



## **Master in Biomedical Research**

**2024-2025**

List of potential laboratories

Other laboratories would also be accepted

(by alphabetical order using the last name of each principal investigator)

Note: **admissions to the Master in Biomedical Research (BIOMED) are prioritized for students that have been accepted to do his/her research *practicum* in a research laboratory.**

A main orientation of the BIOMED master is to continue towards a PhD thesis, and the majority of students enrolling in this master have, at the time of pre-registration, been accepted in research groups to do a PhD after they finish the master. However, there are students who have a motivation to do this master, and eventually a PhD, but who may not know how to contact a suitable laboratory.

This document contains a few guidelines to help candidate students in finding a research group, and also a list of potential laboratories to which they can submit applications. **These groups have expressed their interest for hosting a master student.**

This list is orientative, and students can do their research in another laboratory that they can find on their own. Also, this list is not just to choose a laboratory: you must contact the group you are interested in, arrange an interview, and get the written acceptance of the investigator in charge of that group.

## “How to: getting accepted in a research laboratory”

**1- You have to know what you would like to work on.**

**2- Be specific: you should be able to say what are the questions that are important to you and why.**

*Not very good: I want to work in neurosciences, I have always liked it.*

*Much better: I want to understand the processes and mechanisms that make neurons more sensitive to oxidative stress and oxygen deprivation in patients with neurodegenerative diseases such as...*

*Not very good: I want to work in regenerative medicine, I think that stem cells have a lot of potential to cure diseases.*

*Much better: I want to contribute to the identification of proteins that when expressed in a differentiated cell such as a fibroblast, can cause it to dedifferentiate and acquire functional characteristics of a pluripotent cell.*

**3- Find out who is working on what.**

Websites of universities and research centers, PubMed searches, Google...

*It takes time! (don't wait till last minute to begin looking for your favorite lab)*

**4- Write to the group that interests you.**

**5- Contacting a group.**

**a) Motivation letter:** tell them why you want to work with them (for this, you need to know something about what they do and about current questions in the field).

*It takes time! (don't wait till last minute to begin looking for your favorite lab)*

Also tell them why you are good. Labs appreciate commitment, responsibility, ability to work in a team, ability to persevere and a strong motivation.

Ask them for an interview to show them how good you are.

Do not write a generic letter to copy-paste and send to ten different laboratories changing only the name of the group leader.

Choose your labs and send a personal, specific letter to each one.

**b) Keep in mind** that a person working full time in a cellular-molecular biology lab can spend more than 1000 euros/month in materials, besides a lot of time required to train you and supervise you until you begin to get solid results.

Expect that during the first 6 months it is more likely that you will produce more trouble and expenses than productive results. Laboratories are very careful with how they

spend their money because they get their funding from competitive grants that are given or denied based on productivity (that means getting results) and publications in internationally respected journals.

Do not get discouraged with rejections, learn from them to improve your application.

**c) Do not forget important details in your CV:**

1- Give names of senior persons that can be a reference.

Be careful with “clone” reference letters from teachers that don’t really know you and will just say general things.

2- Include your university scores. If they are not too good, you should be ready to explain why, either in your application letter or in an interview. Be honest and realistic about it. If the teaching/exam system of your university hasn’t worked for you, you will know the reasons better than anyone else, so be prepared to speak frankly about it.

University scores are not an exact indicator of who will become a successful scientist, but they say that a person has gone through 4-5 years of serious effort with a better than average performance.

Most people will interpret this as a sign of self-discipline, organization, capacity to work even if you have a bad day, and to get things done regardless of whether they are more fun or plainly boring.

## **Master in Biomedical Research**

**2024-2025**

### List of potential laboratories

(the list is in alphabetical order using the last name of each principal investigator)

*Note: the list of groups is orientative. Students can also find by themselves research groups (in Spain or abroad) that are not in this list, and submit the acceptance letter of the researcher responsible (principal investigator) of the group together with the rest of the documentation when they send their application to this master*

*See also important information about projects and groups in the next page*

Admissions to the Master in Biomedical Research (BIOMED) are prioritized for students that have been accepted in a research group for their *practicum*.

If more than two students are opting to the same group, the master coordinator may recommend that some of the applicants be redirected to other groups based on their motivation profile and academic criteria.

**a) If you have already been accepted to a research group** for doing the master research *practicum*, please submit the acceptance letter by the group's supervisor/director. The group can be in Spain or in a foreign country.

**b) If you do not have a host research group at the time of registering**, you must indicate your first 5 choices, in order of preference, **from the list of groups** offered by the BIOMED master. Knowing these choices gives us additional information to assess your application.

Important:

**b1)** When listing your 5 choices, please write the name of the PI for each group. Don't just say "group in tumor modelling" or something like that.

Besides telling us your 5 choices from the list, you can search for a research group (in Spain or abroad) that is not in this list. You do not need to indicate that in addition to your 5 choices above.

**b2)** Keep in mind that indicating your choices does not mean that you will be assigned to a group automatically. You are encouraged to actively seek acceptance in a group because having a group will increase your chances of being accepted to the master.

You must contact the group you are interested in (from the list provided here or from your own searches), arrange an interview, and get the written acceptance of the investigator in charge of that group.

The next page outlines some guidelines to help candidate students to find a research group.

It also has a list of potential laboratories to which you can submit applications. This list can be updated with some new groups in the next months.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Antiprions: structural variomics against neurodegenerative diseases

**Project supervisor** (principal investigator of the laboratory/group)

Name: Martí Aldea

eMail: [marti.aldea@ibmb.csic.es](mailto:marti.aldea@ibmb.csic.es)

Group name: Spatial control of biomolecular dynamics

Institution: Molecular Biology Institute of Barcelona (IBMB-CSIC)

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/>

**Main grant associated with this project:**

Principal investigator: Martí Aldea

Agency: *Agencia Estatal de Investigación*

Reference/ years: PID2022-141460NB-I00, 3 years

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Prions and prion-like aggregates have been directly implicated in more than 20 human diseases, among them neurodegenerative pathologies such as Alzheimer's, Parkinson's and Huntington's diseases, and amyotrophic lateral sclerosis. Prion and prion-like proteins are self-propagating protein isoforms that accumulate as large structure-driven aggregates. It is generally accepted that their accumulation in the human brain is a direct cause of neuronal degeneration. However, appropriate therapeutic approaches and effective treatments are largely lacking, and efforts to decrease the rate of prion-like aggregation with peptides have produced very limited results. We have recently performed a highly-sensitive variomics screen on A $\beta$ 42, the  $\beta$ -amyloid peptide that accumulates in the brain of patients with Alzheimer's disease, and we have identified a number of A $\beta$ 42 mutants with strong antiprion properties. In this specific project you will perform comprehensive functional assays of these candidate antiprions to validate their ability to counteract prion-like aggregation and deleterious effects in neuronal connectivity and survival. The discovery and functional analysis of these antiprions will constitute the foundational basis of preclinical and clinical assays directed to test and refine their use in Alzheimer's disease.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** METABOLIC CONTROL OF IMMUNE RESPONSES

**Project supervisor** (principal investigator of the laboratory)

Name: **Jose Aramburu**

Mail: jose.aramburu@upf.edu

Group name: GENIMMUNE

Institution: Universitat Pompeu Fabra

Webpage of the group: <https://www.upf.edu/web/genimmune>

<https://www.upf.edu/web/jose-aramburu>

**Main grant associated with this project:**

Principal investigator: Jose Aramburu and Cristina López-Rodríguez

Agency: Plan Estatal I+D+i, Ministerio de Ciencia e Innovación, Agencia Estatal de Investigación

Reference/ years: PID2021-128721OB-I00 (2022-2025)

**Brief summary of the project or current research lines of the group**

Metabolism regulates immune responses, both ensuring energy and metabolites necessary for immune functions as well as influencing gene expression and functional specialization of immune cells. Metabolism and immune response co-regulate each other, and immune cells are capable of adapting their metabolism to be able to function in different niches and even hostile conditions, while on the other hand altered metabolic conditions can lock immune cells in a detrimental functional state.

We are currently studying how metabolism influences diverse immune responses in two pathological settings, obesity and cancer. We have combined high-throughput RNA-sequencing analyses and metabolomics to identify metabolic pathways and metabolism-regulated gene signatures and functions in different populations of immune cells in these scenarios.

We offer a master position in a project that will aim at modifying specific metabolic pathways in T lymphocytes and macrophages to redirect immune responses and enhance their therapeutic effectiveness in cancer and obesity. The selected candidate will acquire conceptual fluency in current trends in immunometabolism research, in parallel with hands-on experience in diverse cellular, molecular and immune function techniques (for instance flow cytometry, CRISPR-directed deletions, gene expression, chromatin analyses, cell differentiation assays, metabolic activity, antitumor function) of primary immune cells isolated from gene-edited mice under different pathological settings.

Leading publications of the group:

Riera-Borrull et al., 2023 under review

Lunazzi et al., 2021 Journal of Immunology

Huerga Encabo et al., 2020 Journal of Experimental Medicine

Aramburu and López-Rodríguez, 2019 Frontiers in Immunology

Buxadé et al., 2018 Journal of Experimental Medicine

Tellechea et al., 2018 Journal of Immunology

Aramburu et al., 2014 Science Signaling

Berga-Bolaños et al., 2013 Proc Natl Acad Sci USA

Buxadé et al., 2012 Journal of Experimental Medicine

Ortells et al., 2012 Nucleic Acids Research



**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Investigating DYRK1A-regulated functions in early neocortical development using mouse models and 3D human brain organoids.

**Project supervisor** (principal investigator of the laboratory/group)

Name: Mariona Arbonés

eMail: [marbmc@ibmb.csic.es](mailto:marbmc@ibmb.csic.es)

Group name: Proliferation and Differentiation of the Nervous System

Institution: Institut de Biologia Molecular de Barcelona, IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/proliferation-and-differentiation-of-the-nervous-system/>

**Main grant associated with this project:**

Principal investigator: Mariona Arbonés

Agency: AEI (Agencia Estatal de Investigación), Spanish Ministry of Science, Innovation and Universities

Reference/ years: PID2022-137564OB-I00/2023-2026

**Brief summary of the project or current research lines of the group**

DYRK1A, an ubiquitously expressed protein kinase, is a critical player in various signalling pathways crucial for nervous system development and adult homeostasis. Human *DYRK1A* is located on chromosome 21, and its overexpression due to trisomy contributes to morphological and functional brain alterations observed in Down syndrome. Moreover, *de novo* mutations in a single allele of *DYRK1A* give rise to a distinct syndromic form of intellectual disability and autism known as DYRK1A syndrome, characterized by intrauterine growth restriction, microcephaly, speech delay or complete absence of communicative language, motor dysfunctions and a distinctive facial gestalt.

This project aims to elucidate the cellular and molecular events regulated by DYRK1A during early neurogenesis, with a specific focus on the neocortex, the brain region responsible for higher cognitive functions. We will use a *Dyrk1a*<sup>+/-</sup> mouse model closely mirroring the neurological features of DYRK1A syndrome, alongside brain-specific mouse conditional *Dyrk1a* mutants and 3D brain organoids derived from isogenic human embryonic stem cells carrying pathogenic mutations in *DYRK1A*.

The master's student will actively participate in the analysis of brain tissues and 3D organoids using confocal microscopy, as well as the examination of DYRK1A-substrate interactions through co-immunoprecipitation assays.

This research holds potential to unveil novel therapeutic avenues for treating and managing patients with DYRK1A syndrome and related syndromes, including Down syndrome.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Cell Cycle Control: Regulation of the G1/S Transition

**Project supervisor** (principal investigator of the laboratory/group)

Name: José Ayté

eMail: jose.ayte@upf.edu

Group name: Oxidative Stress and Cell Cycle

Institution: UPF

Webpage of the group: <https://www.upf.edu/web/osccg/>

**Main grant associated with this project:**

Principal investigator: José Ayté

Agency: Ministerio de Ciencia, Innovación y Universidades

Reference/ years: PID2022-136449NB-I00 (2023-2026)

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

In our laboratory, we are ultimately interested in deciphering the mechanisms that control cell cycle progression using fission yeast as model organism. Recently, we have started a new project to obtain a more profound understanding of how CDKs (the central core of the cell cycle machinery) are regulated. Cdc2 (CDK1) is the single CDK kinase described to regulate cell cycle progression in fission yeast, like in all other yeasts. However, despite several global phosphoproteomic and genome-wide studies to determine which are the target(s) of the CDK activity in the G1/S transition, up-to-now it is unknown which are the primary targets of Cdc2 in this cell cycle phase. We have been recently investigating the role of an alternative CDK, Pef1, which was originally described to be involved in controlling TORC1 pathway and autophagy. Interestingly, we have observed an impact on cell cycle regulation, since cells lacking Pef1 are smaller than wild type cells. Using different biochemical and cell biology approaches, we can demonstrate that Pef1 is required for completion of mitotic S phase. We have analysed the proteome and phosphoproteome of a wild type,  $\Delta pef1$ , and a conditional Pef1 strains from asynchronous cultures and we have found several candidate proteins that can be phosphorylated by Pef1. The candidate will determine direct targets of Pef1 involved in cell cycle progression using some wide-range technologies, including protein purification, microscopic fluorescence quantification, cytometry and CRISPR-directed mutagenesis.

Some related publications from the group are:

Borao et al. (under revision)

Salat-Canela et al. **Trends Cell Biol.** 33:124-137

Hummer et al. **Cell Rep.** 37:109893

Salat-Canela et al. **Cell Rep.** 37: 109951

González-Medina et al. **Nucleic Acids Res.** 47:8439-8451

Alves-Rodrigues et al **Cell Reports** 14:885-895

Eckert et al. **PLoS Genet.** 12:e1005768

Gomez-Escoda et al. **EMBO Rep.** 12:84-89

Moldon et al. **Nature** 455:997-1000

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Unraveling the therapeutic potential of human serine proteases in breast cancer. Novel chemical probes and inhibitors.

**Project supervisor** (principal investigator of the laboratory/group)

Name: Marta Barniol-Xicota

eMail: [marta.barniol@upf.edu](mailto:marta.barniol@upf.edu)

Group name: Lab of Chemical Biology

Institution: Universitat Pompeu Fabra

Webpage of the group: [www.barniolxicotalab.com](http://www.barniolxicotalab.com)

**Main grant associated with this project:**

Principal investigator: Dr. Marta Barniol-Xicota

Agency: LaCaixa / UPF Internal

Reference/ years: 2022-2025

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Our research group uses an interdisciplinary approach that blending medicinal chemistry, molecular biology and cell biology, with the ultimate goal of **discovering new therapeutic targets and biomarkers, with a focus on breast cancer and metastatic progression**. For this, my lab will focus on these specific projects:

1. **Lipid metabolism as target for breast and resistant cancers.** Aberrant lipid metabolism and is a hallmark of cancer that affects cell proliferation and response to therapeutics. We develop new chemical tools and inhibitors for key enzymes involved in lipid metabolism, to validate their use as prognostic biomarkers and in cancer therapeutics.

2. **Targeting intramembrane proteolysis in human pathology.** Rhomboid proteases are embedded in the lipid bilayer where they cleave protein substrates. Despite being linked to important diseases, their specific roles remain undefined, hampering their use in therapeutics. We aim to elucidate: (1) What are the roles of human rhomboids? (2) Can we enable their use as therapeutic targets?

3. **Chemically modified phage display to develop substrate and covalent probes for medically relevant enzymes,** allows us to overcome the greatest challenge in the development of chemical probes, that is to prepare selective probes in an unbiased and timely manner. In my lab we are optimizing and expanding the scope of this methodology to selectively target cancer-linked enzymes.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Role of Histone H1 on genome stability

**Project supervisor** (principal investigator of the laboratory/group)

Name: Jordi Bernués

eMail: jbmbmc@ibmb.csic.es

Group name: Chromatin structure and function

Institution: IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-structural-and-molecular-biology/chromatin-structure-and-function/>

**Main grant associated with this project:**

Principal investigator: F. Azorín/J. Bernués

Agency: Ministerio de Ciencia e Innovación

Reference/ years: PID2021-123303NB-I00/Sept2022-Sept2026

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Our current research lines include:

(I) the analysis of the contribution of **linker histones H1** to the epigenetic regulation of chromatin structure and function

(II) the study of the factor and mechanisms involved in formation and maintenance of the **tridimensional (3D) organization of chromatin** inside the nucleus

(III) the analysis of the **epigenetic** factors and mechanism that regulate **transcription**

(IV) the study of **centromeric chromatin** and its contribution to chromosome segregation and mitosis progression

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

Project Title: Investigating the molecular roles of the functional domains of macroH2A histone variants: Unstructured Linker (project 1), Metabolite Binding Macrodomain (project 2)

Project supervisor (principal investigator of the laboratory/group)

Name: Marcus Buschbeck

eMail: mbuschbeck@carrerasresearch.org

Group name: Chromatin, Metabolism and Cell Fate

Institution: Josep Carreras Leukaemia Research Institute

Webpage of the group: <https://buschbecklab.org/>

Local co-supervision by Oliver Meers and Jonathan Blickenberger

Main grant associated with this project:

Principal investigator: NucleoSense - Enhancer regulation and nuclear metabolite sensing by histone variants Agency: Ministerio de Ciencia e Innovación (MCIN)

Reference/ years: (PID2021-126907NB-I00), 2022-2025

Brief summary of the project or current research lines of the group (please do not include pictures or logos and do not exceed this page):

MacroH2A histone variants (macroH2A1.1, macroH2A1.2, macroH2A2) can replace the H2A component of the nucleosome and have various known effects on nuclear and chromatin structure and gene expression (Corujo et al., 2022). The proteins each consist of 3 domains, a H2A-homologous histone fold, a short, unstructured linker domain and a metabolite binding macrodomain. The linker domain has been shown in vitro to affect protein aggregation and protection of extra-nucleosomal DNA from digestion (Chakravarthy et al., 2012; Muthurajan et al., 2011). MacroH2A1.1's macrodomain is known to bind ADP-ribosylated PARP1 (Guberovic et al., 2021; Marjanović et al., 2017), but the macrodomains of macroH2A1.2 and macroH2A2 remain orphan. We are currently progressing our understanding of these proteins on two fronts:

1. Our current linker research expands on the previous in vitro research. We have shown that this domain also affects nuclear organization, heterochromatin composition and chromatin expansion after DNA damage (Douet et al., 2017; Kozłowski et al., 2018). Our unpublished work shows that the linker is also responsible for changes in gene expression and could be responsible for macroH2A chromatin enrichment. We aim to further investigate this and elucidate the molecular mechanism of this domain.

2. Building on the knowledge and relevance of macroH2A1.1 on NAD<sup>+</sup> homeostasis (Guberovic et al., 2021), we want to further dissect isoform-specific functions of the macrodomains, especially in human cancers. We currently focus on computational, biochemical and cell biological approaches to identify ligands or interaction partners of the two orphan macrodomains. Subsequently, we will dissect the cellular consequences of binding and elucidate the underlying molecular mechanisms.

Both of these projects can potentially include a variety of techniques and analysis:

- Molecular and Cell Biology: Western Blotting, cloning, generation of complex cell lines, RTq-PCR, variety of cellular assays, imaging
- Genomic analysis: NGS, CUT&RUN
- Proteomics: IP-Mass Spectrometry, molecular modelling

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Deciphering cellular crosstalk between leukemic and stromal cells in co-culture models

**Project supervisor** (principal investigator of the laboratory/group)

Name: Dr. Marcus Buschbeck, Dr. René Winkler (co-supervisor)

eMail: [mbuschbeck@carrerasresearch.org](mailto:mbuschbeck@carrerasresearch.org), [rwinkler@carrerasresearch.org](mailto:rwinkler@carrerasresearch.org)

Group name: Chromatin, metabolism and cell fate

Institution: Josep Carreras Leukaemia Research Institute

Webpage of the group: <https://www.carrerasresearch.org/en/research/chromatin-metabolism-and-cell-fate>, <https://buschbecklab.org/>

**Main grant associated with this project:**

Principal investigator: Marcus Buschbeck

Agency: Asociación Española Contra el Cáncer (AECC)

Reference/ years: PRYGN222668BUSC

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Myelodysplastic syndrome (MDS) is a hematopoietic disorder marked by ineffective blood cell production, where mesenchymal stromal cells (MSCs) in the bone marrow play a crucial role. In MDS, an altered bone marrow microenvironment favors the clonal growth of mutant stem cells, contributing to disease progression. To investigate new MDS treatment approaches, we established a 2D co-culture system of MSCs and MDS cells, aiming to understand the relationship between these cells better. Our goal is to use genetic approaches, such as knock-down and knock-out, and small molecules targeting epigenetic modifiers to manipulate MSCs. As read-out, we use colony formation assays, proliferation, and flow cytometry-based measurements to characterize the effects on MDS cells. Importantly, we also isolate primary hematopoietic stem cells from patient samples and use them in our co-culture systems to gain insights into physiological cell-to-cell communication. The goal of our research is to develop novel therapeutic strategies for MDS.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title: Investigating the bioenergetic link between cannabinoid receptors and Alzheimer Disease**

**Project supervisor** (principal investigator of the laboratory/group)

Name: Arnau Busquets Garcia

eMail: abusquets@researchmar.net

Group name: Cell-type Mechanisms in Normal and Pathological Behavior

Institution: Hospital del Mar Research Institute

Webpage of the group:

[https://www.imim.cat/programesrecerca/neurociencies/en\\_mecanismes\\_cellulars.html](https://www.imim.cat/programesrecerca/neurociencies/en_mecanismes_cellulars.html)

**Main grant associated with this project:**

Principal investigator: Arnau Busquets Garcia

Agency: Agencia estatal de Investigación

Reference/ years: ASTROCAD project (ref. PID2021-122795OB-I00; 2022-2025)

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Alzheimer's disease (AD) has been associated with numerous alterations of brain bioenergetic processes including alterations in astroglial metabolism and mitochondrial functions. Type-1 cannabinoid (CB1) receptors, one of the main components of the endocannabinoid system (ECS), control different intercellular processes such as cell metabolism. Importantly, alterations of different components of the ECS have been described both in AD animal models and in human patients. Moreover, *in vitro* and *in vivo* pharmacological activation of CB1 receptors displayed efficacy in reducing the neurotoxic effects of amyloid-B peptide and to counteract the cognitive impairment found in AD mouse models. However, further research is needed to decipher the specific mechanisms linking ECS and AD.

CB1 receptors are functionally present in different cell-types (neurons and astrocytes) both in the plasmatic and mitochondrial membranes. In this project, we hypothesed that CB1R-dependent cell-type specific bioenergetic processes participate in the pathophysiology of AD. To address this hypothesis, different state-of-the-art approaches and the coordinated use of suitable animal models, combined with *in vivo* and *ex vivo* genetic, behavioral, viral, pharmacological and biochemical approaches will be adopted.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Understanding the involvement of brain circuits in higher-order conditioning

**Project supervisor** (principal investigator of the laboratory/group)

Name: Arnau Busquets Garcia

eMail: abusquets@researchmar.net

Group name: Cell-type Mechanisms in Normal and Pathological Behavior

Institution: Hospital del Mar Research Institute

Webpage of the group:

[https://www.imim.cat/programesrecerca/neurociencies/en\\_mecanismes\\_cellulars.html](https://www.imim.cat/programesrecerca/neurociencies/en_mecanismes_cellulars.html)

**Main grant associated with this project:**

Principal investigator: Arnau Busquets Garcia

Agency: European Research Council

Reference/ years: Highmemory project (ref. 948217; 2021-2026)

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Animals and humans adapt to changes in the environment encoding and storing previous experiences. Although associative learning involving a reinforcer has been the major focus in the field of cognition, other forms of learning are gaining popularity as they are likely to be both more significant and frequent in human daily choices. Indeed, associations between non-reinforcing stimuli represent the most evolutionarily advanced way to increase the chances of predicting future events and adapting an individuals' behaviour. Animals are also able to utilise these higher-order conditioning processes, but more research is needed to understand how the brain encode and store these complex cognitive processes.

This Master project will be performed in the general frame of the HighMemory project, which is investigating brain circuits in higher-order conditioning processes in mouse models. These processes explain why individuals are very often repulsed or attracted by stimuli (people, places, sounds), which have no intrinsic repellent or appealing value and have never been explicitly paired with negative or positive outcomes. A possible explanation for these "ungrounded" aversions or repulsions is that these stimuli have been incidentally associated with other directly reinforced cues. This is called higher-order conditioning or mediated learning (ML). Importantly, these behavioural processes involve the hippocampus, are characterised by defined and accessible phases and involve several brain regions, making them perfect models for studying tight behaviour regulation by different brain circuits. By using genetic (viral and chemogenetic techniques), Ca<sup>2+</sup> imaging, and mouse behavioural (sensory preconditioning) approaches, the aim of the HighMemory project is at dissecting and characterizing, at the macro- (brain regions), meso- (cell sub-types), and micro-scales (activity changes), the causal involvement of hippocampo-cortical projections in higher-order cognitive processes.



## Call for project proposals

Master in Biomedical Research practicum, 2025

Department of Medicine and life Sciences, Universitat Pompeu Fabra

**Project Title:** Metastasis immunity and mRNA therapy

**Project supervisor** (principal investigator of the laboratory/group)

Name: Toni Celià-Terrassa

eMail: [acelia@researchmar.net](mailto:acelia@researchmar.net)

Group name: *Cancer Stem Cells & Metastasis Dynamics Lab*

Institution: Hospital del Mar Research Institute (IMIM); PRBB

Webpage of the group: <https://celiaterassalab.com/>

### **Main grant associated with this project:**

Principal investigator: Toni Celià-Terrassa

Agency: CAIXA HEALTH RESEARCH\_23

Reference/ years: 2023-2026

**Brief summary of the project or current research lines of the group (please do not include pictures or logos)**

### **Research Lines of the group:**

- Immunotherapy and mRNA therapies in metastatic breast cancer
- Immune interplay of Cancer Stem Cells (CSCs) in breast cancer metastasis
- Epithelial-to-mesenchymal transition (EMT) and cancer cell plasticity during metastasis

Note: candidates may choose the topic depending on priorities.

### **Breast cancer metastasis immunity and immunotherapy**

**Summary.** Breast cancer contains subpopulations of aggressive cancer stem-like cells (CSCs) responsible for initiating tumor growth, metastasis, and relapse after therapy. By activating stem cell-like programs, breast cancer cells acquire properties similar to those of normal mammary stem cells (MaSCs). We have recently revealed that breast CSCs emerging from ICB therapy have an altered transcriptome and signalling pathways that may confer their resistance. The research plan is designed to determine the dynamic evolution of metastatic stem cells (MetSCs)-immune interactions in the metastatic organs and immunotherapy resistance. Preclinical experimental models of breast cancer metastasis in vivo will be used to decipher MetSC and immune interactions. In addition, patient-derived xenografts, and patient clinical samples will be used to validate the clinical significance of our results. Despite the widely known aggressiveness of CSCs – including MetSCs – no specific inhibitors are effective against this population; therefore, better understanding of their biology will offer innovative new mRNA therapeutic opportunities using nanoparticles in combination with immunotherapy.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Overcoming the tumor microenvironment-induced resistance to therapy

**Project supervisor** (principal investigator of the laboratory/group)

Name: Alexandre Calon

eMail: [acalon@researchmar.net](mailto:acalon@researchmar.net)

Group name: Translational Research in Tumor Microenvironment

Institution: Hospital del Mar Research Institute

Webpage of the group: <https://orcid.org/0000-0002-3398-6131>; <https://twitter.com/CalonLab>

**Main grant associated with this project:**

Principal investigator: Alexandre Calon

Agency: Spanish Association Against Cancer (AECC)

Reference/ years: LABAE235294CALO (2023-2026)

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

The recent advances in the clinical management of cancer highlighted the immense tumor heterogeneity between patients. Common biomarkers used in the clinical practice are capturing features intrinsic to cancer cells to try to dissect such heterogeneity and guide therapeutic strategies. However, results are often contradictory, thus leading to patients being either over or undertreated. Importantly, cancer cells do not exist as isolated entities, but rather reside in an interactive tumor microenvironment composed of non-malignant cells that largely contributes to cancer progression. Our project aims to identify the tumor microenvironment parameters that modulate treatment response. Specifically, we are investigating the biological and molecular basis of the stromal heterogeneity and plasticity upon systemic and targeted therapy using state of the art patient-derived organoids, cancer-associated fibroblasts and immune cells, ex vivo immunocompetent models together with transcriptomics, tissue bio-imaging and in vivo models. Understanding the impact of the non-malignant cells permeating the tumor on treatment outcomes will be crucial to improve patient treatment guideline.

Our laboratory aims at bridging the gap between basic scientific findings and their application in clinical settings. This translational research seeks to translate insights from laboratory studies into practical applications that can improve the diagnosis, treatment, and understanding of cancer in clinical practice.

Recent lab publications related to the topic:

**Long-term platinum-based drug accumulation in cancer-associated fibroblasts promotes colorectal cancer progression and resistance to therapy, *Linares et al., Nat Commun 2023***

**Peptide–Platinum(IV) Conjugation Minimizes the Negative Impact of Current Anticancer Chemotherapy on Nonmalignant Cells, *Linares et al., J. Med. Chem. 2023***

**Targeted immunotherapy against distinct cancer-associated fibroblasts overcomes treatment resistance in refractory HER2+ breast tumors, *Rivas et al., Nat Commun 2022***

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Unraveling the cell biology of the closest relatives of animals

**Project supervisor** (principal investigator of the laboratory/group)

Name: Elena Casacuberta/ Iñaki Ruiz-Trillo

eMail: elena.casacuberta@ibe.upf-csic.es/inaki.ruiz@ibe.upf-csic.es

Group name: MulticellGenome Lab

Institution: Institute of Evolutionary Biology (CSIC-UPF)

Webpage of the group: <https://multicellgenome.com>

**Main grant associated with this project:**

Principal investigator: Elena Casacuberta/ Iñaki Ruiz-Trillo

Agency: Ministerio Español de Ciencia e Innovación

Reference/ years: Reference/ years: PID2020-120609GB-I00 2021/2024

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Have you ever wondered how in evolution the transition from unicellular to multicellular organisms took place? In our laboratory we study this enigmatic and crucial evolutionary question from a diverse combination of approaches from genome wide analysis, cell and molecular biology as well as phylogenetics. More specifically, we aim to understand the origins of cell differentiation, the importance of phenotypic plasticity, reconstruct the genome of the unicellular ancestor or understand the evolutionary history of genome regulation at the onset of animals.

With these objectives in mind, we are working with the closest unicellular relatives of animals belonging to the four known clades at the moment, choanoflagellates, filastereans, ichthyosporeans and corallochytreaans. In the last years we have advance substantially in making them experimentally treatable expanding notably the possibilities to functionally test hypothesis. In this framework we have a range of Master projects that can be offered to students of the Biomedical Research Master from the University Pompeu Fabra. The Master Student would receive training and experience in latest advances in molecular and cell biology and high-resolution optical and fluorescent microscopy.

More information and pictures and videos of those taxa can be found at:  
"<https://www.flickr.com/people/146564503@N06/>"  
"<https://www.youtube.com/user/multicellgenomeLab>")

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** CRISPR-Cas TO MODEL HUMAN GENETIC DISEASES IN *Caenorhabditis elegans*

**Project supervisor** (principal investigator of the laboratory/group)

Name: JULIAN CERON MADRIGAL

eMail: jceron@idibell.cat

Group name: Modelling human diseases in *C. elegans*

Institution: Bellvitge Biomedical Research Institute (IDIBELL)

Webpage of the group: [www.ceronlab.com](http://www.ceronlab.com) and [www.idibell.cat](http://www.idibell.cat)

**Main grant associated with this project:**

Principal investigator: Julián Cerón Madrigal

Agency: Ministerio de Ciencia e Innovación

Reference/ years: Modelling cancer mutations in *C. elegans*, until 2025, Ref WORMVUS, from Plan de Recuperación, Transformación y Resiliencia - Financiado por la Unión Europea – NextGenerationEU. Renewing Plan Nacional Grant in 2024.

)

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Our lab uses the powerful genetic model *C. elegans* to investigate human diseases: rare diseases (Kukhtar et al, 2020) or cancer (Serrat et al, 2019). We have broad expertise in CRISPR technologies that are being applied to model human diseases (*ex. by introducing human mutations in C. elegans*). Moreover, we have an active research line on optimizing CRISPR genome editing by creating methodologies or by using distinct Cas9 enzymes (Vicencio et al, 2019; 2021). Thus, a Master's research project in our lab would include molecular biology, CRISPR, and classic genetics. The student will participate in any ongoing projects focused on modeling genetic diseases in *C. elegans* with the chance to explore innovative CRISPR methods.

**Recent publications:**

*Caenorhabditis elegans* for research on cancer hallmarks. Cerón J. *Dis Model Mech*. 2023 Jun 1;16(6):dmm050079. doi: 10.1242/dmm.050079.

Genome editing in animals with minimal PAM CRISPR-Cas9 enzymes. Vicencio et al, *Nature Communications*. 2022 May 12;13(1):2601. doi: 10.1038/s41467-022-30228-4.

Mimicking of splicing-related retinitis pigmentosa mutations in *C. elegans* allow drug screens and identification of disease modifiers. Kukhtar D, et al. *Human Molecular Genetics* 2020 doi: 10.1093/hmg/ddz315.

CRISPR editing of *sftb-1/SF3B1* in *Caenorhabditis elegans* allows the identification of synthetic interactions with cancer-related mutations and the chemical inhibition of splicing. Serrat X et al, *PLoS Genetics*. 2019 Oct 21;15(10):e1008464. doi: 10.1371/journal.pgen.1008464

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Analysis of the human genome diversity in order to unravel demographic and genomic processes

**Project supervisor** (principal investigator of the laboratory/group)

Name: David Comas

eMail: david.comas@upf.edu

Group name: Human Genome Diversity Group

Institution: UPF

Webpage of the group: <https://www.biologiaevolutiva.org/dcomas/>

**Main grant associated with this project:**

Principal investigator: David Comas

Agency: Ministerio de Ciencia e Innovación (Spain)

Reference/ years: PID2022-138755NB-I00 (2023-2026)

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

The interests of our research are focused on the **human genome diversity analysis** in order to infer the (genomic and population) processes responsible for this diversity and try to establish the (population and epidemiological) consequences of the human genetic variability. Thus, our main research lines are focused on aspects of human genome diversity, population genetics, genome variation and disease susceptibility, and genome evolution and disease.

**1. Population processes** Concerning **population processes** that have modeled the human genetic diversity, we have focused our research on the use of molecular tools to reconstruct the human population history through the phylogeny of genetic markers. Our interest has been focused on the genetic consequences at population level of human migrations and admixtures. The use of well-established phylogenies in the mitochondrial and Y-chromosome human genomes allowed us to unravel the population history of several populations. Nonetheless, we have recently used whole genome variation in the autosomes in order to establish the structure of human populations.

**2. Genomic processes** Concerning **genomic processes** that have modeled the human genetic diversity, our research has been focused on the relationship between human diversity and complex traits, including complex diseases. The genetic analysis in human populations of genes of biomedical interest might shed light on the evolution of these genes. In this context, we have focused our research in the analysis of genes that have been previously associated to complex diseases, such as psychiatric and immunological diseases. The analysis of these genes has allowed us to conclude that some of the failures in replicating genetic associations are due to extreme genetic differences between populations. In addition, we are also interested in other complex traits, such as height, not directly related to disease.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and Life Sciences, Universitat Pompeu Fabra**

**Project Title:** Functionalization of Bacterial Flagella for Biotechnology and Biomedicine.

**Project supervisor** (principal investigator of the laboratory/group)

**Name:** Ulrich Eckhard

**eMail:** [ulrich.eckhard@ibmb.csic.es](mailto:ulrich.eckhard@ibmb.csic.es)

**Group name:** Synthetic Structural Biology

**Institution:** Molecular Biology Institute of Barcelona (IBMB-CSIC)

**Webpage of the group:** <https://www.ibmb.csic.es/en/department-of-structural-and-molecular-biology/synthetic-structural-biology/>

**Main grant associated with this project:**

**Principal investigator:** Ulrich Eckhard

**Agency:** Ministerio de Ciencia e Innovación

**Reference (years):** RYC2020-029773-I (2022-2027), PID2021-128682OA (2022-2025)

**Brief summary of the project or current research lines of the group:**

We are excited to welcome motivated students to join our research team for their master thesis projects. Our focus is on advancing biotechnological and biomedical applications built on our discovery of [proteolytic flagellins](#), a family of flagellin proteins exhibiting enzymatic capabilities, and nature's proof for functionalized bacterial flagella. Projects within our lab entail diverse molecular biology, biochemistry, and microbiological tasks, including but not limited to molecular cloning utilizing Golden Gate assembly, recombinant protein expression and purification, bacterial motility testing, functional testing, microbial imaging, structural modeling and characterization, and structure-function analysis. Throughout these activities, students will receive comprehensive guidance and supervision from experienced researchers within the lab. Furthermore, it is of great importance to us to create a supportive environment conducive to student training and development, and we are dedicated to assisting students in navigating their academic and scientific journeys, guiding them towards their next steps in their careers.

**About the lab:** We are a newly established research team dedicated to investigating the biological implications of proteolytically active flagella and exploring the biotechnological and biomedical potential of functionalized flagella. Situated at the [Parc Científic de Barcelona](#), a prominent life science research hub in Spain, our location provides proximity to over 90 companies alongside major research institutes like the Institute for Research and Biomedicine (IRB), the Institute of BioEngineering of Catalonia (IBEC), and our institute, the [IBMB-CSIC](#). As a part of the Structural and Molecular Biology Department, we have close collaboration with numerous distinguished research teams and enjoy access to cutting-edge research equipment and facilities, including the [IBMB Molecular Imaging Platform](#), the [Automated Crystallography Platform](#), and [JEMCA, the CryoEM facility at ALBA Synchrotron](#).

**Candidate specifications:** We welcome motivated candidates with a strong interest in Molecular Biology, Microbiology, Biochemistry, Structural Biotechnology, and/or Biomedicine to submit their CV and academic records to Ulrich Eckhard ([ulrich.eckhard@ibmb.csic.es](mailto:ulrich.eckhard@ibmb.csic.es)). Fluency in English, a collaborative spirit, and excellent communication and problem-solving skills are highly valued.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Uncovering the mechanisms driving fetal conversion in colorectal cancer

**Project supervisor** (principal investigator of the laboratory/group)

Name: Lluís Espinosa

eMail: lespinosa@researchmar.net

Group name: Molecular Mechanisms of Cancer and Stemness

Institution: Hospital del Mar Research Institute (FIMIM)

Webpage of the group: <https://www.imim.es/programesrecerca/cancer/mmcsepinosa.html>

**Main grant associated with this project:** ColoStem, una nueva herramienta diagnóstica para el cáncer colorrectal

Principal investigator: Lluís Espinosa Blay

Agency: Instituto de Salud Carlos III

Reference/ years: DTS23/00005/2024-2026

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

In the recent years, different groups including our group have provided increasing evidence that CRC tumors with fetal features are particularly aggressive due to their increased metastatic capacity and resistance to chemotherapy. Consistent with this observation, we detected the presence of fetal traits in about 20% of tumors from 3 different public datasets associated with poor patient prognosis. Despite there are multiple signatures available for CRC stratification, there are none focused on detecting fetal traits and even less associated with treatment. ColoStem project aims to identify the group of high-risk patients inside the low-risk stage I and stage II/III patients as short-term objective, and even more so of assigning a specific treatment for them (midterm objective). To achieve this second objective, we will perform genomic and transcriptomic analysis of fetal-type tumors and patient-derived organoids (PDOs) and, based on the results obtained, we will design a medium throughput screening platform for the discovery of therapeutic targets, which we will then validate in vivo. The results obtained in this project could be used for the future design of companion diagnostic, which would represent an unparalleled advance in the fight against cancer.

## Call for project proposals

### Master in Biomedical Research practicum, 2025

#### Department of Medicine and life Sciences, Universitat Pompeu Fabra

**Project Title:** Pharmacological and neurophysiological profiling of novel Cav2.1 modulators to develop new therapies for Hemiplegic Migraine and related neurological disorders.

**Project supervisor** (principal investigator of the laboratory/group)

Name: José Manuel Fernández Fernández

eMail: jmanuel.fernandez@upf.edu

Group name: Laboratory of Molecular Physiology

Institution: Department of Medicine and Life Sciences, University Pompeu Fabra

Webpage of the group: <http://www.upf.edu/fisio/>

#### **Main grant associated with this project:**

Principal investigator: José Manuel Fernández Fernández

Agency: National Research Agency (AEI)/MCIN/FEDER

Reference/ years: PID2022-1365460B-I00 / From 09-2023 to 08-2026

**Brief summary of the project or current research lines of the group:** Human mutations in the *CACNA1A* gene (encoding the pore-forming  $\alpha_{1A}$  subunit of the voltage-gated Cav2.1 (P/Q-type) Ca<sup>2+</sup> channel) and N-hypoglycosylation induce Cav2.1 gain-of-function effects causing neuronal hyperexcitability that leads to multiple rare neurological disorders including Sporadic and Familial Hemiplegic Migraine (S/FHM), cerebellar pathologies such as congenital ataxia (CA), as well as stroke-like episodes and cerebellar syndrome associated to the most frequent form of Congenital Disorder of Glycosylation, Phosphomannomutase Deficiency (PMM2-CDG), a metabolic rare disease. Accordingly, there are pharmacological evidences suggesting that reduction of Cav2.1 activity (for example by medicinal plants) has therapeutic potential in the treatment of Hemiplegic Migraine (HM) and the relief of common migraine. At present, the Cav2.1-selective inhibitors available are peptide toxins. They are not suitable therapeutic tools due to both undesirable side effects and, as other peptides, limited utility for *in vivo* studies. We aim to identify novel direct and indirect inhibitory modulators of the Cav2.1 channel and to check *in vitro* and *in vivo* their capability of reversing the pathological Cav2.1 gain-of-function and subsequent neuronal hyperexcitability. For direct and selective Cav2.1 inhibition we will include in the study three novel compounds generated after chemical modifications from an existing state-dependent non-selective inhibitor of voltage-gated Ca<sup>2+</sup> channels, along with sixteen commercially available small molecules selected *in silico* on basis of their expected binding to a specific region of the Cav2.1 channel and that have already shown inhibitory action on Cav2.1 in a preliminary screening. Regarding indirect inhibitory modulation of Cav2.1, we will test the pharmacologically-induced increase in the N-glycosylation pathway using inhibitors of the phosphomannose isomerase (MPI). *In vitro* studies will be done using both, cells heterologously expressing wild-type and HM/CA mutant Cav2.1 channels, as well as networks of cortical neurons in primary cultures obtained from WT and FHM-knockin (KI) mice (the latter expressing the equivalent to human Cav2.1 R192Q mutant linked to FHM). For *in vivo* analysis, we will employ *Caenorhabditis elegans*. The genome of this nematode includes a single gene that codes for the Cav2  $\alpha$  subunit, called *unc-2*, which has a function equivalent to Cav2.1 in mammals. Besides, mutations in *unc-2* analogous to those in Cav2.1 linked to FHM have been reported to induce neuronal hyperexcitability at different levels in *C. elegans*, similarly to that observed in transgenic FHM mice.



**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Analysis of the dual role of Snail1 as transcriptional repressor and activator during epithelial-to-mesenchymal transition

**Project supervisor** (principal investigator of the laboratory/group)

Name: Antonio García de Herreros

eMail: agarcia@researchmar.net

Group name: "Epithelial to Mesenchymal Transition and Tumor Invasion"

Institution: Hospital del Mar Research Institute (IMIM)

Webpage of the group: <https://www.imim.es/programesrecerca/cancer/ubcm.html>

**Main grant associated with this project:**

Principal investigator: Antonio García de Herreros /Josep Baulida

Agency: Ministerio de Ciencia e Innovación -Agencia Estatal de Investigación (Retos de Investigación)

Reference/ years: PID2022-136968OB-I00; 1/09/2023 – 31/08/2026

**Brief summary of the project or current research lines of the group:**

Epithelial tumors, such as those generated in colon or breast, often generate metastatic foci that eventually compromise the patients' life. Current chemotherapies mostly reduce tumor burden, but their efficacy is too limited since many tumor cells acquire resistance to the treatment. Cancer cells activate epithelial-to-mesenchymal transition (EMT) to dissociate from the primary tumor and invade the neighbor tissues; moreover, EMT also impinges in other tumoral traits since it provides cancer stem cell characteristics and a higher resistance to chemotherapeutic drugs. EMT is controlled by a collection of transcriptional factor collectively known EMT-TFs; among them, Snail1 plays the most relevant role since it is stimulated by factors inducing an EMT before the rest of EMT-TFs and controls the expression of these.

The lab has maintained a long-standing interest on Snail1 and its mechanism of action during EMT. Results from our group, also corroborated by other labs, has demonstrated that in this process Snail1 initially acts as a transcriptional repressor blocking the transcription of CDH1 whereas at longer times is detected bound to activated gene's promoters. The precise role of Snail1 in these activated genes is unknown; the molecular basis for this switch has not been established either although it has been associated to a Snail1 post-translational modification (PTM) (acetylation). This project is addressed to analyze the mechanism promoting this switch and assess the role of Snail1 PTM on its ability to bind repressed (epithelial) and activated (mesenchymal) promoters, as well as in the interaction with known co-activators and co-repressors. For more information, please read Garcia de Herreros, A "Dual role of Snail1 as transcriptional repressor and activator", *Biochim Biophys Acta Rev Cancer* 2024, 1879, 189037.

# Call for project proposals

## Master in Biomedical Research practicum, 2025

### Department of Medicine and life Sciences, Universitat Pompeu Fabra

**Project Title:** Exploring the impact of synonymous mutations in protein assembly in a cancer context.

**Project supervisor** (principal investigator of the laboratory/group)

Name: Hector Garcia-Seisdedos

eMail: [hgsbmc@ibmb.csic.es](mailto:hgsbmc@ibmb.csic.es)

Group name: Structural Systems Biology

Institution: IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-structural-and-molecular-biology/structural-systems-biology/>

**Main grant associated with this project:**

Principal investigator: Hector Garcia Seisdedos

Agency: Ministerio de Ciencia e Innovacion

Reference/ years: CNS2023-144842/ 2024-2026

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Understanding the relationship between phenotype and genotype is central to biology. Despite advances in sequencing technology and the identification of millions of mutations, our understanding of the molecular effects of genetic variations remains limited. Mutations can impact protein structure, stability, solubility, function, and protein-protein interactions. We recently discovered that mutations may also trigger new self-interactions, leading to changes in protein localization and supramolecular assembly. However, while most studies focus on mutations that alter the amino acid sequence, much less is known about synonymous mutations, which do not change the protein sequence.

Recent research suggests that synonymous mutations can impact transcription, splicing, mRNA stability, translation efficiency and fidelity, and co-translational folding. We aim to determine whether synonymous mutations can trigger new self-assembly modes and changes in subcellular protein localization. The study will use yeast genetics, high-throughput microscopy, Mass Spectrometry, and RNA sequencing, combined with biophysical and structural techniques to achieve three aims:

- 1) genotype-phenotype characterization of synonymous mutations in symmetric proteins.
- 2) mechanistic insights on the phenotypic diversity of synonymous mutants.
- 3) assessing the self-interaction potential of synonymous mutations in cancer.

Overall, through this proposal we aim to will reveal how synonymous mutations can create new protein interactions and shed light on the potential mechanisms of the pathogenicity of this phenomenon.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Bioactive lipids in membrane traffic

**Project supervisor** (principal investigator of the laboratory/group)

Name: María Isabel Geli

eMail: mgfbmc@ibmb.csic.es

Group name: the membrane cytoskeleton interface in endocytosis

Institution: Institute for Molecular Biology of Barcelona (IBMB-CSIC)

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/the-endocytic-pathway-and-the-actin-cytoskeleton/>

**Main grant associated with this project:**

Principal investigator: María Isabel Geli

Agency: AEI

Reference/ years: PID2020-120053GB-I00 (2021-2024)

**Brief summary of the project or current research lines of the group**

We investigate how cellular membranes are shaped to generate tubular or vesicular transport intermediates, loaded with the appropriate cargo, which, after scission, travel through the crowded cytosol to reach the adequate acceptor compartment. This is a process essential to build a cell and hence, for life. As a model system, we use **endocytic uptake** from the plasma membrane in *S. cerevisiae* because its genetic amenability has allowed development of unique protocols to follow membrane budding with nanometric resolution. Further, the machinery involved in endocytosis is well conserved and mutation of the human homologues is linked to a myriad of rare diseases, which mostly develop as **neurological pathologies**. Therefore, through the analysis of these proteins in the context endocytosis, we learn about their function and regulation and we seed avenues for therapeutic intervention. Further, endocytosis is the obliged **entry gate for many pathogens** including SARS-Cov2 or HIV and thus, by understanding the mechanisms involved, we can more efficiently prevent pathogen invasion.

While most of the research on endocytic budding has focused on proteins, we have recently collected evidence supporting a very important role of **sterols and ceramides** (two lipid species particularly linked to neurological diseases) in membrane deformation. Using a multidisciplinary approach that combines **lipid-click chemistry, biophysics, live-cell fluorescence microscopy** of single endocytic events **and Time Resolved Electron Microscopy (TREM)**, we study the functional architecture of the sterol and ceramide metabolic networks and the mechanism whereby these lipids are directly transferred from the endoplasmic reticulum (ER) to endocytic sites in a non-vesicular manner. In addition, we investigate how an interplay between the membrane **phosphoinositides** and the cytosolic **inositol-polyphosphates** modulates the frequency and speed of endocytic events to facilitate cell adaptation to insults such as heat or osmotic shocks, by for example contributing to membrane healing, disposal of unfolded proteins or maintenance of membrane tension.

**Related literature**

Encinar del Dedo J, Fernández-Golbano I-M, Pastor, L., Meler P, Ferrer C, Rebollo E, Geli MI. Coupling of the sterol synthesis and transport machineries at ER-Endocytic Contact Sites. (2021) *J. Cell Biol.* 220(10):e202010016.

Encinar del Dedo J, Idrissi F-Z, Fernandez-Golbano I M, Garcia P, Rebollo E, Krzyzanowski M K, Grötsch H, Zimmermann T, Geli MI. ORP-mediated ER contact with endocytic sites initiates actin polymerization. (2017) *Dev. Cell.* 43:588-602.

Fernández-Golbano IM, Idrissi FZ, Giblin JP, Grosshans BL, Robles V, Grötsch H, Borrás MM and Geli MI. A cross-talk between PI(4,5)P<sub>2</sub> and CK2 modulates actin polymerization during endocytic uptake (2014) *Dev. Cell.* 30(6):746-58.

Idrissi FZ, Blasco A, Espinal A and Geli MI. "Ultrastructural dynamics of proteins involved in endocytic budding" (2012) *Proc Natl Acad Sci U S A.* 109: E2587-94.

## **Call for project proposals, master in Biomedical Research practicum, 2025, UPF**

**Project Title:** *Biochemistry and molecular Biology of proteins of biomedical interest.*

**Project supervisor** (besides the name, title and position, please include the relevant contact information: e-mail, phone postal address, and webpage).

F. Xavier Gomis-Rüth  
Proteolysis Laboratory  
Department of Structural Biology  
Barcelona Science Parc, Helix Building  
C/ Baldiri Reixac, 15-21  
08028 Barcelona  
Tel. 934020186 / Fax. 934034979 / e-mail. [xgrcri@ibmb.csic.es](mailto:xgrcri@ibmb.csic.es) /  
<https://www.ibmb.csic.es/proteolysis>

**Main grant associated with this project:** PID22-137827OB-001

Principal investigator: F. Xavier Gomis-Rüth

Agency: State Research Agency (AEI), Ministry of Science and Innovation

Reference/ years: 2023-2026

**Summary of project summary or current research lines** (less than 300 words).

The research group is centered on the study of proteolytic enzymes of biotechnological or biomedical interest, their protein inhibitors and regulation. Target molecules include proteins of mammals, protozoans and prokaryotes, as well as their interacting partners. The student would participate in the cloning, overexpression and purification of such protein targets under the supervision of experienced members of the lab. In addition—or alternatively—, she/he could participate in the biochemical, biophysical, functional, and structural characterization. The candidate should have strong marks and a very hard-working capacity, dedication and lab skills. In principle, this work could be continued within the frame of a Ph.D. thesis.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:**

**Project supervisor**

Name: Prof. Marcelo E. GUERIN

eMail: [mrccri@ibmb.csic.es](mailto:mrccri@ibmb.csic.es)

Group name: Structural Glycobiology

Institution: Institute of Molecular Biology of Barcelona (IBMB) – Parc Científic Barcelona (PCB)

Webpage of the group: <https://sites.google.com/site/querinlab>

**Main grant associated with this project:**

Principal investigator: Prof. Marcelo E. GUERIN

Agency: MICINN

Reference/ years: PID2022-138694OB-I00 (2023-2025)

**Brief summary of the project or current research lines of the group**

Red blood cell antigens play critical roles in blood transfusion since donor incompatibilities can be lethal. The most well-known and clinically relevant blood groups are ABO. Discovered in 1900 by Karl Landsteiner through agglutination tests, the antigens present in these groups are composed of specific oligosaccharides, A, B and H. The enzymatic modification of one sugar in the oligosaccharide could change the blood group of red blood cells into another one, being the conversion of A, B, and AB blood groups into O, the called universal blood, highly important for the universal blood supply of blood banks in emergency situations. Recipients with the rare total deficiency in H antigen, the Oh Bombay phenotype, can only be transfused with group Oh blood to avoid serious transfusion reactions. Our group recently identified an enzyme capable of converting universal O into rare Bombay type blood (Anso et al., Nat. Commun. (2023) 14:1765). The main goal of the project is to identify and engineer new enzymes to convert blood groups facilitating transfusion and organ transplantation.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** [Recording single cell dynamics with CRISPR barcoding.](#)

**Project supervisor** (principal investigator of the laboratory/group)

Name: Irene Hernando Herraéz

eMail: [ihbmc@ibmb.csic.es](mailto:ihbmc@ibmb.csic.es)

Group name: Epigenetic regulation and single cell dynamics.

Institution: Instituto Biología Molecular de Barcelona, IBMB – CSIC, located at PCB.

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/epigenetics-and-single-cell-dynamics/>

**Main grant associated with this project:**

Principal investigator: Irene Hernando Herraéz

Agency: Agencia Estatal de Investigación

Reference/ years: PID2022-137540NA-I00 - 2023/2027

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

The cells in our body divide constantly throughout life. As they divide, the transmission of epigenetic and transcriptional states establishes a form of cellular memory, where daughter cells retain very similar properties to their ancestors. This allows distinct gene expression patterns to persist in different cell types despite a common genotype. But why does this form of cellular memory change over time? Ageing is an extraordinary complex process, and our understanding is still very limited. My main interest is understanding how the accumulation of errors in the epigenome can lead to the degradation of cell identity, ultimately contributing to age-related dysfunction and disease such as cancer.

In this project, you will analyse a single-cell dataset from our novel cellular barcoding approach to investigate the fundamental basis of cellular heterogeneity within the neural stem cell pool. You will not only explore one of the greatest mysteries in biology but also acquire valuable transferable skills in cutting-edge techniques, including CRISPR barcoding, single-cell multiomics, and machine learning modelling.

## Call for project proposals

Master in Biomedical Research practicum, 2025

Department of Medicine and life Sciences, Universitat Pompeu Fabra

**Project Title:** Mitochondrial metabolism, reactive oxygen species and aging

### **Project supervisor:**

Elena Hidalgo

[elena.hidalgo@upf.edu](mailto:elena.hidalgo@upf.edu)

Oxidative Stress and Cell Cycle Group

Universitat Pompeu Fabra

[www.upf.edu/osccg](http://www.upf.edu/osccg)

### **Main grant associated with this project:**

Principal investigator: Elena Hidalgo

Agency: MICINN (Spain)

Reference/ years: 2022-2025

### **Brief summary of the project or current research lines of the group:**

Our group is interested in studying the components and molecular mechanisms controlling cellular fitness, in particular during aging. Thus, the master project proposal will be chosen among, but not exclusively, the following:

- (i) Study cellular processes linked to healthy aging; selection of fission yeast strains with altered lifespan, and characterization of the selected mutants, especially regarding mitochondrial homeostasis and ROS production.
- (ii) Use of genetically encoded H<sub>2</sub>O<sub>2</sub> biosensors to measure peroxides emanating from the mitochondria and controlling longevity
- (iii) Role of chromatin architecture and histone marks in transcription dynamics, using the stress response

We use the fission yeast ***Schizosaccharomyces pombe*** as a model system. To obtain more information about the laboratory and about our research interests, please consult our group's web page ([www.upf.edu/osccg](http://www.upf.edu/osccg)). Some recent publications include:

Vega et al. 2023. Nucleic Acids Res. (in press)  
Salat-Canela et al. 2023. TiCB 33:124.  
Vega et al. 2022. BMC Biol. 20:160.  
Salat-Canela et al. 2021. Cell Rep. 37: 109951.  
Corral-Ramos et al. 2021. Autophagy 23:1-16.  
Boronat et al. 2020. iScience 23:101725  
Cabrera et al. 2020. Cell Rep. 30:2430-2443  
Carmona et al. 2019. Nat. Commun. 10:4526.  
García-Santamarina et al. 2014. Nature Protocols 9:1131.  
Calvo, I.A. et al. 2013. Cell Reports 5:1413.  
Zuin, A. et al. 2010. EMBO J. 29:981.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Filming cell dynamics in live embryos to understand self-protection at the beginning of development.

**Project supervisor** (principal investigator of the laboratory/group)

Name: Esteban Hoijman

eMail: ehkbmc@ibmb.csic.es

Group name: Embryonic Cell Bioimaging

Institution: Institute of Molecular Biology of Barcelona (IBMB), CSIC

Webpage of the group: [www.embryobioimaging.com](http://www.embryobioimaging.com)

**Main grant associated with this project:**

Principal investigator: Esteban Hoijman

Agency: Spanish Ministry of Science and Innovation

Reference/ years: 2024-2027

**Brief summary of the project or current research lines of the group (please do not include pictures or logos)**

Errors in stem cells are considered the main cause of human preimplantation failures. In our group we study how early embryos can recover homeostasis by self-healing, before the formation of the immune system. We recently discovered an epithelial phagocytic program able to detect and remove defective stem cells (Nature 2021, [www.nature.com/articles/s41586-021-03200-3](http://www.nature.com/articles/s41586-021-03200-3)). Using quantitative imaging of live zebrafish embryos, we study single cells dynamics during tissue repair. In this project we want to elucidate the signals regulating this protective program of the embryo, with the long-term aim of improving embryo survival.



**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Deciphering the genome and epigenome in space and time to identify new leukemia therapeutic approaches.

**Project supervisor**

Name: Biola M. Javierre

eMail: [bmjavierre@carrerasresearch.org](mailto:bmjavierre@carrerasresearch.org)

Group name: 3D chromatin organization

Institution: Josep Carreras Leukaemia Research Institute (IJC)

Webpage of the group: <http://www.carrerasresearch.org/en/research/3d-chromatin-organization> & <https://www.javierrelab.com/> & <https://scholar.google.com/citations?user=8vZo2j8AAAAJ&hl=ca>

**Main grant associated with this project:** La Caixa Health Research 2023 and AECC Lab 2022

**Brief summary of the project or current research lines of the group**

Multiple blood malignancies (e.g., B-cell acute lymphoblastic leukemia) arise from the malignant transformation of undifferentiated B-cells, but the mechanisms underlying this transformation and disease relapse are not fully understood. While the causes are not completely understood, our preliminary data suggest that noncoding alterations, including mutations and epimutations (epi-/mutations), might play an important role by altering distal regulatory elements (DREs) (e.g., enhancers, super-enhancers, silencers, and super-silencers), ultimately triggering gene transcriptional deregulation. However, the cell-type specific repertoire of DREs that control each gene in each cell type and condition remains challenging since i) these elements are highly dynamic, ultimately driving specific gene transcription profiles; ii) these can regulate different genes in each cell condition. Besides, DREs cannot simply be assigned to the nearest gene because their target genes can be up to a few megabases away, often jumping over several intervening genes. However, DREs function through spatial proximity to the genes they regulate in vivo, and this proximity is facilitated by DNA loops. Thus, the three-dimensional (3D) genome organization, unique to each cell type, plays a fundamental role in regulating gene expression in both physiological and disease conditions, and its study can help us understand the contribution of non-coding alterations to disease.

To link DREs and their target genes, the Javierre Team has recently created a low-input Capture Hi-C (liChI-C) method (Tomas-Daza, et al; Nature Commun 2023). This method allows the systematic genome-wide identification of the promoter interactome, a term used to define the genomic regions, including enhancers and other DREs, in physical proximity with more than 22,000 promoters. Combining liChI-C and other omic techniques (e.g., CUT&RUN, ATAC-seq, RNA-seq), we have identified the repertoire of DREs and their target genes controlling normal B cell differentiation, determined non-coding epi-/mutations in B cell-associated leukemias that might be causative of malignant transformation and identified genes and pathways deregulated by these noncoding epi-/mutations. Now, our efforts focus on validating the key identified DREs and epi-/mutations using multiple techniques i) CRISPR/Cas9 genome editing to evaluate changes in target gene expression, ii) 3D DNA-FISH to measure distances between DREs and their target gene(s), and iii) other relevant molecular techniques such as UMI-4C, quantitative ChIP or quantification of nascent transcription; which will also be the main focus of future MSc students\*.

As a whole, our research aims to advance our comprehension of cancer etiopathology by unraveling the molecular intricacies underpinning leukemia development and relapse. Moreover, it holds the potential to pave the way for the implementation of pioneering therapeutic strategies, ultimately improving patient survival rates.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:**

**Project Title:** Unrevealing mechanism for p53-mediated tumour suppression

**Project supervisor** (principal investigator of the laboratory)

Name: Ana Janic

Mail: [ana.janic@upf.edu](mailto:ana.janic@upf.edu)

Group name: Cancer Biology

Institution: Department of Experimental and Health Sciences

Webpage of the group: <https://www.upf.edu/web/cancer-biology/>

**Main grant associated with this project:**

Principal investigator: Ana Janic

Agency: Plan Estatal

Reference/ years: 2022-2025

**Brief summary of the project or current research lines of the group (please do not include pictures or logos)**

The tumour suppressor gene p53 is mutated in half of the human cancers, and there is still extensive morbidity and mortality associated with cancers bearing p53 mutations. Given the difficulties in developing strategies for targeting wild-type or mutant p53, further understanding of its basic biology is required for successful clinical translation. Recent studies, including ours, have challenged the previously understood model of how the p53 gene is involved in tumour suppression. We found that several p53 activated target genes implicated in DNA repair have critical functions in suppressing blood cancer development. Based on this observation, we hypothesise that coordination of DNA damage repair is the most critical mechanism by which p53 suppresses tumour development. In line with this hypothesis our laboratories current research focuses on answering following questions i) how p53 controls a DNA repair–coordinated program to protect tumorigenesis; ii) how tissue specificity controls which p53-regulated DNA repair effectors are crucial for tumour suppression and iii) how we could use p53-dependent DNA repair signalling therapeutically to kill tumour cells.

Selected publications:

Abad, preprint, 2023

Brennan, CDD, 2023

Best et al., CDDis 2020

Janic et al., Nat Medicine 2018

Valente et al., Oncogene 2016

Valente et al., Cell Reports 2013

More info about our research at <https://www.upf.edu/web/cancer-biology>

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:**

**Project supervisor** (principal investigator of the laboratory/group)

Name: Gerardo Jiménez

eMail: gjcbmc@ibmb.csic.es

Group name: Gene expression and signaling

Institution: Institut de Biologia Molecular de Barcelona (CSIC), ICREA

Webpage of the group:

<https://www.ibmb.csic.es/en/department-of-cells-and-tissues/gene-expression-and-signaling/>

<https://www.icrea.cat/Web/ScientificStaff/gerardo-jimenez--canero-307>

**Main grant associated with this project:**

Principal investigator: Gerardo Jiménez

Agency: Ministerio de Ciencia e Innovación

Reference / years: PID2020-119248GB-I00 (2021-2024)

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

During animal development, the formation of tissues, organs and actually all body structures is under strict control by signaling pathways, transcription factors and effector genes. We are investigating these regulatory processes using the fruit fly *Drosophila*, a powerful experimental model for genetic and molecular analyses. We are particularly interested in the Ras-MAPK signaling pathway and in mechanisms of transcriptional repression, which we are studying by focusing on highly conserved molecules that are also implicated in human diseases, including cancer. The student will be exposed to these projects and the use of advanced technologies such as CRISPR-Cas9 and confocal microscopy. See our web pages for further details.

## **Call for project proposals**

### **Master in Biomedical Research practicum, 2025**

#### **Department of Medicine and life Sciences, Universitat Pompeu Fabra**

#### **Project Title: Function and specificity of human histone H1 variants in the organization and control of the genome.**

#### **Project supervisor** (principal investigator of the laboratory)

Name: Albert Jordan

Mail: ajvbmc@ibmb.csic.es

Group name: Chromatin regulation of human and viral gene expression

Institution: Institut de Biologia Molecular Barcelona IBMB-CSIC, Dept. Structural and Molecular Biology

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-structural-and-molecular-biology/chromatin-regulation-of-human-and-viral-gene-expression/#lab-presentation>

#### **Main grant associated with this project:**

Principal investigator: Albert Jordan

Agency: Ministerio de Ciencia e Innovación – Plan Nacional BFU

Reference/ years: PID2020-112783GB-C21 (2021-24)

#### **Brief summary of the project or current research lines of the group**

Histone H1 participates in the stabilization of DNA around the core histone octamer that constitutes the nucleosome, in the spacing between adjacent nucleosomes, in nucleosome mobility, and in further levels of chromatin compaction. As a consequence, H1 is seen as a chromatin structural protein that might be involved in DNA compaction, heterochromatin formation and stabilization, and in the regulation of nuclear processes such as transcription, replication, DNA repair, etc. Nonetheless, in mammals, histone H1 is not a single protein but an evolutionary diverse gene family that comprises up to seven members in somatic cells. Although considered for long time H1 variants to be redundant, we and others have described structural and functional differences between variants that include their distribution within the genome and nuclei, and diverse consequences upon depletion of particular H1 variants.

We have recently described that H1 variants show distinct abundances among different repetitive and transposable elements (TE), with an enrichment of H1 variants that are located within high GC regions (H1X and H1.4) at TE that have incorporated recently into the human genome along its evolution. These variants may be involved in the repression of these TE. In parallel, we have found that depletion of these variants causes transcription from cryptic promoters. On the other hand, variants enriched within low GC DNA (H1.2, H1.3, H1.5 and H1.0) are enriched at TE incorporated early in evolution, are preferentially located at peripheral heterochromatin and may have a role in maintaining heterochromatin identity and tethering to nuclear lamina. Our hypothesis is that histone H1 participates in the repression of such elements by participating in heterochromatin maintenance, and does this in a variant-specific manner. Besides, depletion of multiple H1 variants induces an interferon response in some cell types that could be used to induce immunoresponse against tumors.

We propose to study the involvement of histone H1 variants:

1. In the repression of cryptic transcription.
2. In the control of transposable elements.
3. In maintaining heterochromatin identity and the organization of nuclear compartments.
4. In preventing the viral mimicry phenomenon that may induce an interferon response in tumor cells, and its consequences in cancer progression and sensitivity to immunotherapy.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Design and Synthesis of New Tools for Photopharmacology

**Project supervisor:**

Name: Xavier Just Baringo

eMail: xavier.just@ub.edu

Group name: Just Baringo Group

Institution: Universitat de Barcelona

Webpage of the group: <http://justbaringochem.com>

**Main grant associated with this project:**

Principal investigator: Ben Bradshaw

Agency: Ministerio de Ciencia e Innovación

Reference/ years: PID2022-139257NB-I00 (2023-2026)

**Brief summary of the project or current research lines of the group:**

Photopharmacology has recently appeared as a unique way of turning drugs on and off using light. ***Switching drugs off after their therapeutic use renders them inactive to avoid off-target effects and the chances of resistance appearing as their accumulation does not increase their evolutionary pressure.***<sup>[1]</sup> Most approaches to photoswitchable drugs rely on the use of azobenzenes, which require the use of harmful UV light for their activation. However, these can be modified to cause a red-shift that allows ***activation with visible light that does not harm tissues and can penetrate deeper than shorter wavelengths.***

In our laboratory, we have recently developed the first photoswitchable antimicrobial peptides fully operated by visible light, allowing us to control the activity of the compounds with harmless illumination and enabling deactivation by simple exposure to sunlight. For instance, we found that linear analogues of tyrocidine A granted the best photocontrol of their antimicrobial activity, leading to ***compounds whose activity against Acinetobacter baumannii or Streptococcus pyogenes can be turned on and off at will.***<sup>[2]</sup> We are currently developing methods to access highly coveted photoswitches whose synthesis is very elusive. ***This new methods allow us to access state-of-the-art photoswitches for the development of new therapeutic agents.***<sup>[3]</sup>

The student will join a ***multidisciplinary project*** to work on ongoing research that covers the design, synthesis and characterisation of state-of-the-art photoswitches currently being developed in the group, which can be used as a ***platform to develop new biomedical tools.***

**References:**

- [1] Kobauri, P.; Dekker, F. J.; Szymanski, W.; Feringa, B. L. *Angew. Chem. Int. Ed.* **2023**, *62*, e202300681.
- [2] Just-Baringo, X.; Yeste-Vázquez, A.; Moreno-Morales, J.; Ballesté-Delpierre, C.; Vila, J.; Giralt, E. *Chem. Eur. J.* **2021**, *27*, 12987.
- [3] Ruiz-Soriano, A.; Lamelza, L.; Pizzamiglio, E.; Just-Baringo, X. *ChemRxiv* **2023**. DOI: 10.26434/chemrxiv-2023-dr111

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

Project Title:  
PROTECTING HEMATOPOIETIC STEM CELLS (HSC) FROM INFLAMMATORY STRESS

Project supervisor (principal investigator of the laboratory)

**Name: Cristina Lopez-Rodriguez**

**Mail: [cristina.lopez-rodriguez@upf.edu](mailto:cristina.lopez-rodriguez@upf.edu); [jose.aramburu@upf.edu](mailto:jose.aramburu@upf.edu)**

Group name: GENIMMUNE

Institution: Universitat Pompeu Fabra, Department of Medicine and Life Sciences

Webpage of the group: <https://www.upf.edu/web/genimmune>

**<https://www.upf.edu/web/cristina-lopez-rodriguez>**

Main grant associated with this project:

Principal investigator: Cristina Lopez-Rodriguez

Agency: ICREA Acadèmia 2023- 2027 ([https://www.upf.edu/recercaupf/-/asset\\_publisher/RVNxhLpxnc9g/content/four-upf-lecturers-scale-up-their-research-thanks-to-icrea-s-2022-academia-grants/10193](https://www.upf.edu/recercaupf/-/asset_publisher/RVNxhLpxnc9g/content/four-upf-lecturers-scale-up-their-research-thanks-to-icrea-s-2022-academia-grants/10193))

Brief summary of the project or current research lines of the group (please do not include pictures or logos and do not exceed this page):

We will address a major challenge related to stem cell fitness and aging, supported by the interdisciplinary expertise of our group, cutting-edge methodologies, experimental models of disease in mice, access to state-of-the-art core facilities, expert collaborators, and funding from national and international grants.

Hematopoietic reconstitution after stress, trauma or infection requires hematopoietic stem cells (HSCs) mobilization from quiescence, a process that makes them highly vulnerable to inflammatory signals that can exhaust the HSC pool. We have identified a mechanism that limits systemic production of type I interferons (IFN-I) by inflammatory cells in vivo, thus protecting HSCs from excess exogenous IFN-I while allowing for IFN-I protection against infection.

In this project, we will study what inflammatory signals target directly stem cells under chronic inflammatory stress signals that increase during aging. We will also analyse how stem cells deploy protective mechanisms that safeguard their viability, long-term progenitor potential and reconstitution function in response to chronic inflammation. Elucidating these mechanisms has interest in clinical hematology, and could advance knowledge on stem cell function in other systems.

Leading publications of the group:

Traveset and Cerdán Porqueras et al., 2023 under review

Lunazzi et al., 2021 Journal of Immunology

Huerga Encabo et al., 2020 Journal of Experimental Medicine

Aramburu and López-Rodríguez, 2019 Frontiers in Immunology

Buxadé et al., 2018 Journal of Experimental Medicine

Tellechea et al., 2018 Journal of Immunology

Berga-Bolaños et al., 2013 Proc Natl Acad Sci USA

Buxadé, Lunazzi and Minguillón et al., 2012 Journal of Experimental Medicine

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:**

Project supervisor (principal investigator of the laboratory/group)

Name: Marta Llimargas Casanova

eMail: mlcbmc@ibmb.csic.es

Group name: Mechanisms of morphogenesis and organogenesis

Institution: Institut de Biologia Molecular de Barcelona, IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/mechanisms-of-morphogenesis-and-organogenesis/>

**Main grant associated with this project:**

Principal investigator: Marta Llimargas Casanova

Agency: Ministerio de Ciencia e Innovación

Reference/ years: PID2021-126689NB-I00 - 2022-2025

**Brief summary of the project or current research lines of the group (please do not include pictures or logos and do not exceed this page):**

Intrinsic and extrinsic mechanisms regulating the morphogenesis of tubular epithelia

A fundamental question in biology is to understand how organs form during development, i.e., how individual cells organise and coordinate to generate a functional structure. Intrinsic genetic programs as well as interactions with the surrounding environment are known to instruct organogenesis.

We investigate organ formation using the tracheal system of the fruitfly *Drosophila melanogaster* as an amenable and tractable model. The tracheal system consists of a network of epithelial tubes that oxygenate the organism.

Our projects focus around three main questions:

- 1) Interactions and requirements of the extracellular matrices (apical and basal ECMs) with the tracheal epithelia during morphogenesis. This approach should provide new insights into how organs form in the context of a whole organism and may inform about tissue engineering oriented studies.
- 2) Remodelling and contribution of epithelial cell adhesion and cell polarity to tracheal formation. Cell adhesion and polarity are key features of epithelial tissues and this approach may help to understand tissue remodelling and homeostasis.
- 3) Unbiased analysis of tracheal morphogenesis. We identified in previous screens in the lab different factors with putative roles in tracheal morphogenesis. The comprehensive analysis of these factors is helping us to understand how to generate a mature and functional organ.

Our work lies at the interface of developmental biology, morphogenesis and cell biology, and requires advanced imaging techniques, as well as genetic, cell biology, molecular biology and biochemical techniques.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** The insect insulin receptors tangle

**Project supervisor** (principal investigator of the laboratory/group)

Name: José Luis Maestro

eMail: joseluis.maestro@ibe.upf-csic.es

Group name: Nutritional signals in insects

Institution: Institute of Evolutionary Biology (CSIC-UPF)

Webpage of the group: <http://www.biologiaevolutiva.org/jmaestro/>

**Main grant associated with this project:**

Principal investigator: José Luis Maestro and Xavier Bellés

Agency: Agencia estatal de investigación. Ministerio de ciencia e innovación.

Reference/ years: TED2021-130489B-I00/ December 2022-December 2024

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

The projects that we are carrying out in our laboratory are related to the study of the functions of the insulin pathway in insects, in particular in processes related to reproduction, development and growth. It has recently been described that insects have two insulin receptors (InR) and that in some evolutionary lineages, such as that of cockroaches and nearby groups, a third InR has been acquired. The present project is directed to the study of the functions of the different InRs in the German cockroach, *Blattella germanica*. This is a very common domestic pest with which our group have been working for decades and for which a large amount of information (including transcriptomes generated in the group or the sequenced genome produced also with our contribution), and tools (we have found that it is very sensitive to the interfering RNA (RNAi), which is extremely useful for functional genomics studies), are available. We will pay especial attention in the analysis of the function of the new InR that appeared in this evolutionary line and the possible evolutionary fate of this new molecule: non-functionalization, subfunctionalization or neofunctionalization.

Some of the methodologies that will be used are:

-RNAi methodologies, which include cloning, synthesis of dsRNA and treatment of the animals, etc.

-Expression studies in different treatments and physiological conditions: tissue dissections, RNA extraction, cDNA synthesis and real time-quantitative PCR.

-Microscopy techniques.



**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Stop codon readthrough: function, mechanism, and implications for biomedicine

**Project supervisor** (principal investigator of the laboratory/group)

Name: Marco Mariotti  
eMail: marco.mariotti@ub.edu  
Group name: Comparative Genomics and Recoding lab  
Institution: Universitat de Barcelona  
Webpage of the group: <https://www.mariottigenomicslab.com/>

**Main grant associated with this project:**

Principal investigator: Marco Mariotti  
Agency: Ministerio de Economía y Competitividad  
Reference/ years: RYC-2019-027746-I 2021/2026

**Brief summary of the project or current research lines of the group:**

Our lab studies the mechanisms of gene expression and protein synthesis, by combining bioinformatic and experimental approaches. We focus in particular on “recoding” events, i.e. programmed exceptions to the genetic code. In particular, stop codon readthrough is a form of recoding wherein specific stop codons support the insertion of an amino acid instead of causing protein synthesis termination. Notably, ~11% of human genetic diseases are caused by stop codons mutations, and so they may be potentially rescued by readthrough. Our lab studies natural mechanisms of stop codon readthrough and how to adapt them for personalized gene therapy.

The insertion of selenocysteine relies upon stop codon readthrough. This special amino acid is present in human and many other species, but it is not among the canonical 20 residues of the genetic code. Instead, it is encoded by the UGA codon, which is normally a stop, recoded to selenocysteine via a highly regulated mechanism involving local signals and a dedicated genetic machinery. Selenocysteine is found in the catalytic site of specialized enzymes known as selenoproteins, which have several essential functions, most notably in redox homeostasis. Many cancer types heavily rely on selenoprotein function, which is being explored as liability for therapy.

While readthrough is relatively rare in vertebrates, insects use it extensively to regulate gene expression: >5% of *Drosophila* genes undergo functional readthrough, and it appears important for the development of the nervous system. We are investigating the function and regulation of readthrough, and characterizing the sequence elements that drive this process.

The student may contribute to various projects in the lab, such as:

- Development of molecular tools to induce targeted stop codon readthrough for rescuing human pathogenic mutations
- Bioinformatic analysis of selenoprotein function and regulation in cancer
- Computational and/or experimental characterization of functions and evolution of stop codon readthrough in insects

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Epigenetics in neurodevelopment: role of PHF8

**Project supervisor** (principal investigator of the laboratory/group)

Name: Mária Martínez Balbás

eMail: mmbbmc@ibmb.csic.es

Group name: Signaling to chromatin

Institution: IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-structural-and-molecular-biology/molecular-signaling-and-chromatin/>

**Main grant associated with this project:**

Principal investigator: Mária Martínez Balbás

Agency: Ministerio de Ciencia e Innovación

Reference/ years: **PID2021-125862NB-I00. 09/2022-09/2025**

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

During neurogenesis a delicate balance between stability and plasticity is essential; in one sense, **stability** to allow the pool of neural progenitor cells to proliferate and propagate the cell identity to daughter cells; and **plasticity** to provide a window to modify this identity and allow differentiation. This balance is preserved by the coordinated action of epigenetic regulators and transcription factors that respond to developmental signals.

Our group is focused on understanding the role of **epigenetics** in gene transcription during **neurogenesis**. The final goal is to define the principles that control the activity of our genome to establish and maintain the cell identity during early neurogenesis. We study mechanisms of gene expression at different levels, from the action of epigenetic regulators, their interplay with developmental signals, to how the genomic loci are positioned in the 3D space within cell nuclei.

Moreover, epigenetic alterations are common in a wide spectrum of diseases, from developmental disorders to many types of cancers. With our studies we expect to pave the way towards deeper understanding on how deregulation of these epigenetic factors leads to disease focusing in intellectual disability, helping to develop therapeutic and diagnostic tools.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Apaf-1 role in the development of angioimmunoblastic T cell lymphoma

**Project supervisor** (principal investigator of the laboratory/group)

Name: Laura Mondragón Martínez

eMail: [lmondragon@carrerasresearch.org](mailto:lmondragon@carrerasresearch.org)

Group name: T cell lymphoma

Institution: Josep Carreras Leukaemia Research Institute

Webpage of the group: <https://www.carrerasresearch.org/en/research/t-cell-lymphomas%0D>

**Main grant associated with this project:**

Principal investigator: Laura Mondragón Martínez

Agency: State Investigation Agency

Reference/ years: PID2020-116049RA-I00 (09/2021-08/2024). 1 year prorogue asked and a new funding application has been submitted 01/2024 (response July 2024).

We also have structural fundings from our research centre (IJC303) valid up to April 2026 enough to cover students' expenses.

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Our line of research is focused on the molecular characterization and new therapies development in the context of angioimmunoblastic T cell lymphoma (AITL). This haematological malignancy affects people of 60 years of age and its often diagnosed in stages III/IV. There are no current specific therapies for its treatment and patients' overall survival 5 years after diagnosis is no more than 32%.

To achieve our objective, we make use of two animal models deficient for the proapoptotic protein Apaf-1. These animals spontaneously present at late stages of life a lymphoproliferative disorder with similar characteristics to human AITL. We regularly breed and aged both strains of mice and develop characterization studies regularly.

The student joining our lab will participate in the characterization process of these animals' models and will receive formation in animals handling and the development of flow cytometry, cell sorting, mRNA quantification assays. Depending on the date of incorporation, the student will also have the opportunity to participate in ex vivo and in vivo treatment validation experiments.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Genetic engineering of TGF- $\beta$  resistant NK cells for cancer immunotherapy

**Project supervisor** (principal investigator of the laboratory/group)

Name: Aura Muntasell

eMail: [amuntasell@researchmar.net](mailto:amuntasell@researchmar.net); Aura.Muntasell@uab.cat

Group name: Immunity and Infection

Institution: Hospital del Mar Research institute (IMIM)

Webpage of the group:

[https://www.imim.es/programesrecerca/rct/receptorscellularsnkiinfecci\\_.html](https://www.imim.es/programesrecerca/rct/receptorscellularsnkiinfecci_.html)

**Main grant associated with this project:**

Principal investigator: Aura Muntasell

Agency: Ministerio de Ciencia e Innovación, Instituto de Salud Carlos III

Reference/ years: 2023-2025

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Natural Killer (NK) cells are cytotoxic lymphocytes capable of recognizing and killing tumor cells in addition to orchestrate the development of tumor-specific adaptive immunity. Our previous studies support the contribution of tumor-infiltrating NK cells to the clinical efficacy of anti-HER2 antibody-based treatment in breast cancer patients. However, not all patients respond to treatment or eventually relapse and there is a need to develop novel strategies to enhance the activity of anti-HER2 therapies. In this regard, adoptive transfer of allogeneic NK cells is a growing area of immunotherapy discovery, since it is a strategy showing a great safety profile and off-the-shelf potential. Allogeneic NK cell adoptive transfer protocols showed clinical efficacy for some haematological cancers, yet their efficacy for the treatment of solid tumors remains limited, owing to immunosuppressive factors in the tumor microenvironment that lead to NK cell exclusion and functional impairment. In this project, we aim at building human NK cells resistant to TGF- $\beta$ , one of the main suppressive factors in solid tumors. The methodology includes the implementation of dual genetic engineering technology by CRISPR/Cas9 as well as the study of the engineered NK cell anti-tumor function in *in vitro* and *in vivo* preclinical HER2-positive breast cancer models.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Study of the role of mechanoreceptors in amyloid toxicity in Alzheimer's disease

**Project supervisor** (principal investigator of the laboratory/group)

Name: Francisco J. Muñoz López

eMail: paco.munoz@upf.edu

Group name: Aging Brain and Neurodegeneration

Institution: MELIS-Universitat Pompeu Fabra

Webpage of the group: <https://www.upf.edu/web/lmp/aging-and-neurodegeneration>

**Main grant associated with this project:**

Principal investigator: Francisco J. Muñoz

Agency: Spanish Science and Innovation Ministry

Reference/ years: AEI/PID2020-117691RB-I00/AEI/10.13039/501100011033

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Alzheimer's disease (AD) is due to the extracellular aggregation of the amyloid  $\beta$ -peptide ( $A\beta$ ) into oligomers and fibrils, which are synaptotoxic leading finally to cell death. There is not specific treatments that can cure, prevent or retard the disease.

The hypothesis proposes that oligomeric  $A\beta$  ( $oA\beta$ ) directly or indirectly (by oxidative stress) affect to the physiological function of mechanoreceptors that will have deleterious effects in the growth and maintenance of the synaptic spines, and a rise in intracellular calcium.

The objectives is the characterization of the effect of the  $oA\beta$  binding and/or the oxidative stress induced by  $oA\beta$  on TRPM7 and Piezo1 functions in the synaptic spines. The mechanical forces that drives dendrite growth are related to mechanoreception. In particular, spine growth and the maintenance of the functional shape of the spines are under the control of mechanoreceptors that regulates actin cytoskeleton. Therefore, we will study the effect of  $oA\beta$  on these receptors and how it will affect to synaptic plasticity and the existing spines, and we will also address the study of their role in the dysregulation of intracellular calcium.

The biological materials will be cell lines, neuronal primary cultures from mice and hiPSCs. Results will be validated in brain samples from APP<sup>swe</sup>/PSEN1<sup>dE9</sup> transgenic mice and AD patients and no demented individuals.

The methodology includes molecular biology of proteins and mRNA, gene overexpression and silencing, siRNAs, confocal microscopy, spectrofluorometry, calcium image, patch-clamp, flow cytometry and in silico studies.

The expected results of our project are the identification of new molecules involved in  $A\beta$  pathophysiology that would be considered as therapeutic targets for the treatment of AD.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Molecular signatures of synaptic aging

**Project supervisor** (principal investigator of the laboratory/group)

Name: Andrés Ozaita

eMail: andres.ozaita@upf.edu

Group name: Laboratory of Neuropharmacology

Institution: Universitat Pompeu Fabra

Webpage of the group: [www.upf.edu/neurophar](http://www.upf.edu/neurophar)

**Main grant associated with this project:**

Principal investigator: Andrés Ozaita

Agency: Spanish Agency for Research

Reference/ years: PID2021-123482OB-I00, 2022-2025

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Preventing cognitive decline and neurodegeneration is a relevant problem in biomedical research. Aging is associated to the development of cognitive impairment, which can be reduced by treatments that target the endogenous cannabinoid system. Such treatments have been found to improve synaptic plasticity and cognitive performance. We have found that synaptic transcriptional programs in the brain might be modulated by pathological and treatment conditions that result in different cognitive functionality. In this project we will explore such transcriptional programs and validate the main observations of the transcriptomic analysis. We will use *in vivo* (mouse models and behavioural analysis) and *in vitro* techniques (immunoblot, qPCR, immunofluorescence, confocal microscopy analysis, among others) to further explore the concept of synaptic aging associated to cognitive decline, as well as the synaptic impact of treatments that improve cognition.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Comprehensive analysis of RNA-seq data in murine fibroblast cells during *Trypanosoma cruzi* infection.

**Project supervisor** (principal investigator of the laboratory/group)

Name: Julio Alonso Padilla

eMail: [julio.a.padilla@isglobal.org](mailto:julio.a.padilla@isglobal.org)

Group name: Chagas Initiative

Institution: Barcelona Institute for Global Health (ISGlobal)

Webpage of the group: <https://www.isglobal.org/-/chagas>

**Main grant associated with this project:**

Principal investigator: Julio Alonso Padilla

Agency: RNAseq funded with an ISCIII FIS project (PI18/1054 - ChagasPATH).

Reference/ years: present

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

*Trypanosoma cruzi*, the causative agent of Chagas disease, is a protozoan parasite that infects millions of individuals worldwide, leading to a significant public health concern. Understanding the host-pathogen interactions during *T. cruzi* infection is crucial for developing effective therapeutic strategies. RNA sequencing (RNA-Seq) is a powerful tool to investigate the dynamic changes in gene expression that occur within host cells during infection. This proposed master's thesis project aims to perform a comprehensive analysis of RNA-Seq data from murine cells infected with *T. cruzi*.

The primary objectives of this project are as follows: a) pre-process RNA-Seq data from murine cells infected with *T. cruzi*; b) perform differential gene expression analysis to identify genes and pathways affected by the infection; c) investigate the regulatory networks and signalling pathways associated with host responses to *T. cruzi* infection; d) explore potential therapeutic targets for Chagas disease based on the identified gene signatures.

To successfully undertake this master's thesis project, the student should possess the following:

- R, as this programming language is essential for the analysis of RNA-Seq data. Experience in other programming languages such as Python will be valued.
- Basic knowledge of command line prompts and the ability to navigate and execute commands in a terminal environment is also important.
- An understanding of biological databases, such as GenBank, Ensembl, or NCBI, and experience in retrieving and interpreting biological information from these databases will be valuable.
- The student must own a working laptop or desktop computer. This project can be carried out remotely, with the possibility of working on-site if the student decides to.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Epigenetics and metabolism in the model organism *C. elegans*

**Project supervisor** (principal investigator of the laboratory/group)

Name: Marcos Francisco Pérez Browne

eMail: mpbbmc@ibmb.csic.es

Group name: Epigenetics and Metabolism

Institution: CSIC IBMB

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/epigenetics-and-metabolism/>

**Main grant associated with this project:**

Principal investigator: Marcos Francisco Pérez Browne

Agency: Mineco

Reference/ years: RYC2021-034496-I

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

We are interested in the direct metabolic roles of epigenetic processes, in particular histone methylation. While histone methylation is commonly considered to be an almost digital way of encoding information for the purpose of directing gene regulation, it is tied in to metabolic networks and occurs on a scale with the potential to alter the flux of metabolites through these networks. Histone methylation can thus be considered to be a metabolic process – and we believe this metabolic role can be important, for example in cancers.

We use the model organism *C. elegans* to investigate this process. *C. elegans* is a highly tractable and popular model organism – “Nature’s gift to science”, in the words of Nobel laureate Sydney Brenner. Due to the conservation of basic cellular processes in animals, we expect that the discoveries we make in *C. elegans* can often be translated to humans.

Additionally, we have an interest in the regulation of gene expression by transcription factors in *C. elegans*.



**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Understanding stress adaptation

**Project supervisor** (principal investigator of the laboratory/group)

Name: Francesc Posas

eMail: [francesc.posas@irbbarcelona.org](mailto:francesc.posas@irbbarcelona.org); [francesc.posas@upf.edu](mailto:francesc.posas@upf.edu)

Group name: Cell Signaling Group

Institution: IRB Barcelona

Webpage of the group: <https://www.irbbarcelona.org/en/research/cell-signaling>

**Main grant associated with this project:**

Principal investigator: Francesc Posas

Agency: Spanish Government

Reference/ years: ANALISIS DE LAS ACTIVIDADES REQUERIDAS PARA LA CORRECTA ADAPTACION Y SUPERVIVENCIA CELULAR AL ESTRES (PID2021-124723NB-C21). Ministerio de Ciencia, Innovación y Universidades. 2022-2024

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

We aim to unravel how cells detect and respond to environmental changes. We focus our studies on the characterisation of stress signal transduction pathways, especially those regulated by MAP kinases of the Hog1/p38 family, also known as the stress-activated MAP kinases (SAPKs). Proper adaptation to stress involves the modulation of several basic aspects of cell biology, among them the cell cycle and gene expression. Using *S. cerevisiae* budding yeast as a model organism, as well as higher eukaryotic cells, we are dissecting the molecular mechanisms underlying cell response to changes in the extracellular environment and characterising the adaptive responses required for cell survival. Based on our knowledge of signal transduction and using synthetic biology, we also seek to modify cell behaviour to reprogram cell response to specific inputs/stimuli.

*Research lines:*

- SAPK signalling: Using quantitative data in single cells and mathematical modelling, together with mutational analyses, we study the basic signalling properties of stress-responsive MAP pathways and how to alter them.
- SAPK targets: Using proteomics, biochemistry and genetics, our main goal is to identify new targets for SAPKs and thus widen our understanding of cellular adaptation to stress. This information is expected to facilitate the characterisation of the bases of adaptation in eukaryotes.
- Cell cycle control: SAPKs act in several phases of the cell cycle to allow prompt response to extracellular stimuli and the maintenance of cell integrity. We are uncovering the mechanisms by which Hog1 and p38 SAPKs regulate the cell cycle.
- Regulation of mRNA biogenesis: SAPKs control critical steps of mRNA biogenesis and are thus key regulators of stress-responsive gene expression. Our main aim is to determine the contribution of multiple factors to overall gene expression in response to stress. We are also using genome-wide CRISPR screening to identify essential genes for stress adaptation.

# Call for project proposals

## Master in Biomedical Research practicum, 2025

### Department of Medicine and life Sciences, Universitat Pompeu Fabra

**Project Title:** Gene Regulation in Stem Cells, Cell Differentiation & Cancer

**Project supervisor** (principal investigator of the laboratory/group)

Name: Antonio Postigo

eMail: [idibaps.postigo3@gmail.com](mailto:idibaps.postigo3@gmail.com)

Group name: Group of Gene Regulation in Stem Cells, Cell Plasticity, Differentiation, and Cancer

Institution: FCRB-Institute of Biomedical Research IDIBAPS / ICREA

Webpage of the group: <https://www.clinicbarcelona.org/idibaps/areas-y-programas/oncologia-y-hematologia/regulacion-genica-en-celulas-madre-la-plasticidad-y-diferenciacion-celular-y-el-cancer>

#### **Main grant associated with this project:**

Principal investigator: Antonio Postigo

Agency: AEI/Ministry of Science, Innovation and Universities

Reference/ years: PID2020-116338RB-100 / 2022-2024

#### **Brief summary of the project or current research lines of the group**

The group investigates the regulation of cellular and metabolic plasticity by the EMT factors ZEB1 & ZEB2 in health and disease. The ongoing research lines of the group are: a) Immunometabolism of macrophages in inflammation, b) Macrophage plasticity in tissue injury and regeneration, and c) Cell plasticity in the tumor microenvironment (tumor-associated macrophages). The selected candidates will have the opportunity to contribute to the project(s) aligning with their own background and expertise. Our research utilizes a diverse range of cutting-edge techniques, including unique transgenic mouse models and OMICs technologies such as scRNAseq, and spatial transcriptomics. Successful candidates will become part of a vibrant and international group. IDIBAPS is located in downtown Barcelona.

**Essential Requirements:** BSc (Grado) in Biology, Biotechnology or related biomedical discipline awarded after January 2021. Mark/grade above 8.7/10.0.

**Desirable Experience:** Although it is **not** required, candidates with the following criteria will received higher consideration: Previous lab experience, In possession (or in the process of getting it) of an official certificate to work with experimental mouse models. MSc in Immunology or with interest and knowledge in immunology

**ADDITIONAL INFORMATION & APPLICATIONS** Additional information about the group: <https://bit.ly/3zWD3DG> Applications should include: a) complete CV, b) the names & contact details of 2-3 PIs that have supervised the candidate's work. Inquiries and applications should be sent to: [idibaps.postigo3@gmail.com](mailto:idibaps.postigo3@gmail.com) indicating "**Candidate MSc UPF 2024**" on the subject of the email.

**SELECTED PUBLICATIONS** (as corresponding author since 2017): *Gut* 66:666. IF: 23.0 / *Gut* 68:2129. IF: 23.0 / *Nat Commun* 14:8316. IF: 16.6 / *Nat Commun* 14:7471. IF: 16.6 / *Nat Commun* 10:1364. IF: 16.6 / *Nat Commun* 9:2424. IF: 16.6 / *Sci Advances* 7:abd7455 IF: 14.1 / *Nuc Acids Res* 46:10697. IF: 14.1 / *EMBO J* 36:3336. IF: 11.6 / *PNAS* 120:e2301120120. IF: 11.1 / *Cell Rep* 42:113222. IF: 9.9 / *Cell Rep* 41:111452. IF: 9.9

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Regulation of the centrosome cycle in G2 and M

**Project supervisor** (principal investigator of the laboratory/group)

Name: Joan Roig Amorós

eMail: joan.roig@ibmb.csic.es

Group name: Cell Cycle and Signaling

Institution: Institut de Biologia Molecular de Barcelona IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/cell-cycle-and-signaling/>

**Main grant associated with this project:**

Principal investigator: Joan Roig

Agency: Plan Nacional de I+D, Ministerio de Ciencia e Innovación, Spain

Reference/ years: PID2021-127045NB-I00, 2022-2024

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Our group is interested in understanding how the G2 and M phases of the cell division cycle are regulated and executed. We focus our research on the roles of the signaling axis formed by the cyclin-dependent kinase CDK1, the polo-like kinase PLK1 and its downstream partners NEK9, NEK6 and NEK7, three related NIMA-family kinases that are activated at the centrosomes. Our previous work has shown how these kinases are central for the control of centrosome separation and maturation during mitotic entry (Bertran *et al.* (2011) *EMBO J.* **30**: 2634-2647; Sdelci *et al.* (2012) *Curr. Biol.* **22**: 1516-1523; Eibes *et al.* (2018) *Curr. Biol.* **28**: 121-129.e4; Gallisà-Suñé *et al.* (2023) *Nat. Commun.* **14**:1–20).

Using engineered animal models, genetically modified cell lines produced through CRISPR-Cas9 technology and RNAi, the project will involve characterizing novel functions of PLK1 and NEK9/NEK/7 in G2 and early M, and seek to understand how malfunction of these kinases and their substrates may result in abnormal chromosome segregation and the onset of aneuploidy. This will involve the use of a variety of techniques, including different types of microscopy. We will relate our observations with clinical data with the aim of assessing the possible involvement of the studied kinases and their substrates in the process of cell transformation and the apparition of cancer as well as the onset of developmental abnormalities.

We are additionally interested in understanding the roles of the NEKs in the organization and functioning of the primary cilia, a cellular structure organized by the centrosome that has important signaling functions in development and organogenesis as well as during tissue maintenance. A project tackling this could also be considered.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** The coordinated activity of beta-catenin at adherens junctions, centrosome, and nucleus defines the fate of developing neural tube stem cells

**Project supervisor** (principal investigator of the laboratory/group)

Name: Sebastian Pons

eMail: [spfbmc@ibmb.csic.es](mailto:spfbmc@ibmb.csic.es)

Group name: neural proliferation control

Institution: IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/neural-proliferation-control/>

**Main grant associated with this project:**

Principal investigator: Sebastian Pons

Agency: Agencia Estatal de Investigación

Reference/ years: PID2023-146672NB-I00 2025-2027

**Brief summary of the project or current research lines of the group**

Beta-catenin is a multifunctional protein with activity in adherens junctions (AJs), where it binds to N-cadherin, and acts as a cofactor in transcription mediated by TCFs. Oncogenic mutations in b-catenin are responsible, among other things, for Wnt subtype medulloblastomas, a childhood cerebellar tumor. These tumors exhibit slow growth and, among medulloblastomas, have a better prognosis. Moreover, the presence of oncogenic b-catenin can improve the prognosis of other medulloblastomas, suggesting that b-catenin displays oncogenic and tumor suppressor activities simultaneously. In previous studies, we demonstrated that oncogenic forms of b-catenin generate a characteristic lesion in the epithelium of the embryonic chicken neural tube (NT), referred to as intrusion. Within these intrusions, neural stem cells (NSCs), while remaining as progenitors, undergo restricted growth. We attributed the low proliferation to increased adhesion resulting from the significant accumulation of AJs proteins induced by oncogenic b-catenin.

Thus we propose the hypothesis that b-catenin, by recruiting actin binding proteins such as vinculin, plays a crucial role in regulating tension forces between apical AJs and the actin cytoskeleton in NSCs during NT development. Furthermore, we suggest that the aberrant growth induced by oncogenic forms of b-catenin is primarily attributed to its abnormal accumulation, not only within AJs but also at the centrosome.

So, we intend to demonstrate that hypothesis with the following objectives: Study how b-catenin and vinculin regulate tension between AJs of neighboring cells. Additionally, investigate the consequences of reducing the tension dependent on b-catenin and vinculin on the behavior of apical pole organelles such as the centrosome and the primary cilium. Study the impact of the abnormal accumulation of oncogenic forms of b-catenin at AJs and the centrosome on both inter- and intracellular forces. Investigate the mechanisms through which this accumulation disrupts the typical movements of the centrosome and determine whether these disruptions are linked to abnormalities in the growth of the neuroepithelium and alterations in the differentiation processes.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:**

**Project supervisor** (principal investigator of the laboratory/group)

Name: Murielle Saade

eMail: msabmc@ibmb.csic.es

Group name: Centrosome and Cilia in Normal and Pathological Neural Development

Institution: IBMB-CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-cells-and-tissues/a-new-vision-of-centrosome-cilia-in-normal-and-pathological-neural-development/>

**Main grant associated with this project:**

Principal investigator: Murielle Saade

Agency: Spanish Ministry of Science and Innovation

Reference/ years: CNS2023-144942 Proyectos de Generación de Conocimiento 2022 and PID2022-140285NB-I00

**Brief summary of the project or current research lines of the group**

Neurodevelopmental disorders (NDDs) impose a global burden, affecting an increasing number of individuals. While some causative genes have been identified, understanding the human-specific mechanisms involved in these disorders remains limited. Traditional gene-driven approaches for modeling brain diseases have failed to capture the diverse and convergent mechanisms at play. Centrosomes and cilia act as intermediaries between environmental and intrinsic signals, regulating cellular behavior. Mutations or dosage variations disrupting their function have been linked to brain formation deficits, highlighting their importance, yet their precise contributions remain largely unknown. This research aims to investigate the hypothesis that the centrosome/cilia axis is crucial for brain development and serves as a hub for human-specific mechanisms disrupted in NDDs. State-of-the-art experimental designs will be employed, utilizing various model systems and omic approaches. We will explore i) the cellular functions of centrosomes/cilia in processes such as progenitor proliferation, positioning, and neuronal migration in both developing chick embryos *in vivo* and human *in vitro* systems and ii) the decoding of convergent and divergent pathways involved in these processes. By integrating expertise in ciliated cells' involvement in mammalian neurogenesis and NDD modeling using animal and human-specific models, our lab aims to significantly contribute to the limited knowledge of NDD occurrence. This research addresses fundamental questions regarding the diversity and convergence of gene function in development and disease manifestation. It has the potential to uncover novel pharmacological targets, facilitate personalized medicine, and ultimately improve the lives of individuals affected by brain disorders through targeted cilia-based therapies. The anticipated outcome of this study instills optimism for advancements in understanding and treating NDDs.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Exosomes as mediators of clock-dependent interorgan crosstalk

**Project supervisor** (principal investigator of the laboratory/group)

Name: Jacob Smith

Email: jacob.g.smith88@gmail.com

Group name: Circadian rhythms and intercellular crosstalk

Institution: Universidad de Barcelona

Webpage: <https://sites.google.com/view/smithlab-ibub>

**Main grant associated with this project:**

Principal investigator: Jacob Smith

Agency: AEI

Reference/ years: RYC2022-035133-I / 5 years

**Brief summary of the project or current research lines of the group**

The key questions addressed by the Smith Lab are 1. How is biology coordinated in space and time? 2. What happens when this coordination breaks down? 3. Can we restore synchrony to prevent or cure disease?

A key initial focus of the lab is on the role of endogenous timekeepers (circadian clocks) in signalling between organs. Specifically, we are considering communication between clocks in skeletal muscle and liver, two tissues integral for systemic energy metabolism. This builds upon our previous work which showed the muscle clock communicates with the liver via (as-of-yet) unidentified blood-borne signals (Greco\*, Koronowski\*, Smith et al., Science Advances 2021). New data suggests the same occurs in the opposite direction (Smith\*, Koronowski\* et al., Cell Reports 2023; Smith et al., in preparation). We aim to identify factors that enhance circadian function in liver and muscle, with the long-term goal of identifying new therapies against devastating diseases that have so far been difficult or impossible to treat, such as metabolic syndrome, hepatocellular carcinoma and muscle wasting disorders.

Project:

Based on exciting preliminary data, we will explore the hypothesis that exosomes are mediators of clock-dependent interorgan crosstalk. As these nanosized extracellular vesicles can pass between cells and tissues, we will explore whether exosomes are capable of conveying timing information and/or metabolic status between muscle and liver.

Candidate information:

Applications are welcomed from candidates from diverse fields, skillsets and backgrounds. Key attributes are enthusiasm, a positive attitude to problem solving and teamwork. During your project, you will be coached to develop scientific and critical thinking skills, improve written and presentation abilities, and will gain progressive independence throughout. Support for grant writing and applications is provided for highly motivated students wishing to apply for PhD fellowships. Happy to chat informally (email/zoom) to discuss the project more and answer any questions.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Circadian control of cell-cell communication

**Project supervisor** (principal investigator of the laboratory/group)

Name: Jacob Smith

Email: jacob.g.smith88@gmail.com

Group name: Circadian rhythms and intercellular crosstalk

Institution: Universidad de Barcelona

Webpage: <https://sites.google.com/view/smithlab-ibub>

**Main grant associated with this project:**

Principal investigator: Jacob Smith

Agency: AEI

Reference/ years: RYC2022-035133-I / 5 years

**Brief summary of the project or current research lines of the group**

The main questions we are addressing are: 1. How is biology coordinated in space and time? 2. What happens when this coordination breaks down? 3. Can we restore synchrony to prevent or cure disease?

A key unanswered question in the circadian field is how biological rhythms, and their associated processes, are coordinated within a tissue. The complexity of this challenge is underscored by the diverse cell types present within a given organ/tissue, an addition to the changing cellular milieu under different conditions such as inflammation during disease and aging. Recent published studies in liver have revealed that cellular clocks in one cell type (in this case hepatocytes), can affect rhythms in other cell types within the same tissue (Guan et al., Science, 2020). Our exciting preliminary data suggest for the first time that this also occurs between different cell types in skeletal muscle. Here, we will define this further and ask whether paracrine (cell-cell) signalling is involved in this process.

Project:

In collaboration with Pascal Maire (Institute Cochin, Paris), Irene Hernandez (IBMB, Barcelona), and Valentina Sica (UPF), we are using cell-cell ligand receptor analyses to identify novel candidate paracrine factors (using published and our unpublished datasets). We will study these new molecules and compare with genetic clock disruption *in vitro* in coculture experiments, to test the capacity of these novel clock-regulated factors to mediate intercellular crosstalk.

Candidate information:

Applications are welcomed from candidates from diverse fields, skillsets and backgrounds. Key attributes are enthusiasm, a positive attitude to problem solving, and working well within a team. During your project, you will be coached to develop scientific and critical thinking skills, improve written and presentation abilities, and will gain progressive independence throughout. Support for grant writing and applications is provided for highly motivated students wishing to apply for PhD fellowships. Happy to chat informally (email/zoom) to discuss the project more and answer any questions.

## Call for project proposals

Master in Biomedical Research practicum, 2025

Department of Medicine and life Sciences, Universitat Pompeu Fabra

**Project Title:** Effect of plastic PE on mitochondrial activity

**Project supervisor** (principal investigator of the laboratory/group)

Name: Maria Solà

eMail: maria.sola@ibmb.csic.es

Group name: MitoLab

Institution: Institute of Molecular Biology of Barcelona - CSIC

Webpage of the group: <https://www.ibmb.csic.es/en/department-of-structural-and-molecular-biology/mitochondrial-macromolecules/#lab-presentation>

### **Main grant associated with this project:**

Principal investigator: Maria Solà

Agency: **R&D project, Societal Challenges State Plan, Life Sciences Subplan**, Ministry of Science, Innovation and Universities. "Structural analysis of proteins essential for mitochondria and pathogen viability - 2".

Reference/ years: Ref: PID2021-129038NB-I00. 1.9.2022-31.08.2025.

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Most part of the ATP, the molecule that stores the energy in a chemical form, is produced in mitochondria, the power plants of our body. Other essential functions also take place in these organelles, such as the last steps of catabolism, nucleotide synthesis, fatty acid and cholesterol synthesis, etc., it induces the major mode of apoptosis, and maintains cellular proteostasis. Dysfunctional mitochondria lose membrane potential and integrity, cause oxidative stress in the cell and irreversible cell damage, and eventually triggers cell apoptosis by the caspase pathway. Alterations of mitochondrial activity is associated to several diseases, including age-related diseases such as neurological disorders. On another line, it is well known that plastic derived from fossil fuel (gas, oil) is part of the vast majority of human activities (construction, machinery, daily life supplies, etc). Plastic consists of polymerization of monomers that, in the presence of additives (plasticizers, stabilizers), acquires specific properties of great durability, resistance, hardness, color, flexibility, transparency, etc. This is, also, the problem: plastic is currently a global environmental challenge as it accumulates in landfills, bio-accumulates in marine animals (cetaceans, fish, and birds), and it also threatens human health by accumulating in all organs.

We aim at understanding the effects of polyethylene (PE) toxicity in mammalian mitochondria. It has been shown that nano-PET (polyethylene terephthalate), present in the air, is internalized by pulmonary cells and alters mitochondrial activity. It has already been shown the adverse effect of PVC (polyvinyl chloride), polystyrene, and polypropylene on mitochondria. In this respect, the effects of PE are still to be characterized. In collaboration with the mitochondria experts, the candidate will learn to measure mitochondrial membrane (de)polarization, respiration levels, mitochondrial integrity (fusion, fission) by employing fluorescent tags and light microscopy, mtDNA copy number determination by PCR, ATP production levels, etc, of cells exposed to micro- and nano-plastics of PE versus control samples.



**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Somatic and germinal landscape of therapy related myeloid neoplasms

**Project supervisor** (principal investigator of the laboratory/group)

Name: FRANCESC SOLE

eMail: fsole@carrerasresearch.org

Group name: MDS GROUP

Institution: INSTITUT DE RECERCA CONTRA LA LEUCEMIA JOSEP CARRERAS

Webpage of the group: <https://www.carrerasresearch.org/en/research/myelodysplastic-syndromes>

**Main grant associated with this project:**

Principal investigator: FRANCESC SOLE

Agency: ISCIII. FIS PROJECT

Reference/ years: 2024-2026

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Myelodysplastic syndromes or myelodysplastic neoplasms (abbreviated MDS) could be de novo or secondary to a previous cancer. It is currently estimated that 3 out of 5 people will develop cancer. Due to advances in medicine, more cancer patients are being cured. However, cancer patients treated with chemotherapy and/or radiation for the primary tumor have an increased risk of developing therapy-related myeloid neoplasms (TRMN), which are very aggressive clonal hematologic neoplasms with poor prognosis that affect the myeloid lineage.

Likewise, genetic predisposition might be studied also for primary MDS, mainly in those cases where there's an early age of onset of the disease or family aggregation of myeloid neoplasms. These patients comprise the entity of germline MDS (gMDS), which is determined by the presence of germline mutations in genes that predispose to the development of MDS.

Thus, a proposed series of 50 TRMN patients will be studied at the somatic and germline level. Moreover, we will evaluate the prevalence of genetic predisposition to MDS (gMDS) in MDS patients, with the aim to explore the pathogenic mechanisms that trigger different germline dependent MDS scenarios and provide an accurate and differential diagnosis. Transcriptome studies and accurate (cyto)genetic analysis using WES and Optical Genome Mapping in bone marrow samples from TRMN and gMDS patients, will allow the determination of key molecular driver pathways.

In this project a proposed series of TRMN patients will be studied at the somatic and germline level to unravel the pathogenic mechanisms involved in the development of this disease, and to evaluate the possible relationship between TRMN progression and the previous presence of CHIP or germline predisposition. This would enable the development of adequate methods for early and differential diagnosis of the different TRMN scenarios, and guide therapies in the clinical practice of primary tumors in patients at risk of developing a TRMN. Moreover, the prevalence of genetic predisposition to MDS (gMDS) in MDS patients younger than 50 years old will also be evaluated, with the aim to explore the pathogenic mechanisms that trigger different germline dependent MDS scenarios and provide an accurate and differential diagnosis. Transcriptome studies and accurate (cyto)genetic analysis by whole exome sequencing (WES) and Optical Genome Mapping in bone marrow samples from TRMN and gMDS patients will be carried out to determine the key molecular driver pathways involved in the tumorigenesis.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:**

**Project supervisor** (principal investigator of the laboratory/group)

Name: Gregoire Stik

eMail: gstik@carreras.research.org

Group name: Nuclear Architecture in Leukemia

Institution: Josep Carreras Leukemia Research Institute

Webpage of the group: <https://www.carrerasresearch.org/en/directory/gregoire-stik-402>

**Main grant associated with this project:**

Principal investigator: Gregoire Stik

Agency: Plan Estatal de Investigación Científica y Técnica y de Innovación

Reference/ years: call 2022 – PID2022-140859OA-I00

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

Recent advances in molecular techniques to map the 3D conformation of chromosomes have enhanced our understanding of how genome architecture affects function in different tissues and diseases. It is now **well-established that chromatin is highly organized in 3D**, enabling **precise regulation of gene expression**. Cancer cells, particularly leukemic cells, have been shown to exhibit alterations in their genome organization also impact gene regulation.

Leukemia is one of the **first cause of death in children**, with B-cell Acute Lymphoblastic Leukemia (B-ALL) being the most common subtype. One of the most prevalent forms of B-ALL is caused by a chromosomal translocation t(1;19), which results in the formation of the **E2A-PBX1 chimeric transcription factor**. The role of E2A-PBX1 in the **molecular mechanisms underlying the 3D genome organization in B-ALL are still unknown** and its impact on gene expression needs further investigation. Our recent research suggests that this chimeric **protein E2A-PBX1 has phase separation (PS) properties** that could potentially play a **role in the formation of a 3D genome structures**, in the deregulation of the gene expression and in the **leukemogenesis**.

Therefore, we propose to combine and integrate **multi-omics experimental and computational approaches** encompassing state-of-the-art technologies (e.g. **Hi-C, CRISPR/Cas9 genome editing, degron system**, optogenetic) to elucidate the role of the chimeric protein E2A-PBX1 on 3D chromatin organization and B-ALL malignancy. First, we will shed the light on **the biomolecular properties of the chimeric protein** and on its precise subcellular localization. Second, I will decipher the **direct role of E2A-PBX1 on 3D genome organization**, gene expression and epigenetic landscape. Finally, using primary cells, we will link the biomolecular properties of E2A-PBX1 to its role on genome organization and on **leukemogenesis**.

Understanding how **translocation can affect biochemical properties** of protein and **alter the genome organization** and the gene expression will offer potential **new biomedical applications** for the treatment of haematological malignancies.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Physiological correlates of synthetic learning in the hippocampus

**Project supervisor** (principal investigator of the laboratory/group)

Name: Manuel Valero García

eMail: [mvalero@researchmar.net](mailto:mvalero@researchmar.net), [valegarman@gmail.com](mailto:valegarman@gmail.com)

Group name: Neural Computation laboratory

Institution: Hospital del Mar Research Institute

Webpage of the group: <https://valeroneuroscience.com/>

**Main grant associated with this project:**

Principal investigator: Manuel Valero García

Agency: Ministerio de Ciencia e Innovación | Ministerio de Ciencia e Innovación

Reference/ years: RYC2021-032543-I / 2023-2027 | PID2022-143259OA-I00 / 2024 - 2028

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

The main goal of our laboratory is to provide a deep understanding of how groups of neurons interact and tune their connections to generate neural representations of the world, and ultimately, learning and memories. We seek to reveal the neuronal mechanisms underlying cognition and how imbalances in the network lead to pathological conditions. To address these questions we are implementing and developing electrophysiological, optogenetic and computations tools for accessing the activity of neurons at a large scale in animal models and humans solving memory tasks. Our main research lines are:

- Principles of neuronal connectivity. Inhibitory neurons present the highest degree of neuronal diversity in the cortex. Developing tools to identify the distinct inhibitory neuron types is a bottle neck to be able to decipher their role in circuit dynamics (rhythms) and network computation (algorithms). Here, we are manipulating several neuronal types of the hippocampal area CA1 by optogenetic approaches (Valero et al, 2021 Nat Neurosci) and inferring their connectivity by high-resolution optogenetics.
- Synaptic dynamics during learning. We have developed a method to reveal the subthreshold dynamics of large numbers of neurons based on high-resolution optogenetic stimulation (Valero et al, 2022, Science; Valero et al, 2022 Cell Reports). Using this technology, we are monitoring the changes in the synaptic integration of single neurons during learning events in normal and diverse neuropathological conditions, including epilepsy and Alzheimer's disease. With this technology, we expect to provide a mechanistic link between synaptic changes and learning.
- Artificial neural operations. In the brain, information from the environment and its relationship with the subject is represented by the activation of different neuronal assemblies. Little is known about how these assemblies are formed, and the operations they perform. In this project we are combining pattern-detection methods with high resolution optogenetic circuit interventions to experimentally recreate and manipulate neuronal assemblies operations in animal models solving cognitive tasks. Knowing the basic rules of brain assemblies processing will help identify the essential processes underlying cognition and its alterations upon several pathologies.

**Call for project proposals**  
**Master in Biomedical Research practicum, 2025**  
**Department of Medicine and life Sciences, Universitat Pompeu Fabra**

**Project Title:** Deciphering the role of circadian disruption along the microbiota-gut-brain axis in cancer and ageing

**Project supervisor** (principal investigator of the laboratory/group)

Name: Patrick-Simon Welz

eMail: pwelz@researchmar.net

Group name: Intercellular Communication in Cancer and Ageing

Institution: Hospital del Mar Research Institute

Webpage of the group:

[https://www.imim.es/programesrecerca/cancer/en\\_intercellular\\_communication.html](https://www.imim.es/programesrecerca/cancer/en_intercellular_communication.html)

**Main grant associated with this project:**

Principal investigator: Patrick-Simon Welz

Agency: Agencia Estatal de Investigación

Reference/ years: PID2020-113317RA-I00/ 2021-2025

**Brief summary of the project or current research lines of the group** (please do not include pictures or logos and do not exceed this page):

The fascinating symbiotic relationship between the intestinal microbiota and the host plays an important role in maintaining health. In line with that, altered communication in the microbiota – gut – brain axis contributes to the development of a wide range of diseases, such as cancer and dementias. What's more, disruption of circadian rhythmicity is a hallmark of tumour development, as well as of many ageing-related pathologies. How deteriorating diurnal signalling in the microbiota – gut – brain axis might impact on disease pathogenesis is only poorly understood.

In our laboratory, we are studying how disturbances in the daily communication along the microbiota – gut – brain axis impact on the development of colorectal cancer and Alzheimer's disease. We combine multi-omics approaches, including microbiome sequencing, metabolomics and transcriptomics, with behavioural studies, histopathology (e.g. immunohistochemistry, microscopy), and molecular biology analyses (e.g. western blot, immunoprecipitation, PCRs, cell sorting), to decipher mechanisms of disease *in vivo* and *in vitro* on a holistic level. Thus, this interdisciplinary project allows to obtain hands-on experience in various state-of-the-art techniques and to gain insights into the processes underlying some of the most devastating and prevalent human diseases.