer : official journal of the International Gynecological Cancer Society*, *35*(4), 100056. https://doi.org/10.1016/j.ijgc.2024.100056
- Cabrera, S. *et al.* (2024). Molecular classification improves preoperative risk assessment of endometrial cancer. *Gynecologic oncology*, *189*, 56–63. https://doi.org/10.1016/j.ygyno.2024.07.003
- Felip, I., Moiola *et al.* (2019). Therapeutic potential of the new TRIB3-mediated cell autophagy anticancer drug ABTL0812 in endometrial cancer. *Gynecologic oncology*, *153*(2), 425–435. https://doi.org/10.1016/j.ygyno.2019.03.002

## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - PhD in Biology, or similar sciences degrees
  - Fluency in English
  - First author in publications
  - Passionate, proactive, willing to learn and committed

- **Experience and Knowledge**
  **Required:**

  - Previous experience in a translational research group
  - Experience in mass spectrometry and immunoassay, including experimental work and data analysis

  **Desirable:**

  - Previous experience with handling of complex human samples
  - Previous postdoc experience
  - Efficient in manuscript drafting

## CONTACT INFORMATION

**Dra. Eva Colás**: apply

AI, Data, Imaging & Digital Health

Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.010

# Beyond Gap Junctions: Connexins as Orchestrators of Tumor–Stroma Communication

## BACKGROUND

We are looking for an ambitious postdoctoral researcher to develop an independent Marie Skłodowska-Curie Fellowship project at the interface of cancer cell communication, metabolism, and tumor microenvironment biology.
Connexin 43 (Cx43) presents a long-standing paradox in cancer: it can act as either a tumor suppressor or a tumor promoter depending on context. Beyond its classical role as a gap junction protein, Cx43 regulates tunneling nanotube (TNT) formation, mitochondrial transfer, and extracellular vesicle (EV) signalling. Its alternatively translated isoform GJA1-20k further controls actin remodeling and mitochondrial dynamics, suggesting that connexins play a broader role in coordinating communication between tumor cells and their microenvironment.
This project will test the hypothesis that Cx43 acts as a master regulator of intercellular communication, linking these pathways to tumor plasticity and therapeutic resistance across breast, lung, and pancreatic cancer.

## OBJECTIVE

The aim is to define how connexin-regulated tumor-stroma communication drives cancer progression and therapy resistance. Possible research directions include: (1) how Cx43 and GJA1-20k regulate TNT formation and organelle transfer; (2) non-canonical mitochondrial exchange pathways including annular gap junctions; (3) Cx43 control of EV biogenesis and signalling; and (4) how these pathways collectively regulate metabolic adaptation and therapy resistance. The project will be developed together with the fellow based on their expertise and interests.

## THE POSITION

This is not a position where you execute someone else's project. You will define your own questions, develop your own ideas, and build your scientific identity. Mentoring and support for the MSCA application are part of the deal, but scientific ownership is yours.
The position is based at the Vall d'Hebron Research Institute (VHIR) in Barcelona, one of Europe's leading biomedical research institutes, embedded within a major university hospital. You will have access to advanced live-cell and super-resolution imaging, cell

engineering platforms, and well-established cancer models, as well as active collaborations with Marc Liesa (mitochondrial metabolism and redox biology) and Henrique Girão (EV biology and connexin signalling).
The goal is to provide the successful candidate with both scientific freedom and strong mentorship, supporting not only the MSCA application but also the longer-term transition toward research leadership.

## THE GROUP

The Intercellular Communication Group, led by Trond Aasen, studies connexin-regulated tumor progression, metastasis, and therapy resistance in breast, lung, and pancreatic cancer. We combine mechanistic cell biology with advanced imaging and functional cancer models. Recent tools include dual-color HiBiT-based mitochondrial tracing systems for quantifying intercellular organelle transfer. This is a growing field with real room for discovery.

## GROUP'S RELATED PUBLICATIONS


- **Aasen T**, Mesnil M, Naus CC, Lampe PD, Laird DW. *Gap junctions and cancer: communicating for 50 years.* Nat Rev Cancer. 2016 Dec;16(12):775-788. doi: 10.1038/nrc.2016.105. PMID: 27782134
- Tishchenko A, Azorín DD, Vidal-Brime L, Muñoz MJ, Arenas PJ, Pearce C, Girao H, Ramón Y Cajal S, **Aasen T.** *Cx43 and Associated Cell Signaling Pathways Regulate Tunneling Nanotubes in Breast Cancer Cells.* Cancers (Basel). 2020 Sep 29;12(10):2798. doi: 10.3390/cancers12102798.
- Martins-Marques T, Costa MC, Catarino S, Simoes I, **Aasen T**, Enguita FJ, Girao H. *Cx43-mediated sorting of miRNAs into extracellular vesicles.* EMBO Rep. 2022 Jul 5;23(7):e54312. doi: 10.15252/embr.202154312.


## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**
  - PhD in cancer biology, cell biology, molecular biology, biochemistry, or related discipline.
  - Excellent scientific communication in English

  **Desirable:**
  - Strong publication record.
  - International research experience

- Experience with fellowship writing is a plus

- **Experience and Knowledge Required:**

  - Strong experimental background in mammalian cell biology
  - Experience in molecular cloning and cell engineering

  **Desirable:**

  - Experience in one or more of the following:
  - mitochondrial biology, cancer metabolism, extracellular vesicles, tunneling nanotubes, live-cell imaging, CRISPR editing, omics or bioinformatics. Experience in fluorescence microscopy/confocal imaging
  - Demonstrated ability to drive independent research
  - Strong written and oral communication skills

## CONTACT INFORMATION

**Dr. Trond Aasen:** apply

Innovative & Advanced Therapies

Ref: VHIR-MSCA-PF-2026.011

# Understanding primary and adaptive tumor immune resistance

## BACKGROUND

Cancer immunotherapy has reshaped the treatment landscape for multiple cancers, offering long-lasting clinical benefit to some patients. However, a large proportion of tumors remain unresponsive or eventually acquire resistance, revealing a major gap in our understanding of how cancer cells communicate with and manipulate the immune system. Increasing evidence suggests that stress-signalling pathways within tumor cells can profoundly influence immune surveillance, inflammation, and the composition of the tumor microenvironment. In this context, p38α emerges as a compelling molecular hub linking tumor adaptation, immune suppression, and therapeutic response.

## OBJECTIVE

The proposed project will investigate how tumor cell–intrinsic p38α controls immune evasion and shapes the tumor microenvironment in the context of resistance to immune checkpoint blockade. Despite major advances in cancer immunotherapy, the mechanisms driving primary and acquired resistance remain insufficiently understood. Recent findings indicate that p38α regulates the expression of immune checkpoint molecules and promotes an immunosuppressive microenvironment by enhancing macrophage and myeloid-derived suppressor cell activity while limiting $CD8^+$ T-cell and NK-cell cytotoxicity; importantly, pharmacological inhibition of p38α synergizes with PD-1 blockade to induce durable tumor regressions in vivo. The successful candidate will define the transcriptional and post-transcriptional programs controlled by p38α, identify the immune cell populations first affected by tumor-derived p38α signalling, and determine how these interactions remodel the TME. This project aims to uncover actionable mechanisms of immunotherapy resistance and support the rational development of combination treatments, including strategies to select patients most likely to benefit from p38α inhibition plus immune checkpoint therapy.

## THE POSITION

The Biomedical Melanoma Research Group at the Vall d'Hebron Research Institute (VHIR), Barcelona, invites expressions of interest from outstanding postdoctoral

researchers interested in developing competitive applications for the next Marie Sklodowska-Curie Postdoctoral Fellowships (MSCA-PF) and/or AECC Talent postdoctoral calls.
VHIR offers an excellent setting for fellowship development at the interface of basic, translational, and clinically relevant cancer research. Candidates joining our group will have the opportunity to develop a high-impact project supported by complementary expertise in in vivo models, human samples, and omics-driven discovery.

## THE GROUP

Our group focuses on skin cancer, investigating the molecular mechanisms that drive disease to design effective therapies. We **work in close proximity to patients** so that discoveries can be translated to the clinic as rapidly as possible. Accordingly, we are a **multidisciplinary team that brings together dermatologists, oncologists, pathologists and basic scientists** who jointly define **clinically relevant questions** and generate the biological answers needed to address them. Our workflow integrates **patient-derived information** (tumor deep sequencing, immunohistochemistry, clinical history, etc.) and transfers these data into animal models and in-vitro systems. Experimental results guide the **development of new therapeutic strategies** and preclinical studies that, in turn, inform clinical implementation.

## GROUP'S RELATED PUBLICATIONS


- **p38α Orchestrates *BRAF$^{V600E}$*-Melanoma Progression and Immunotherapy Response via Tumor Fitness and Immune Microenvironment Modulation**
  Granado-Martínez P, McGrail K, Orsenigo R, Caratù G, Nieto P, Ding Y, Nebreda A, Martín-Caballero J, Ortega I, Eixarch H, Espejo C, Hernandez-Losa J, Ferrer B, Heyn H, Muñoz-Couselo E, García-Patos V and Recio JA. Nat Commun. 2026 Under review.
- **Transcriptional reprogramming triggered by neonatal UV radiation or Lkb1 loss prevents *BRAF$^{V600E}$*-induced growth arrest in melanocytes.**
  McGrail K, Granado-Martínez P, Orsenigo R, Caratù G, Nieto P, Heyn H, Ferrer B, Hernández-Losa J, Muñoz-Couselo E, García-Patos V, Recio JA.
  Oncogene. 2025 Jun;44(21):1592-1608. doi: 10.1038/s41388-025-03339-7. Epub 2025 Mar 8.
- **BRAF activation by metabolic stress promotes glycolysis sensitizing *NRAS$^{Q61}$*-mutated melanomas to targeted therapy.**
  McGrail K, Granado-Martínez P, Esteve-Puig R, García-Ortega S, Ding Y, Sánchez-Redondo S, Ferrer B, Hernandez-Losa J, Canals F, Manzano A, Navarro-Sabaté A, Bartrons R, Yanes O, Pérez-Alea M, Muñoz-Couselo E, Garcia-Patos V, Recio JA. Nat Commun. 2022 Nov 19;13(1):7113. doi: 10.1038/s41467-022-34907-0.

## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - PhD degree with **1–2 years of postdoctoral experience**, with the **most recent years of research experience obtained outside Spain**.

  **Desirable:**

  - Knowledge bioinformatics

- **Experience and Knowledge**
  **Required:**

  - We are looking for **highly motivated candidates** interested in m**elanoma biology, tumor immunology, cancer metabolism, and therapy response or resistance** and experience in one or more of the following areas melanoma biology, tumor immunology, immunotherapy, cancer metabolism, immunometabolism, animal models, single-cell or spatial omics, bioinformatics, systems oncology or translational cancer research.

  **Desirable:**

  - Knowledge in bioinformatics and FACs

## CONTACT INFORMATION

**Dr. Juan A. Recio:** apply

Rare Diseases

Transplant & Immunomodulation

Ref: VHIR-MSCA-PF-2026.012

# Exploring the Interplay of Chronic Stress and Antiphospholipid Autoantibodies on Trophoblast Function and the Induction of Preeclampsia

## BACKGROUND

Preeclampsia is a severe pregnancy complication characterised by hypertension and multi-organ dysfunction, representing a major cause of maternal and perinatal morbidity. Increasing evidence indicates that preeclampsia originates from defective embryo implantation and abnormal placentation during early pregnancy. Chronic psychological stress is known to disrupt neuroendocrine and immune homeostasis, while antiphospholipid autoantibodies (aPL) are strongly associated with obstetric complications, including preeclampsia.
Despite these associations, the molecular and cellular mechanisms linking chronic stress, autoimmunity, and placental dysfunction remain poorly understood. Recent advances in human trophoblast organoid models provide a unique opportunity to study early placental development and maternal–fetal interactions in a physiologically relevant system. Understanding how stress hormones and aPL synergistically impair trophoblast function is essential to uncover novel pathogenic pathways involved in preeclampsia.

## OBJECTIVE

The main objective of this project is to determine whether the synergistic action of chronic stress and antiphospholipid autoantibodies disrupts trophoblast function and uterine immune regulation, leading to abnormal implantation, defective placentation, and the development of preeclampsia.
Specifically, the project aims to:

1. Assess the effects of stress hormones and aPL on trophoblast and uterine NK cell function using human trophoblast organoid models.
2. Identify molecular, transcriptomic, and proteomic alterations induced by stress and autoimmunity during early placentation.
3. Validate key pathogenic mechanisms in a preclinical mouse model of stress- and autoimmunity-induced preeclampsia.

## THE POSITION

The postdoctoral researcher will join a highly multidisciplinary research environment at **Vall d'Hebron Institut de Recerca (VHIR)**, working at the interface of immunology, reproductive biology, and translational medicine. The position offers access to state-of-the-art facilities, including spectral flow cytometry, spatial transcriptomics, proteomics platforms, and advanced organoid culture systems.
The researcher will collaborate closely with clinical and basic scientists and will be actively involved in experimental design, data analysis, dissemination of results, and preparation of competitive funding applications, including MSCA Postdoctoral Fellowship

## THE GROUP

The project will be hosted by the **Systemic Autoimmune Diseases Research Group** at VHIR, and will be led by Francesc Miró Mur. The group has extensive expertise in autoimmune diseases, obstetric antiphospholipid syndrome, immune profiling, and translational research. The team has built large clinical cohorts, leads international collaborations, and has a strong publication record in high-impact journals.
The group collaborates closely with clinicians and international research centres specialising in maternal–fetal medicine, placental biology, and immunology, providing a robust and interdisciplinary training environment for postdoctoral researchers.

## GROUP'S RELATED PUBLICATIONS


- Anunciación-Llunell A, et al. Differences in Antiphospholipid Antibody Profile between Patients with Obstetric and Thrombotic Antiphospholipid Syndrome. Int J Mol Sci, 2022; 23(21):12819. doi: 10.3390/ijms232112819
- Jarne-Borràs M, et al. Antiphospholipid antibodies in women with recurrent embryo implantation failure: A systematic review and meta-analysis. Autoimmunity Reviews, 2022; 21(6):103101. doi: 10.1016/j.autrev.2022.103101
- Alijotas-Reig J, et al. Pathogenesis, Diagnosis and Management of Obstetric Antiphospholipid Syndrome. Journal of Clinical Medicine, 2022; 11(3):675. doi: 10.3390/jcm11030675
- Esteve-Valverde E, et al Low complement levels are related to poor obstetric outcomes in women with obstetric antiphospholipid syndrome. The EUROAPS Registry Study Group. Placenta. 2023; 138:20. doi: 10.1016/j.placenta.2023.04.016


## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

- PhD in Immunology, Reproductive Biology, Biomedical Sciences, or a related field.

**Desirable:**

- Background in placental biology, autoimmunity, or maternal–fetal medicine.

- **Experience and Knowledge**
  **Required:**

  - Strong experience in cellular and molecular biology techniques.
  - Demonstrated ability to analyse and interpret complex biological data.

  **Desirable:**

  - Experience with organoid models, flow cytometry, multi-omics approaches, or animal models.
  - Scientific writing experience and interest in competitive funding applications

## CONTACT INFORMATION

**Dr. Francesc Miro Mur**: apply

Vall d'Hebron Research Institute

Environmental & Public Health | Innovative & Advanced Therapies | Transplant & Immunomodulation

Ref: VHIR-MSCA-PF-2026.013

# Inducing CD8$^+$T cell Tissue Resident Phenotypes

## BACKGROUND

CD8$^+$ resident memory T cells (**CD8$^+$T$_{RM}$**) permanently reside within tissues, where they continuously survey the local microenvironment and provide rapid protection against invading pathogens and malignant cells. Beyond their effector functions, CD8$^+$T$_{RM}$ play key roles in maintaining tissue immune homeostasis. The differentiation of memory precursors into CD8$^+$T$_{RM}$ is shaped by instructive cues derived from both secondary lymphoid organs and peripheral tissue niches. A detailed understanding of the mechanisms governing CD8$^+$T$_{RM}$ development, establishment, and regulation in mucosal tissues will be critical to harnessing these cells for the elimination of persistent antigens and for enhancing vaccine-induced immune responses.

## OBJECTIVE

The primary **objective** of this proposal is to identify mechanisms that are key for ultimately inducing a CD8$^+$T$_{RM}$ response to migrate and establish for long-term protection in multiple peripheral tissues and mucosa. Understanding how to best engage innate immunity at mucosal sites and how induce long-term CD8$^+$T$_{RM}$ in these sites will be key for adjuvant and vaccine formulation or to potentiate these responses to eliminate persistent antigens located in these tissues.

## THE POSITION

The research group led by **Dr. Meritxell Genescà** at the **Vall d'Hebron Institute of Research (VHIR)** investigates the mechanisms shaping tissue immune responses, with a particular focus on host-pathogen interactions. The group integrates fundamental immunology with translational approaches to understand how immune cells are regulated within tissue environments and how these responses can be modulated to improve protection against infection and disease. The group is embedded within the HIV laboratory and operates in a highly collaborative setting, closely interacting with clinicians and researchers at **Vall d'Hebron University Hospital,** which is a major hospital, enabling access to clinically relevant samples and advanced experimental models. **VHIR** is a leading biomedical research institute in Spain, internationally recognized for its excellence in translational research. Located within the Vall d'Hebron campus in

Barcelona, VHIR provides state-of-the-art infrastructureand an outstanding training environment for students and postdoctoral researchers.

## THE GROUP

The Mucosal Immunology against Infection (MIM), is led by Dr. Meritxell Genescà, a senior researcher at the Infectious Disease dept. of the Vall d'Hebron Insitut de Recerca (VHIR). The main lines of research are:

- Tissue HIV reservoir and new elimination strategies
- STI and co-infections
- Adjuvants and biomarkers of mucosal immunity
- Tissue explant models to study host-pathogen interactions and immunity.

## GROUP'S RELATED PUBLICATIONS

- https://pubmed.ncbi.nlm.nih.gov/34021148/
- https://www.nature.com/articles/s41467-023-37559-w
- https://www.nature.com/articles/s41467-019-12732-2

## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**
  - PhD in Immunology, Biomedical Sciences, Microbiology Biology, Biochemistry, veterinary or a closely related discipline.
  - Solid academic training in cellular and molecular immunology
  - Fluency in spoken and written English

  **Desirable:**
  - PhD with a strong focus on tissue immunity, mucosal immunology, host-pathogen interactions, or infectious diseases
  - Prior exposure to translational or clinically oriented research environments
  - Competitive publication record appropriate to career stage

- **Experience and Knowledge**
  **Required:**
  - Hands-on experience with immunological techniques (e.g. flow cytometry, multiparametric phenotyping, cell culture, functional immune assays)

- Experience working with primary human samples and/or tissue material
- Strong experimental design, data analysis, and interpretation skills
- Ability to work independently while contributing effectively to a collaborative research environment
- Motivation to conduct rigorous, hypothesis-driven research at the interface of basic and translational science
- Good analytical, writing and communicating skills.

**Desirable:**

- Experience collaborating with clinicians or working within multidisciplinary consortia
- Interest in mentoring students and contributing to lab and institutional scientific activities
- Knowledge of infectious diseases, viral immunology, or co-infection biology
- Experience in data analyses, bioinformatics and statistics.

## CONTACT INFORMATION

**Dra. Meritxell Genescà**: apply

Innovative & Advanced Therapies

Transplant & Immunomodulation

Ref: VHIR-MSCA-PF-2026.014

# Identification and Genetic Engineering of Cytotoxic Tissue NK Cells for Cellular Therapy

## BACKGROUND

The project is based on the emerging role of tissue-resident and tissue-adapted immune cells in immune surveillance, chronic inflammation, viral persistence and cancer control. Among them, Natural Killer (NK) cells are promising candidates for immunotherapy due to their intrinsic cytotoxic activity against infected and transformed cells. However, current NK-cell therapies mainly rely on peripheral blood-derived NK cells, which often show limited persistence and tissue functionality. Recent advances in tissue immunology and single-cell technologies have revealed extensive heterogeneity among human tissue NK-cell populations, including subsets with enhanced cytotoxicity, adaptive-like features and tissue-residency programs. Nevertheless, their functional characterization and therapeutic exploitation remain poorly understood.

Our laboratory combines human tissue models with advanced immunological and molecular approaches to identify and characterize tissue-associated NK-cell subsets with potent effector functions. The research integrates high-dimensional phenotyping, functional assays, ex vivo tissue explants and genetic engineering strategies, including CRISPR-based approaches, to develop next-generation NK-cell therapies for cancer and infectious diseases.

The candidate will work at the interface of tissue immunology, translational immunotherapy and cell engineering within a highly collaborative research environment.

## OBJECTIVE

- To identify and characterize human tissue-associated NK-cell subsets with enhanced cytotoxic and persistence capacities using advanced immunophenotyping, single-cell analyses and functional tissue models.
- To define the molecular and functional programs underlying tissue adaptation and antitumor/antiviral activity of NK cells within human tissue microenvironments.
- To develop and evaluate genetically engineered NK-cell products with improved therapeutic potential for next-generation cellular immunotherapy.

## THE POSITION

The host laboratory is part of the Vall d'Hebron Research Institute, closely integrated with Vall d'Hebron University Hospital, providing access to clinical cohorts, patient-derived samples and translational research infrastructures. The group conducts interdisciplinary research at the interface of tissue immunology, virology and cellular immunotherapy, with a strong focus on human tissue models and translational applications.

The laboratory combines high-dimensional flow cytometry, single-cell technologies, ex vivo tissue explant systems and functional assays to study immune responses in physiologically relevant microenvironments. In parallel, the group develops next-generation NK-cell engineering strategies using CRISPR-based editing and gene-delivery technologies for cellular therapy applications.

The candidate will benefit from a highly collaborative international environment and access to state-of-the-art core facilities, including flow cytometry, genomics, imaging and preclinical platforms.

## GROUP'S RELATED PUBLICATIONS


- **Resident CD49a+CD103+NKG2C+ NK cells restrict HIV infection in human lymphoid tissue explants**. Perea D, Gonzalez A, Gallego-Cortés A, Sanchez-Gaona N, Pumarola F, Ortiz N, Llano I, Lorente J, Falcó V, Genescà M, Buzon MJ. *Science Advances*. 2026 Jan 23;12(4):eadz1565. doi: 10.1126/sciadv.adz1565. Epub 2026 Jan 23. PMID: 41576166
- **Identification of inducible HIV reservoirs in tonsillar, intestinal and cervical tissue models of HIV latency.** Gallego-Cortés A, Sánchez-Gaona N, Mancebo-Pérez C, Ruiz-Isant O, Benítez-Martínez A, Landolfi S, Castellví J, Pumarola F, Ortiz N, Llano I, Lorente J, Mañalich-Barrachina L, Prado JG, Martín-Gayo E, Falcó V, Genescà M, Buzon MJ. *Nature Communications*. 2025 Nov 24;16(1):10353. doi: 10.1038/s41467-025-65288-9. PMID: 41285793
- **NKG2C and NKG2A coexpression defines a highly functional antiviral NK population in spontaneous HIV control.** Sánchez-Gaona N, Gallego-Cortés A, Astorga-Gamaza A, Rallón N, Benito JM, Ruiz-Mateos E, Curran A, Burgos J, Navarro J, Suanzes P, Falcó V, Genescà M, Buzon MJ. *JCI Insight*. 2024 Sep 17;9(20):e182660. doi: 10.1172/jci.insight.182660. PMID: 39288262


## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

- PhD in Immunology, Cell Biology, Molecular Biology, Biotechnology, Virology, Biomedical Sciences or a related discipline.
- Excellent communication skills in English (written and spoken).

- **Experience and Knowledge Required:**

  - Minimum of two first-author publications in Q1 peer-reviewed journals.
  - Ability to work independently and collaboratively within an international multidisciplinary environment.

  **Desirable:**

  - Experience in immunology, cellular therapy, tissue immunology and/or NK-cell biology.
  - Hands-on experience with flow cytometry, immune functional assays and mammalian cell culture.
  - Experience in molecular biology and/or genetic engineering techniques (e.g. CRISPR, viral transduction, gene editing approaches) will be highly valued.
  - Experience working with human samples and translational research models.

## CONTACT INFORMATION

**Dra. Maria J. Buzon**: apply

AI, Data, Imaging & Digital Health

Rare Diseases

Ref: VHIR-MSCA-PF-2026.015

# Federated and interpretable clinical AI for earlier diagnosis of inborn errors of immunity

## BACKGROUND

Inborn errors of immunity (IEI) are rare, heterogeneous diseases in which diagnostic delay remains clinically relevant. Current referral pathways often depend on recognition by highly specialised clinicians or tertiary registries, which are accurate but difficult to scale and may amplify inequities in access to expert centres. At the same time, primary care and hospital electronic health records contain longitudinal signal: recurrent infections, bronchiectasis, cytopenias, autoimmunity, growth impairment, immunoglobulin patterns and other coded or laboratory features, that can be transformed into computable phenotypes.

The host group has developed PIDCAP, an expert-derived EHR-integrated scoring system for early identification of IEI in primary care, and is expanding this work through ontology mapping, AI-enabled clinical decision support, calculated globulin/hypogammaglobulinaemia detection, and federated learning collaborations. The proposed MSCA project will build on this platform to create generalisable, transparent and privacy-preserving methods for earlier detection of rare immune disorders in real-world health systems.

## OBJECTIVE

- Develop and validate interoperable EHR phenotypes and clinical features for early suspicion of IEI, combining expert knowledge, ICD/laboratory data and ontology mapping.
- Design interpretable prediction and risk-stratification models that can be calibrated for different clinical settings while minimising alert fatigue.
- Implement a privacy-preserving validation strategy, including federated or distributed analytics, to assess transportability across centres or regions.
- Evaluate clinical workflow integration, fairness and potential impact on referral pathways from primary care to specialised immunology units.

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## THE POSITION

The fellow will join a translational programme at VHIR focused on clinical AI, rare disease screening and digital health implementation. The position will be embedded in the Infection and Immunity in Pediatric Patients group and will work closely with clinical immunologists, data scientists and health informatics teams, including the DS3 group and primary care collaborators in Catalonia. Depending on the candidate's profile, the project may emphasise statistical learning, federated analytics, clinical NLP, ontology-driven feature engineering, or implementation science.

The fellow will have access to an active clinical research environment, real-world EHR use cases, international scientific networks in IEI and AI, and opportunities to contribute to European collaborations on interoperability, privacy-preserving data analysis and learning health systems. Expected outputs include methodological publications, validated reusable code or data models, and a roadmap for clinically safe deployment of IEI screening tools.

## THE GROUP

The Vall d'Hebron Research Institute Infection and Immunity in Pediatric Patients group focuses on improving the understanding, diagnosis and management of congenital errors of immunity in children. Its research combines clinical immunology, genomics, translational immunology, advanced immune monitoring and data science to support earlier and more precise diagnosis and personalised care. The group is closely linked to a highly specialised clinical unit at Vall d'Hebron University Hospital and collaborates with national and international networks in primary immunodeficiencies, rare diseases, artificial intelligence and clinical data interoperability.

In parallel, the group integrates artificial intelligence tools to analyse complex clinical and biological datasets, identify predictive biomarkers and optimise diagnostic workflows. In the last year, the group reported 48 publications, 72.9% in Q1 journals, 140 citations, 12 ongoing projects and 16 active clinical trials, reflecting strong translational and collaborative activity.

## GROUP'S RELATED PUBLICATIONS


- **Rivière JG**, Cantenys-Saba R, Carot-Sans G, Piera-Jiménez J, Butte MJ, **Soler-Palacín P**, Peng XP. Current perspectives and challenges of using artificial intelligence in immunodeficiencies. J Allergy Clin Immunol. 2025 Oct;156(4):878-888.
- **Rivière JG**, Carot-Sans G, Piera-Jiménez J, de la Torre S; PIDCAP expert group; Cos X, Serra-Picamal X, **Soler-Palacin P**. Development of an Expert-Based Scoring System for Early Identification of Patients with Inborn Errors of Immunity in Primary Care Settings - the PIDCAP Project. J Clin Immunol. 2024 Oct 21;45(1):26.


- **Soler-Palacín P**, **Rivière JG**, Burns SO, Rider NL. New tools for diagnosis of primary immunodeficiencies: from awareness to artificial intelligence. Front Immunol. 2025 Jul 10;16:1593897.


## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - PhD in health data science, bioinformatics, biomedical engineering, epidemiology, medicine, immunology, public health, computer science or a related field.
  - Eligibility and mobility requirements for the MSCA Postdoctoral Fellowship call.

  **Desirable:**

  - Background in rare diseases, immunology, clinical epidemiology, digital health, real-world data, health informatics or implementation science.

- **Experience and Knowledge**
  **Required:**

  - Strong quantitative skills
  - Experience with R or Python
  - Reproducible data analysis
  - Statistical modelling or machine learning
  - Capacity to work with multidisciplinary clinical and technical teams
  - Excellent scientific writing in English

  **Desirable:**

  - Experience with EHR data, ICD/SNOMED/ORPHA/HPO/OMOP or openEHR/FHIR standards, federated learning or privacy-preserving analytics, NLP, explainable AI, clinical decision support evaluation, or research software engineering.

## CONTACT INFORMATION

**Dr. Pere Soler Palacín and Dr. Jaques Rivière:** apply

Rare Diseases

Ref: VHIR-MSCA-PF-2026.016

# Functional validation of genetic variants in pediatric inborn errors of immunity

## BACKGROUND

Next-generation sequencing has improved the diagnosis of inborn errors of immunity (IEI), but it has also created a growing bottleneck: candidate variants and variants of uncertain significance (VUS) that cannot be translated into diagnosis, counselling or targeted treatment without functional evidence. This is especially relevant in children with severe infection susceptibility, immune dysregulation, cytopenias, autoinflammation, combined immunodeficiency or complex syndromic presentations.

The project will build on the pediatric IEI programme, the Advanced Immunology Testing Laboratory and the joint laboratory activity established between the Infection and Immunity in Pediatric Patients Research Group (I2P2-RG) and the Translational Immunology at VHIR. It will combine deep clinical phenotyping, advanced immunophenotyping, primary human cell assays, engineered cellular systems and selected *in vivo* models to move from genomic suspicion to experimentally supported pathogenicity.

## OBJECTIVE

- Establish a structured VUS validation pipeline integrating phenotype, segregation, genomic/structural variant analysis, transcript-level evidence, immune profiling and ACMG/AMP-oriented interpretation.
- Develop patient-derived functional assays using PBMCs, lymphocyte subsets, neutrophils and/or fibroblasts, with readouts adapted to the suspected pathway.
- Interrogate priority pathways relevant to IEI, including JAK/STAT, NF-kB, PI3K-AKT/mTOR, lymphocyte activation, antibody deficiency and oxidative burst/host-defence mechanisms.
- Generate complementary human models, including stimulated primary cells, immortalised or CRISPR-edited cell systems, rescue/overexpression experiments and multi-omic readouts where appropriate.
- Use selected animal or preclinical models, according to biological plausibility and feasibility, to assess immune development, infection susceptibility or inflammatory phenotypes in vivo.

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- Translate validated results into variant reclassification, genotype-phenotype refinement, diagnostic reporting and precision therapeutic decisions for children with IEI.

## THE POSITION

The fellow will join a translational wet-lab environment embedded in I2P2-RG and closely connected to the Pediatric Infectious Diseases and Immunodeficiencies Unit at Vall d'Hebron University Hospital. The position will be developed in close collaboration with the Translational Immunology group, creating a joint human immunology/genetics laboratory setting for IEI variant prioritisation and functional validation.

Depending on the candidate's profile, the project may focus on primary immune-cell assays, advanced flow cytometry and phospho-flow, cytokine and Luminex assays, ELISA, Western blotting, transcript/cDNA analysis, cell culture, genome editing, structural variant interpretation or animal/preclinical modelling. Expected outputs include validated functional workflows, mechanistic studies of selected genes or pathways, publications linking genotype to immune phenotype, and clinically actionable evidence for diagnosis and treatment.

## THE GROUP

The Vall d'Hebron Research Institute Infection and Immunity in Pediatric Patients Research Group, led by Dr Pere Soler-Palacín, focuses on pediatric infectious diseases and immunodeficiencies, with a strong translational orientation from bedside phenotyping to laboratory validation and personalised management. The group studies congenital errors of immunity, severe and unusual infections, immune dysregulation, biomarkers, targeted therapies and diagnostic innovation.

The group coordinates an Advanced Immunology Testing Laboratory and works in a shared laboratory ecosystem with the Translational Immunology group at VHIR. This setting provides access to well-characterised pediatric IEI cohorts, clinical samples, immunophenotyping platforms, molecular genetics, bioinformatics, biobank resources, animal facilities and preclinical/functional validation support. In the last year, the group reported 48 publications, 72.9% in Q1 journals, 140 citations, 12 ongoing projects and 16 active clinical trials.

## GROUP'S RELATED PUBLICATIONS


- Batlle-Masó L, Padrosa Pulido J, Esteve-Codina A, **Soler-Palacin P**, et al. Somatic reversion in CD137 deficiency correlating with Epstein-Barr virus control and clinical improvement. npj Genomic Medicine. 2025;10:78. doi:10.1038/s41525-025-00535-y.
- Urban B, Batlle-Masó L, Perurena-Prieto J, Garcia-Prat M, Parra-Martínez A, Aguiló-Cucurull A, Martinez-Gallo M, Moushib L, Antolín M, **Rivière JG, Soler-Palacin P**, Dieli-Crimi R, Franco-Jarava C, Colobran R. Heterozygous Predicted Loss-of-function Variants of TRAF3 in Patients with Common Variable Immunodeficiency. Journal of Clinical Immunology. 2025;45:47. doi:10.1007/s10875-024-01833-3.
- Garcia-Prat M, Batlle-Masó L, Parra-Martínez A, Franco-Jarava C, Martinez-Gallo M, Aguiló-Cucurull A, Perurena-Prieto J, Castells N, Urban B, Dieli-Crimi R, **Soler-Palacín P,** Colobran R. Role of Skewed X-Chromosome Inactivation in Common Variable Immunodeficiency. Journal of Clinical Immunology. 2024;44:54. doi:10.1007/s10875-024-01659z.


## WHAT DO WE LOOK FOR?

- **Education and qualifications**
  **Required:**

  - PhD in immunology, molecular biology, cell biology, genetics, biomedicine, biotechnology, biochemistry, human biology or a related field.
  - Fulfilment of MSCA Postdoctoral Fellowship eligibility and mobility requirements.

  **Desirable:**

  - Previous work in IEI/primary immunodeficiencies, immune dysregulation, autoinflammation, host-pathogen susceptibility, clinical immunology, rare disease genetics or functional genomics.

- **Experience and Knowledge**
  **Required:**

  - Solid wet-lab background
  - Cell culture and cell isolation
  - Advanced flow cytometry and panel design
  - Experimental design
  - Data analysis
  - Excellent scientific writing in English

- Capacity to work across clinical and laboratory teams.

**Desirable:**

- Western blotting, ELISA, Luminex/multiplex assays, phospho-flow, cytokine stimulation assays, lymphocyte proliferation, neutrophil oxidative burst, qPCR/RT-qPCR or cDNA analysis, CRISPR/Cas9 or other genome editing, animal models, biobanking, anonymised patient databases and ACMG/AMP variant interpretation.

## CONTACT INFORMATION

**Dr. Pere Soler Palacín and Dr. Jaques Rivière:** apply