ANNUAL REPORT 2022

Josep Carreras
LEUKAEMIA
Research Institute

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ÍNDEX

FOREWORD

Dr. Manel Esteller
Director

I am delighted to share with you the many achievements and successes of our Institute in this 2022.

With a looking-forward approach, the Josep Carreras Leukaemia Research Institute has undergone an exhaustive analysis of its priorities and goals, which has been translated in the elaboration of the 2023-2027 Strategic Plan. Research and Innovation underpin this Strategic Plan, which allows us to clearly see our opportunities, gaps, and intentions as an institution and will guide us through the next 5 years.

Among the many highlights in 2022 were the ongoing IJC's international leadership consolidation, which is reflected in many alliances and collaborations with key global players of the research sector. This internationalization strategy has led the Institute to participate in major initiatives, such as the Cancer Proteome Project, promoted by the National Cancer Institute (NCI) in the United States.

Last year also saw IJC leading the way in technology and innovation. Our drive for the Single Cell technology has led us to become the first center of Excellence for Tapestri Platform in Europe, named by the company Mission Bio, Inc; and also the first reference center in the country for the Visum Spatial Gene Expression technology, from the company 10x Genomics. In that way, the Institute confirms its position as a leader and pioneer in the use of this technology.

In 2022, we marked a milestone in IJC's growth. We stabilized 42 structural positions, becoming one of the fastest growing research centers in Catalonia. We have also enhanced our partnerships with other research institutions, academia, industry, and local and national authorities. This was reflected in the visits to our facilities of the Minister of Health and the Minister of Science and Innovation of the Spanish Government. The fight against cancer is a national necessity, and that is why the support of the authorities and society is crucial.

When I reflect on this past year, I am struck by all the work, progress and efforts done by all the staff of our Institute. It is thanks to their continued commitment and dedication to IJC's mission that we continue to be at the very forefront of research in cancer, leukemia and other malignant blood diseases. I highly encourage you to browse through this Annual Report to see their remarkable accomplishments.

Sincerely,

ABOUT US

WHO WE ARE

The Josep Carreras Leukaemia Research Institute is a non-profit research institute based in Badalona (Barcelona), **dedicated to biomedical research and personalized medicine in leukemia and other malignant blood diseases.** It conducts excellent research into the basic, epidemiological, preventive, clinical and translational aspects of leukemia and other hematological malignancies.

The Josep Carreras Leukaemia Research Institute, **directed by Dr. Manel Esteller, was launched in 2010 by the Josep Carreras Foundation**, together with the Catalan government, **and is the first European research center devoted exclusively to leukemia and other malignant blood diseases.**

Research efforts are imperative to provide **patients with high-quality healthcare**, and our specialists combine their extensive knowledge to focus on patients' needs. **Our aim is to understand the origins and development of leukemia and other malignant hematological pathologies with a view to their prevention, and our efforts are directed towards identifying new therapeutic targets and developing effective treatments** with fewer side effects through cutting-edge research.

Given our efficient governance, the Josep Carreras Leukaemia Research Institute is a Catalan Research Centre of Excellence and forms part of the Catalan government's current research center network. It is also accredited by the Spanish Ministry of Health as a Health Research Centre of Excellence and by the Spanish Association Against Cancer. The Institute also holds the HR Excellence in Research Award from the European Commission.

The ultimate goal of our interdisciplinary **team is to ensure that leukemia is a curable disease in all cases**, and we won't stop until we have achieved this.

The Josep Carreras Institute is a collaborative hub for basic and translational researchers who work together on the fundamental biological and clinical aspects of leukemia at our state-of-the-art facilities, which provide an excellent work environment and serve as a magnet for outstanding researchers from all over the world.

It is home to 39 research groups and an increasing number of associated clinicians from five independent, coordinated scientific locations: Hospital Clínic, Sant Pau, Can Ruti, Mar and Trueta. Our laboratories on those clinical locations allow us to collaborate closely with clinicians from the five associated hospitals: Hospital Clínic, Hospital de Sant Pau, Hospital Germans Trias i Pujol, Hospital del Mar and Dr. Josep Trueta Hospital.

ABOUT US

MISSION, VISION AND VALUES

MISSION

The IJC's mission is to carry out research into the epidemiological, preventive, clinical, traslational, and basic aspects of cancer, with special emphasis on leukaemia and other malignant blood diseases, with the aim of finding a cure for these diseases through innovation.

VISION

The vision of the Josep Carreras Leukaemia Research Institute is to be a world-class reference and excellent research center that contributes to the improvement of results, and the cure of patients affected by leukemia and other malignant hemopathies, through innovation, sustainability, social responsibility, talent, and professional experience.

VALUES

- Scientific and Social Ethic
- Interdisciplinarity
- Equality and diversity
- Creativity
- Perseverance and continuous improvement
- Sustainability

ABOUT US

GOVERNING BODIES

The highest governing body of the Institute is the Board of Trustees, which is represented by the Josep Carreras Foundation, the Catalan government's Ministry of Research and University, the Catalan government's Ministry of Health, the Autonomous University of Barcelona (UAB), the University of Barcelona (UB), the Badalona City Council, the General Directorate for Health Research and Planning, the Catalan Institute of Oncology, the Northern Metropolitan Territorial Area Administration, the Catalan Foundation for Research and Innovation, Hospital Clínic / the August Pi i Sunyer Biomedical Research Institute (IDIBAPS), the UB Hospital Coordination Committee and the Research Centers of Catalonia Institution Foundation (iCERCA).

BOARD OF TRUSTEES

PRESIDENT

Mr. Josep Carreras i Coll

FIRST VICE-PRESIDENT

- Minister for Health, Generalitat, Government of Catalonia

SECOND VICE-PRESIDENT

- Minister for Research and University, Generalitat, Government of Catalonia

MEMBERS

- Mayor of Badalona
- General Director of Research of the Ministry of Business and Knowledge for the Generalitat, Government of Catalonia
- General Director of Health Planning, Generalitat, Government of Catalonia
- General Sub-Director for Research and Innovation, Ministry of Health, Generalitat, Government of Catalonia
- Managing Director, Catalan Institute of Oncology (ICO)
- Managing Director Northern Metropolitan Territory; Catalan Health Institute
- General Director of Knowledge Transfer at the Universities and Research Department
- General Director, Catalan Foundation for Research and Innovation (FCRI)
- Research Director Hospital Clínic, of IDIBAPS and the Clínic Foundation for Biomedical Research
- President of the Management Committee, Josep Carreras Leukaemia Research Institute
- Treasurer, International Josep Carreras Foundation
- Trustee, International Josep Carreras Foundation
- Rector of the University of Barcelona (UB)
- Rector of the Autonomous University of Barcelona (UAB)
- Vice-Rector of Research at the University of Barcelona (UB)
- Vice-Rector for Research of the Autonomous University of Barcelona (UAB)

SECRETARY

- Director of the Institution CERCA of the Generalitat, Government of Catalonia

MANAGEMENT COMMITTEE

PRESIDENT

Dr. Evarist Feliu
Ombudsman at the Josep Carreras Institute

MEMBERS

- General Director of Research of the Ministry of Business and Knowledge for the Generalitat, Government of Catalonia
- General Director of Health Planning, Generalitat, Government of Catalonia
- General Sub-Director for Research and Innovation, Ministry of Health, Generalitat, Government of Catalonia
- Trustee, International Josep Carreras Foundation
- Vice-Rector for Research at the Autonomous University of Barcelona (UAB)
- Vice-Rector for Research at the University of Barcelona (UB).

SECRETARY

- Director of the Institution CERCA of the Generalitat, Government of Catalonia

INTERNAL SCIENTIFIC COMMITTE

Dr. Josep Maria Ribera Santasusana
Vice-director of Clinical Research

Dr. Anna Bigas Salvans
Vice-director of Basic Research

Dr. Albert Oriol Rocafiguera
Director of Applied Research

Dr. Rafael Marcos-Gragera
Director of Epidemiological Research

Dr. Jordi Esteve Reiner
Coordinator IJC Clinic Location

Dr. Javier Briones Mejide
Coordinator IJC Sant Pau Location

Dr. Francesc Solé Ristol
Coordinator IJC Can Ruti Location

SCIENTIFIC ADVISORY BOARD (SAB)

PRESIDENT

Prof. Luccio Luzzatto
Director of the Instituti Toscani de Tumori in Florence

MEMBERS

Prof. Robert Sackstein,
Professor at the Department of Dermatology and Medicine at Harvard Medical School and Director of the Program of Excellence in Glycosciences

Prof. Alberto Orfao
Titular Professor and Director of the General Cytometry Service of University of Salamanca

Prof. Brigitte Schlegelberger
Professor and Director of Hannover Medical School Genetics Institute

Prof. Maria Luisa Toribio
Research Professor at the Higher Council for Scientific Research (CSIC) at the Severo Ochoa Molecular Biology Center (CBMSO)

Prof. Christoph Plass
Head of Division of Cancer Epigenomics in German Cancer Research Center (DKFZ)

Prof. Teresa Palomero
Pathology and Cell Biology Associate Professor at CUMC (Columbia University Medical Center)

Prof. Francesco Bertroni
Head of Lymphoma Genomics Group at the Institute for Oncological Research in Bellinzona, Switzerland

Prof. Iannis Ainfatis
Pathology Department of the NYU School of Medicine.

Prof. Pura Muñoz-Cánoves
ICREA Research Professor and Cell Biology Professor in the Department of Experimental and Health Sciences at the UPF

ABOUT US

DIRECTOR

Dr. Manel Esteller is Chairman of Genetics at the University of Barcelona's Faculty of Medicine and an ICREA Research Professor. Since May 2019, Dr. Esteller has been the Director of the Josep Carreras Leukaemia Research Institute. Dr. Esteller is considered to be among the top 0.1% of world scientists based on impact by Stanford University (METRICS). He is also a member of numerous international scientific societies and his work has been recognized by numerous awards, including the World Health Summit Award (2010), the Rey Jaime I Research Award (2013), the National Award in Oncology (2014), the Dr. Josep Trueta Medal from the Catalan government (2015), the National Research Award from the Catalan government (2015), the Gold Medal from the Parliament of Catalonia (2016), the International Award of Catalonia (2016), the Innovation in Healthcare Oncology Award (2018), the Narcís Monturiol Medal from the Catalan government (2020), the Fernández-Cruz Award for excellence in biomedical research (2021) and the "Constantes y Vitales" for his scientific career in biomedical research (2022).

Prof. Manel Esteller

Director

Prof. Evarist Feliu

President of the Management Committee

Dr. Josep Maria Ribera

Clinical Research Deputy Director

Dr. Albert Oriol

Applied Research Director

Dr. Jordi Esteve Reiner

IJC Clinic Location Coordinator

Mrs. Ana Garrido Anglada

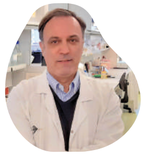
Strategy Director and Acting Managing Director

Dr. Anna Bigas

Basic Research Deputy Director

Dr. Rafael Marcos

Epidemiological Research Director



Dr. Javier Briones Mejide

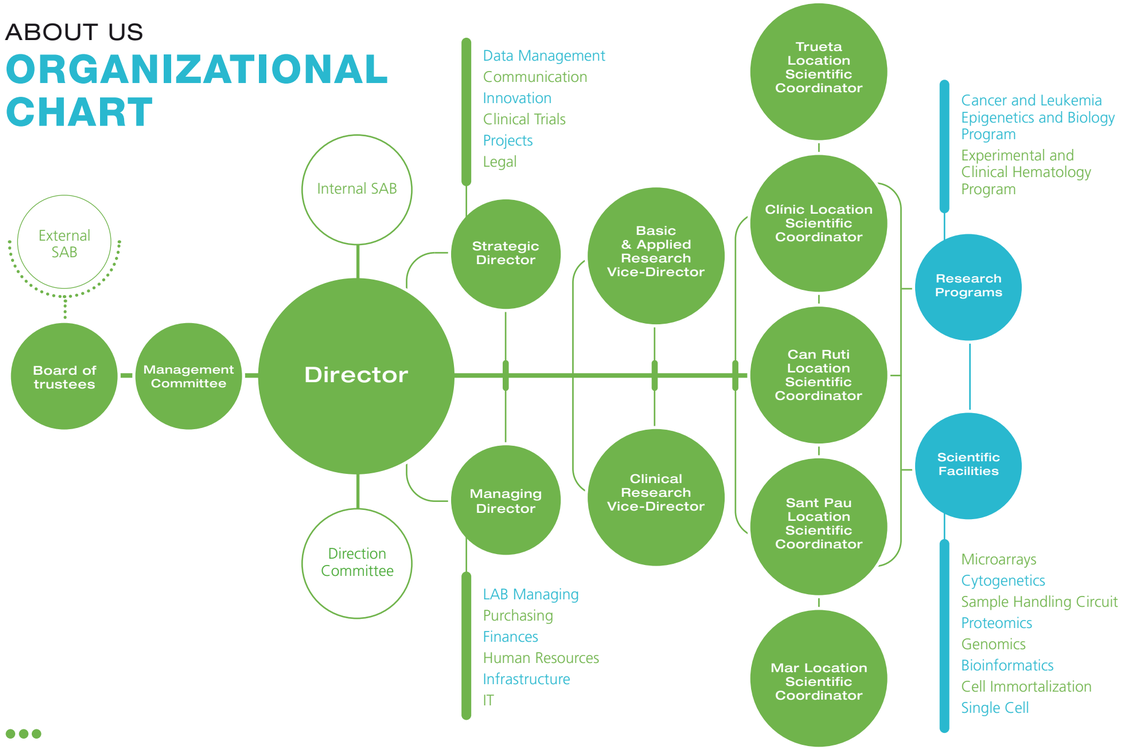
IJC Sant Pau Location Coordinator

Dr. Francesc Solé Ristol

IJC Can Ruti Location Coordinator

ABOUT US

ORGANIZATIONAL CHART

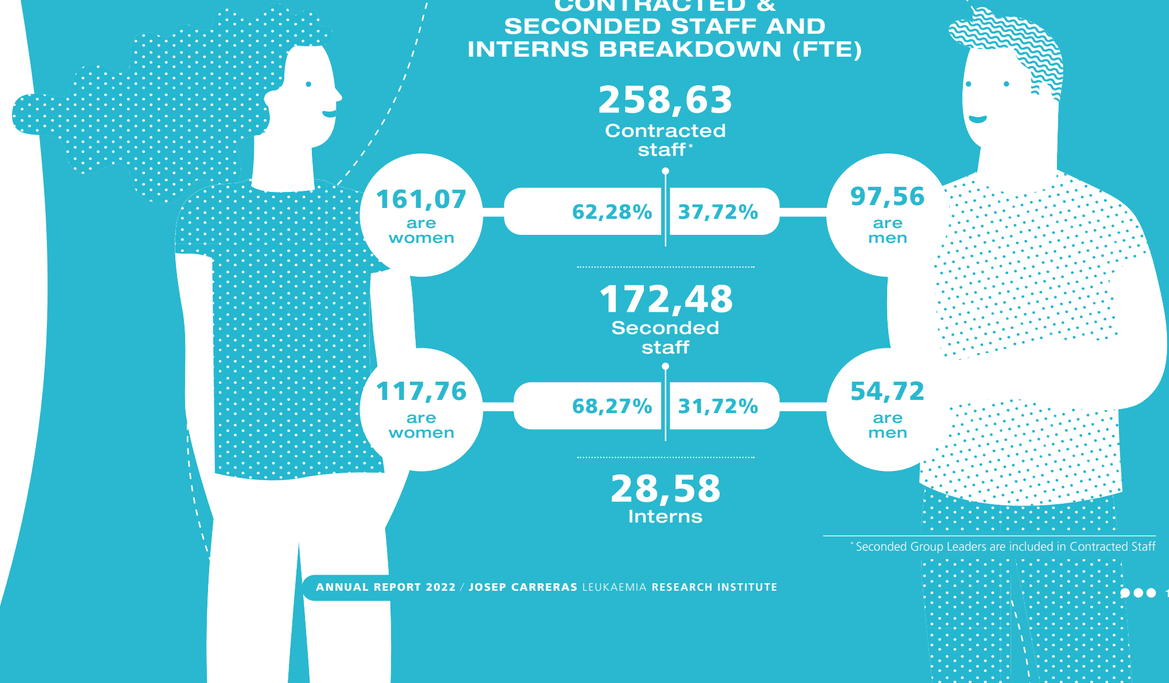


	FTE	Number of People
Contracted Staff	239,63	292
Seconded Staff	19	19
Collaborators	172,48	207
Interns	28,58	63

ABOUT US

OUR STAFF

CONTRACTED & SECONDED STAFF AND INTERNS BREAKDOWN (FTE)



* Seconded Group Leaders are included in Contracted Staff

ABOUT US

OUR STAFF

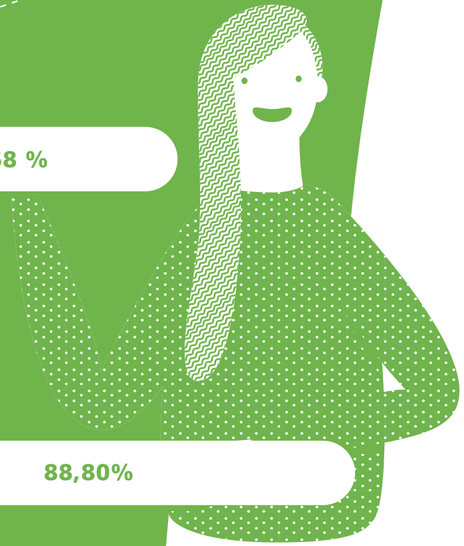
ADMINISTRATION VS. RESEARCH BREAKDOWN (FTE)

258,63 Contracted staff*

- **185,38** research staff — 71,68 %
- **19,72** Research support staff — 7,62%
- **53,53** Administration staff — 20,70%

28,58 Internships

- **25,38** Research Internships — 88,80%
- **3,20** Administration Internships — 11,20%



*Seconded Group Leaders are included in Contracted Staff

ABOUT US / **OUR STAFF**

LOCATIONS STAFF BREAKDOWN

258,63 Contracted staff* (FTE)

- **225,70** Staff in IJC Can Ruti — 87,27%
- **20,40** Staff in IJC Clinic — 7,89%
- **8,50** Staff in IJC Sant Pau — 3,29%
- **2,50** Staff in IJC Trueta — 0,97%
- **1,53** Staff in IJC Mar — 0,58%

172,48 Seconded staff

- **5,04** Staff in IJC Mar — 2,92%
- **11** Staff in IJC Clinic — 6,38%
- **22,63** Staff in IJC Sant Pau — 13,12%
- **37** Staff in IJC Trueta — 21,45%
- **96,81** Staff in IJC Can Ruti — 56,13%

* Seconded Group Leaders are included in Contracted Staff

ABOUT US

RESEARCH PROGRAMS

CANCER AND LEUKEMIA EPIGENETICS AND BIOLOGY PROGRAM (PEBCL)

- Cancer Epigenetics led by Manel Esteller
- Cancer Genetics led by Montse Sanchez-Cespedes
- Chromatin Biology led by Alex Vaquero
- Chromatin, Metabolism and Cell Fate led by Marcus Buschbeck
- 3D Chromatin Organization led by Biola M. Javierre
- Epigenetics and Immune Disease led by Esteban Ballestar
- Lymphocyte Development and Disease led by Maribel Parra
- Regulatory Genomics led by Tanya Vavouri
- Regulatory RNA and Chromatin led by Sònia Guil
- Epigenetic Control of Hematopoiesis led by José Luis Sardina
- Transcriptional Dynamics in Leukemia led by Sergi Cuartero
- Cancer Immunogenomics led by Eduard Porta
- Cancer Heterogeneity and Hierarchies led by Verónica Rodilla
- Leukemia and Immuno-Oncology led by Laura Belver
- Cellular Systems Genomics led by Elisabetta Mereu
- Stem Cells and Cancer led by Anna Bigas
- Endothelial Pathobiology and Microenvironment led by Mariona Graupera
- T-Cell Lymphoma led by Laura Mondragón
- Nuclear Architecture in Leukemia led by Gregoire Stik

EXPERIMENTAL AND CLINICAL HEMATOLOGY PROGRAM (PHEC)

- Acute Lymphoblastic Leukemia (ALL) led by Josep Mª Ribera
- Barcelona Endothelium Team (BET) led by Enric Carreras
- Myeloid Neoplasms led by Lurdes Zamora and Blanca Xicoy
- Immunohematology and Glycobiology led by Fumiichiro Yamamoto
- Leukemia Stem Cell led by Ruth Risueño*
- Lymphoid Neoplasms led by Tomás Navarro
- Multiple Myeloma led by Albert Oriol
- Myelodysplastic Syndromes led by Francesc Solé
- Stem Cell Biology, Developmental Leukemia and Immunotherapy led by Pablo Menéndez
- Cellular Immunotherapy and Gene Therapy led by Javier Briones
- Stem Cell Transplantation and Cellular Immunotherapy led by Álvaro Urbano-Ispizua
- Epigenetic Therapies led by María Berdasco
- Lymphoma Translational led by Gaël Roué
- Descriptive Epidemiology, Genetics and Cancer Prevention led by Rafael Marcos Gragera
- Oncogenesis and Antitumor Drugs led by Ramon Mangues
- Chronic Lymphocytic Leukemia led by Carolina Moreno
- Hematology Research led by David Gallardo
- Myeloid Neoplasms (Clínic) led by Jordi Esteve
- Hematological Diseases, Transplant and Cell Therapy led by Jordi Sierra
- Hematological Diagnosis led by Josep Nomdedéu

* The Leukemia Stem Cell Group left the Josep Carreras Institute on October 31, 2022

ABOUT US

RESEARCH GROUPS

CANCER EPIGENETICS LED BY MANEL ESTELLER

GROUP MEMBERS

ESTELLER BADOSA, MANEL
Group Leader

SETIÉN BARANDA, ESTEBAN FERNANDO
Associate Researcher

DÁVALOS VEGA, MARIA VERÓNICA
Associate Researcher

MUSULÉN PALET, EVA
Associate Researcher

JANIN, MAXIME HENRI
Postdoctoral Researcher

ORTIZ BARAHONA, VANESSA
Associate Researcher

BLECUA CARRILLO ALBORNOZ, PEDRO
Senior Researcher

FERRER AGUILAR, GERARDO
Postdoctoral Researcher

PONTEL, LUCAS BLAS
Postdoctoral Researcher

CAMPILLO MARCOS, IGNACIO
Postdoctoral Researcher

CARRIER, ARNAUD
Postdoctoral Researcher

NOGUERA CASTELLS, ALEIX
Postdoctoral Researcher

ORŠOLIC, INES
Postdoctoral Researcher

GOMEZ PEREIRA, CRISTINA
Researcher Assistant

BUENO COSTA, ALBERTO
PhD Student

MARTINEZ VERBO, LAURA
PhD Student

GARCIA PRIETO, CARLOS ANTONIO
PhD Student

PARRA, JERÓNIMO
PhD Student

CASADO PELAEZ, MARTA
PhD Student

VESELINOVA KALAYDZHIEVA, YOANA
PhD Student

POPOV, ANTON
PhD Student

SANTOS PUJOL, ELOY
Junior Researcher

QUERO DOTOR, CARLOS
Junior Researcher

COLL SAN MARTÍN, LAIA
Lab Technician

SOLER RIERA, MARTA
Lab Technician

OVERVIEW

The group continues the wide-ranging work on epigenetics that Manel Esteller, the group leader, has carried out during his career until now. Current research is devoted to the establishment of the epigenome and epitranscriptome maps for normal and transformed cells, the study of the interactions between epigenetic modifications and non-coding RNAs, and the development of new epigenetic drugs for cancer therapy.

OUR RESEARCH

Our laboratory is one of those responsible for establishing the observation that epigenetic disruption of mRNA transcription, particularly in DNA methylation and histone modification patterns, contribute to the initiation and progression of human tumours (reviewed in Esteller, N Engl J Med 2008; Heyn and Esteller, Nat Rev Genet 2012; Berdasco and Esteller, Nat Rev Genet 2019).

It has also been recognized that microRNAs (small non-coding RNAs that regulate gene expression by sequence-specific base pairing in mRNA targets) also play a key role in the biology of the cell, and can have an impact on the development of cancer. In this context, we characterized the first miRNA undergoing specific cancer-methylation associated silencing (Lujambio et al., Cancer Res 2007), followed by the characterization of many other miRNAs disrupted in the same manner (Lujambio et al., PNAS 2008; Davalos et al., Oncogene 2012).

We have also studied other types of ncRNA, such as subclasses of lncRNA, undergoing aberrant DNA methylation events in human cancer (Lujambio et al., Oncogene 2010; Guil et al, Nat Struc Mol Biol 2012; Liz et al., Mol Cell 2014; Diaz-Lagares et al., PNAS 2016). We have shown that sometimes these epigenetic lesions occur outside the minimal promoters and take place in enhancers (Heyn et al., Genome Biol 2016; Vidal et al, Oncogene 2017) or at cryptic internal promoters (Vizoso et al., Nature Medicine 2015).

OUR GOALS

Our group has had a long-standing interest in translating the use of epigenetic knowledge gained from research into biomarkers to predict clinical outcome and to assay new drugs to reverse the distorted epigenetic landscape (Berdasco and Esteller, Nature Review Genetics 2019). For example, we have used epigenetic markers to predict response to anti-tumour therapies and following the initial observation that MGMT gene methylation predicted response to alkylating agents in glioma (Esteller et al., N Engl J Med 2000).

We have shown the relationship of methylation of MGMT with the response to alkylating agents in lymphoma (Esteller et al., J Natl Cancer Inst, 2002); of WRN with the response to irinotecan (Agrelo et al., Proc Natl Acad Sci USA, 2006); of BRCA1 with the response to PARP inhibitors (Veeck et al., J Natl Cancer Institute, 2010) and of DERL3 with the response to glycolysis inhibitors (Lopez-Serra et al., Nature Communications, 2014). Methylation of SRBC (Moutinho et al. J Natl Cancer Institute, 2014) and SLFN11 (Nogales et al., Oncotarget 2015) have also been identified as resistance markers for platinum derivatives in human tumours and the regulator of EGFR TBC1D16 has been identified as a sensitizer for therapies with BRAF and MEK inhibitors (Vizoso et al., Nature Medicine 2015). Epigenetic loss of SVIP is also related to the response to GLUT1 inhibitors (Llinas-Arias et al. JCI Insight 2019). From a multiomics standpoint, we have contributed to the characterization of drug sensitivity in 1,000 cancer cell lines (Iorio et al., Cell 2016) and unveiled the reasons for those patients described as "exceptional responders" (Wheeler et al., Cancer Cell 2021).

OUR CHALLENGES

Continuing with this translational side of our work, we are also interested in the development and study of new epigenetic drugs that target DNA methylation and histone modification writers, readers and erasers and could have an anti-cancer effect (Lara et al Oncogene 2008; Zubia et al Oncogene 2009; Huertas et al., Oncogene 2012; Perez-Salvia et al., Oncotarget 2017; Perez-Salvia et al., Haematologica 2018).

Interestingly, the "repertoire" of epigenetic modifications of DNA is fairly limited, as we recently reviewed (Heyn and Esteller, Cell 2015). In sharp contrast, more than one hundred post-transcriptional modifications occur in RNA (Esteller and Pandolfi, Cancer Discovery 2017; Davalos et al., Cell 2018;

Rosselló-Tortella, Ferrer and Esteller, Blood Cancer Discovery 2020).

Until very recently it was almost impossible to make a good map of the epigenetic modifications of the RNA molecule, which hampered many studies in this area and prevented advances in the study of the significance of each RNA modification. However, recent methodologies now allow the study of the so-called epitranscriptome. In this field, we have shown aberrant RNA editing mediated by ADAR1 amplification in lung cancer (Anadon et al., Oncogene 2016), altered RNA decapping mediated by NUDT16 epigenetic silencing in T-ALL (Anadon et al., Leukemia 2017), RNA methylation loss in ribosomal RNA in glioma (Janin et al., Acta Neuropathol 2019), unpaired guanine modification of transfer RNA in colon cancer (Rosselló-Tortella et al., PNAS 2020) and m1A defects in Hodgkin's lymphoma (Esteve-Puig et al., Blood 2020) epigenetic disregulation of tRNAs in several tumor types (Rosselló-Tortella et al., Molecular Cancer, 2022) and the contribution of m6A RNA shifts in cellular transdifferentiation (Bueno-Costa et al., Leukemia 2022). Knowledge in this area is limited and its study is the focus of intense research in the lab.

We have also a long-standing vocation for research in monogenic disorders affecting epigenetic genes (Urdinguio et al., Lancet Neurol. 2009), particularly in Rett syndrome. The disease is associated with a germline mutation in MECP2, a protein that it is attracted to methylated DNA. Over the years, we have identified the gene targets for MECP2 (Ballestar et al., EMBO J 2013; Petazzi et al. RNA Biol. 2013, Neurobiol Dis. 2014), studied the genomics of Rett syndrome in detail (Saez et al., Genet Med 2016; Lucariello et al., Hum Genet 2016) and developed pre-clinical drug studies (Szczesna et al., Neuropsychopharmacology et al., 2014; Jorge-Torres et al., Cell Reports 2018).

In a similar context, we are also curious about the epigenomic profiles of common diseases such as cardiovascular alterations (Zaina et al., Circ Cardiovasc Genet. 2014; Valencia-Morales et al., BMC Med Genomics 2015) and Alzheimer and other neurodegenerative diseases (Sanchez-Mut et al., Brain et al., 2013; Hipoccampus 2014; Transl Psychiatry. 2016; Nature Medicine, 2018).

Finally, we have a strong interest in the establishment of new epigenomic platforms to elaborate comprehensive DNA methylome maps, our lab is the pioneer in the validation of the commonly used DNA methylation microarrays such as the 450K (Sandoval et al., Epigenetics 2011) and the EPIC/850K (Moran et al. Epigenomics 2016), plus the mouse DNA methylation microarray (García- Prieto et al., Epigenetics 2022). The use of these approaches has made several breakthroughs possible, such as: the establishment of DNA methylation signatures that are associated with early dissemination in lung cancer (Sandoval et al., JCO 2010); the diagnosis of the tumor type in Cancer of Unknown Primary (CUP) (Moran et al., Lancet Oncology 2016); the better understanding of the response to anti-PD1 immunotherapy (Duruisseaux et al., The Lancet Respiratory Medicine 2018); the obtention of the first DNA methylome of CAR-T cells with clinical value (Garcia-Prieto et al., J National Cancer Institute 2021) or the prediction of COVID-19 clinical severity according to the epigenetic setting in adult (Castro de Moura et al., Lancet EBioMedicine 2021) and children (Davalos et al., Lancet Eclinical-Medicine 2022).

KEYWORDS

Cancer epigenetics; DNA methylation; RNA epitranscriptomics; histone modification; epigenetic gene silencing

ABOUT US

RESEARCH GROUPS

CANCER GENETICS
LED BY MONTSE SANCHEZ-CESPEDES

GROUP MEMBERS

SANCHEZ-CESPEDES, MONTSE
Group Leader

ROMERO FERRARO, OCTAVIO ALFREDO
Senior Researcher

SAIGÍ MORGUÍ, MARIA
Postdoctoral Researcher

FERRERO ANDRÉS, ANA
Postdoctoral Researcher

CUCURULL SALAMERO, MARC
Attending Physician

VILARRUBÍ PORTA, ANDREA
PhD Student

NAVAJAS CHOCARRO, PABLO
PhD Student

MORILLAS VIÑUALES, JUAN
PhD Student

DÍAZ MUÑOZ, ANA CRISTINA
PhD Student

PROS SIMÓN, EVA
Lab Technician

BARTOLESSIS ARIAS, ISABEL
Lab Technician

OVERVIEW

Lung cancer causes over 1.3 million deaths annually and remains the deadliest type of cancer worldwide. Although efforts in recent years to fully characterize human cancer on a genetic and molecular level have provided important insights to increase our understanding of the gene alteration profile underlying the development of Lung Cancer, the impact of this knowledge in the survival of patients remains modest. Our group is devoted to the genetic, epigenetic and molecular study of the mechanisms that drive LC development. Ultimately, our purpose is to implement the clinical management of cancer patients and to design novel therapeutic strategies.

OUR RESEARCH

The complete genetic characterization of tumors is important to understand cancer development, promote the discovery of new drugs and improve the selection of patients that may benefit from a given cancer therapy. Our research uses the latest high-throughput sequencing technologies to create profiles and catalogues of the recurrently altered genes in cancer. We also have a keen interest in understanding the mechanisms by which the abnormal function of these genes contributes to cancer development.

OUR GOALS

Our laboratory is currently engaged in a number of important projects:

1.

Screening for factors that determine tumor immunoescape and the response to immunotherapy.

We have become increasingly interested in the study of those biological factors, which allow tumors to escape control of the immune system and determine the response to immunotherapy.

2.

Genomic and genetic profiling of lung tumors to identify novel targets for therapeutics and determinants for the primary and acquired response to tyrosine kinase inhibitors (TKIs).

We use high-throughput genomic sequencing technologies, such as whole exome and RNA-sequencing, to gather information about the genetic background and gene expression profiles of lung tumors from both smokers and non-smokers.

3.

Genetic alterations at epigenetic factors: biological understanding and opportunity for novel therapeutics.

Over the past 15 years, our group has provided key information to understanding cancer biology. Currently, we are using high-throughput technologies to understand tumor development and to identify molecular vulnerabilities that can be used therapeutically.

OUR CHALLENGES

Recent epidemiological data point to a worrying increase in the incidence of LC in those who have never smoked, particularly women. The reasons are not well understood, a fact that limits the design of prevention measures.

Through our research, we hope to answer the following questions:

1. What are the genetic and molecular abnormalities that trigger the development of cancer, particularly Lung Cancer?
2. How can we use genetic/molecular information to identify novel targets to implement Lung Cancer therapeutics?
3. What is the molecular basis for the lack of response to immunotherapyr?
4. How can we predict and prevent acquired resistance to targeted therapeutics?

KEYWORDS

Targeted therapeutics; immunotherapy; epigenetic regulation; SWI/SNF-complex; MYC/MAX-pathway

ABOUT US

RESEARCH GROUPS

CHROMATIN BIOLOGY LABORATORY LED BY ALEX VAQUERO

GROUP MEMBERS

VAQUERO GARCÍA, ALEJANDRO
Group Leader

VÁZQUEZ PRAT, BERTA NIEVES
Postdoctoral Researcher

ESPINOSA ALCANTUD, MARIA DOLORES
Postdoctoral Researcher

MARAZUELA DUQUE, ANA
Postdoctoral Researcher

FERNÁNDEZ DURAN, IRENE
Postdoctoral Researcher

IANNI, ALESSANDRO
Postdoctoral Researcher

BOSCH PRESEGUÉ, LAIA
Attending Physician

CASTELLÓ GARCÍA, JOSE MANUEL
PhD Student

GÁMEZ GARCÍA - CERVIGÓN, ANDRES
PhD Student

GUITART SOLANES, ANNA
PhD Student

PAÑOS MOLERO, LUIS EULALIO
PhD Student

OVERVIEW

The members of the sirtuin family of NAD+-dependent enzymes are key coordinators of this response, as they play an important role in the crosstalk between the environment and the genome, at both cellular and physiological level. In particular, they play a key role in the maintenance of genome stability, epigenetics, metabolic homeostasis, and cell differentiation and development. The relevance of sirtuin function is highlighted by their involvement in some of the most common human pathologies, including cancer (such as blood malignancies), diabetes and other endocrine-related diseases, neurodegenerative diseases and ageing.

OUR RESEARCH

The chromatin biology lab's primary purpose is understanding the mechanisms of the stress response and their impact on cancer and ageing. Specifically, the group focuses its efforts on defining the contribution of sirtuins to this response in the maintenance of genome stability, epigenetics and metabolic homeostasis.

To fulfil this main objective, the group's work encompasses a wide range of research areas, from basic aspects of sirtuin biology to their contribution in the development of human pathologies such as leukemia and ageing.

OUR GOALS

We aim at the identification of novel mechanisms and factors involved in the onset and development of blood malignances, and the creation of tools that could be helpful for its diagnosis and treatment. In this regard, the group's main objectives are:

1.

To understand the enzymatic duality of sirtuins and their specific contribution to sirtuin function.

In particular, we focus our efforts on the poorly understood topic of ADPRT activity.

2.

To characterize sirtuin-dependent mechanisms of genomic stability,

including constitutive heterochromatin integrity, DNA damage signalling and repair, and cell cycle checkpoint control.

3.

To define the role of sirtuins in B-cell differentiation and characterize their functional implication in cancer,

particularly in the context of hematopoietic pathologies such as leukemia and lymphoma. Our main efforts are currently focused on two types of leukemia, pediatric B-ALL and AML.

4.

To understand the involvement of sirtuin function in the beneficial effects of nutrient restriction on ageing development.

5.

To develop a new methodology to measure the activity of sirtuins *in vivo*.

OUR CHALLENGES

Through our research, we seek to answer the following questions:

1

What is the physiological mechanism associated with the genotoxic and metabolic stress response?

2

What is the contribution of the sirtuin family of enzymes to the maintenance of genome stability after stress?

3

What is the implication of these mechanisms in the onset and development of blood cancers and ageing?

KEYWORDS

Stress response; sirtuins; epigenetics; leukemia; ageing

ABOUT US

RESEARCH GROUPS

CHROMATIN, METABOLISM AND CELL FATE LED BY MARCUS BUSCHBECK

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OVERVIEW

We focus on understanding the molecular aspects of chromatin regulation and have a long-standing interest in the study of histone variants. We want to find ways to translate knowledge about chromatin regulation into therapeutic tools for the management of diseases such as blood cancers.

OUR RESEARCH

We seek to bridge the gap between basic molecular research and translational research by exploring chromatin regulation, in particular the molecular biology of histone variants. We aim to exploit this knowledge for the identification of novel intervention strategies for the treatment of blood cancers. We focus on the continuum of myeloid diseases, ranging from the premalignant expansion of altered clones to chronic myelodysplastic syndromes and acute myeloid leukemia.

OUR GOALS

Through our research, we aim to gain a better understanding of the epigenetic mechanisms that contribute to the development of blood cancers. By functionally mining the chromatin regulatory space, we further aim to provide new starting points by identifying novel drug targets. In this regard, our research focuses on two main lines:

1.
To mine the chromatin regulatory space to identify novel drug targets that can either help improve current treatments or intercept disease at an early asymptomatic stage.

2.
We study histones from the protein core of the nucleosome, particularly the variant macroH2A that led to two major discoveries: its major role in nuclear organization and its ability to bind metabolites through its mostly understood macrodomain, establishing a direct link between chromatin and metabolism.

OUR CHALLENGES

Through our research, we hope to answer the following questions:

1
How do epigenetic mechanisms operate on the molecular level?

2
How do chromatin and, in particular, histone variants contribute to cell fate transitions?

3
How can we exploit this knowledge for the development of novel therapeutic strategies?

KEYWORDS

myelodysplastic syndrome, acute myeloid leukemia, chromatin, nuclear organization, histone variants

ABOUT US

RESEARCH GROUPS

3D CHROMATIN ORGANIZATION LED BY BIOLA M. JAVIERRE

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OVERVIEW

Genetics and epigenetics of normal and malignant haematopoiesis in space and time.

We are a group of passionate scientists with an insatiable thirst for learning about spatiotemporal architecture of the genome and its role in cell differentiation and function in health and disease. Our group combines cutting-edge experimental and bioinformatics approaches to understand the dynamic and specific 3D chromatin organization of normal and malignant haematopoiesis and its interaction with non-coding determinants and trans-regulatory elements. Our long-term goal is to keep making progress in the fight against cancer. We will not stop until a cure is found.

OUR RESEARCH

Enhancers are critical modulators of gene transcription through physical interactions with target promoters that often locate distally in the genome. The physical proximity between enhancers and promoters is ultimately enabled and determined by the three-dimensional folding of the chromatin within the nucleus. Although enhancers can be defined through well-characterized features, predicting their target genes at distal location remains challenging due to the high complexity of studying enhancer-promoter interactions, and the large variability according to cell-type and state. This gap of knowledge is particularly problematic for understanding the molecular mechanisms associated to inherited and de novo acquired mutations and epimutations involved in common human diseases, which are all highly enriched at regulatory elements

To overcome these critical limitations, we have recently developed a low input cost-effective method to robustly map and compare promoter interactomes at high resolution in rare cell populations previously unmeasurable. This new method broadens the capacity for studying organism developments, in vivo cell commitment, cellular response to a wide range of external stimulus and disease pathogenesis.

OUR GOALS

Our lab's main research goals, which are motivated by this gap in the knowledge, are as follows:

1.

To define the cell type-specific 3D chromatin organization in human haematopoietic cells.

Human haematopoietic differentiation dogma is currently a subject of debate. All blood cells originate from haematopoietic stem cells (HSCs), which represent the apex of a differentiation cascade of progenitor cell types that gives rise to billions of new differentiated cells every day. HSC differentiation, which progresses through stepwise hierarchical restriction of lineage potential, has been extensively characterized at epigenetic, transcriptional and functional levels. However, the contribution of genome architecture in regulating haematopoiesis remains unexplored.

Motivated by this gap of knowledge, we aim to investigate whether the dynamic changes in chromatin interactions between gene promoters and regulatory elements can shape transcription decisions controlling haematopoiesis and blood cell function. These insights can lead to improvements in regenerative medicine strategies, especially bone marrow transplants, which represent one of the most promising approaches to treating many diseases, including blood cancer.

2.

To identify the altered DNA topology in blood cancer.

The genome architecture plays a key role in genome expression regulation and DNA repair. Chromatin interactions are therefore crucial for cellular health, and errors in these interactions can give rise to the development of a broad range of diseases, including blood cancer. Research into these altered 3D structures can help improve knowledge of the tumour process, thereby providing new opportunities for the development of novel treatment approaches and diagnostic strategies.

3.

To prioritize new candidate genes and pathways related to leukemias and lymphomas.

During the previous years, thousands of determinants associated with blood cancer have been identified. However, most of them remains unexplored because of these target non-coding regions, frequently enhancers and other distal regulatory elements. Genetic and epigenetic alterations at distal regulatory elements have the potential to alter the regulatory properties and ultimately lead to quantitative changes in expression of distal target genes with pathological outcome. However, in most of the cases, the target genes area unknown. By studying the physical interactions between gene promoters and regulatory elements, we connect blood cancer cis and trans determinants to putative target genes, thereby prioritizing new candidate genes and pathways and offering an insight into the genomic regulatory mechanisms underlying cancer. In addition, the interpretation of the non-coding regions altered in disease will also help us improve patient outcome prediction and allow us to design better, more personalized treatments.

OUR CHALLENGES

Through our research, we hope to answer the following questions:

1. Can the dynamic changes in chromatin interactions shape the transcription decisions controlling haematopoiesis and blood cell function?
2. Which are the blood cell-type specific key factors orchestrating genome architecture?
3. How does the altered genome architecture drive malignant transformation?
4. What is the role of non-coding determinants in cancer predisposition, development and relapse?

Why our research matters

Blood cancers, including leukemias and lymphomas, are a leading cause of mortality in paediatric and adult patients worldwide. We aim to provided fundamental understanding of blood cancer development and relapse to identify new biomarkers and novel therapeutic targets to ultimately improve patient survival.

KEYWORDS

Genome architecture, spatial-temporal chromatin organization, haematopoiesis, blood cancer, cis non-coding determinants, enhancer-promoter interactions

ABOUT US

RESEARCH GROUPS

EPIGENETICS AND IMMUNE DISEASE LED BY ESTEBAN BALLESTAR

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OVERVIEW

We aim at understanding the mechanisms underlying the deposition and removal of epigenetic modifications in immune cells, the influence of genetic and environmental determinants, and the acquisition of epigenetic alterations in immune-mediated disease including primary immunodeficiencies, autoimmune and autoinflammatory diseases. We also investigate the impact of the epigenetic regulation of immune cells in the microtumor environment.

OUR RESEARCH

We started these lines of research more than 10 years ago, by studying the occurrence of DNA methylation alterations in the context of systemic lupus erythematosus (SLE), an archetypical systemic autoimmune disease. Later on, we performed new studies with MZ twins discordant for common variable immunodeficiency (CVID), the most prevalent symptomatic primary immunodeficiency.

More recently, our team also demonstrated the occurrence of DNA methylation alterations in monocytes in representative autoinflammatory syndromes. We have shown that alterations in the DNA methylome of peripheral blood monocytes reflect the disease activity in rheumatoid arthritis mediated by the elevated levels of inflammatory cytokines present in such state.

OUR GOALS

Our main lines of research and specific goals are:

1.

To understand the role of epigenetic control and its upstream determinants in relation with immune function.

We aim at understanding how immune cell-cell crosstalk, cytokines and other factors, cell signalling pathways and transcription factors determine epigenetic control and impact immune cell function.

2.

To identify epigenetic alterations in immune-mediated diseases and investigate their clinical relevance.

Our studies focus on different diseases including primary immunodeficiencies, such as common variable immunodeficiency (CVID) and hyper IgM type 2 syndrome, and autoimmune diseases, such as rheumatoid arthritis, systemic sclerosis and systemic lupus erythematosus.

3.

To investigate the effects of immunomodulators and epigenetic compounds in shaping the epigenome and responses of immune cells.

We dissect the molecular consequences of different immunomodulators as well as inhibitors of epigenetic enzymes in immune cells.

OUR CHALLENGES

The study of epigenetic dysregulation can help understand the determinants of immune dysregulation and can have an impact in the treatment of these diseases. Therefore, with our research we want to answer:

1

How do immune cells translate the surrounding information provided by the direct contact with other cells or the cytokines and other molecules into epigenetic profiles that determine their responses?

2

What is the relevance of the epigenetic alterations that are found in different immune mediated diseases in relation to the aberrant function of these cells?

3

How can we apply the knowledge on the epigenetic dysregulation in immune-mediated disease to the clinics?

KEYWORDS

Epigenetics, DNA methylation, Immune-mediated disease, autoimmune disease, primary immunodeficiency

ABOUT US

RESEARCH GROUPS

LYMPHOCYTE DEVELOPMENT AND DISEASE LED BY MARIBEL PARRA

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OVERVIEW

B-cell lymphopoiesis is a complex developmental process that involves several cellular transitions, including cell commitment and early and late cellular differentiation. Proper transcriptional control at each cellular transition is essential for the correct development of B lymphocytes. How specific gene expression programmes are selected and maintained, thus resulting in the proper generation of B cells, remains a fundamental question in biology. Conversely, how the aberrant establishment of cell- and lineage-specific gene transcriptional programmes leads to the development of B-cell malignancies such as leukemia and lymphoma also requires extensive research.

OUR RESEARCH

Our current research focuses on four main lines:

- **Understanding the entire role of HDAC7 in early and terminal B-cell development.** HDAC7 is an epigenetic modulator that represses functional or lineage-inappropriate gene expression in B lymphocytes
- **Establishing HDAC7 as a novel biomarker and potential therapeutic target in pro-B acute lymphoblastic leukemia (pro-B-ALL) and diffuse large B-cell lymphoma (DLBCL).** We found that the deregulation of HDAC7 may be involved in the pathogenesis of acute lymphoblastic leukemia.
- **Working towards precision medicine against DLBCL heterogeneity using organoid culture systems.** We are investigating additional epigenetic regulators in normal and aberrant B-cell generation and implementing 3D organoid cultures from DLBCL sample patients.
- **Improving immunotherapy combinatorial therapy in DLBCL.** R-CHOP is the gold standard treatment for DLBCL patients. R-CHOP therapy combines anti-CD20 antibody (immunotherapy) with cyclophosphamide, doxorubicin, vincristine and chemotherapy.

OUR GOALS

Acute lymphoblastic leukemia (ALL) is the most common type of cancer in children under a year old. Even though the chances of survival in infants suffering from ALL have improved significantly in recent years, an exhaustive study of the mechanisms underlying this disease is still required to make further therapeutic advances. Therefore, our group focus on:

1.
To understand how gene silencing is established during normal and aberrant B-cell differentiation.

2.
To transfer our basic knowledge in the epigenetics and transcriptional control of B-cell development to the clinical setting for infant B-ALL and DLBCL patients.

- To identify small molecules aimed at HDAC7 targeted modulation for combinatorial and precision medicine in infant pro-B-ALL with MLL-AF4 rearrangement.
- To identify novel targets for the design of next-generation immunotherapies in DLBCL.

3.
To implement a 3D organoid platform for DLBCL patient samples to perform compound library screenings aimed at unveiling new drugs for use in combinatorial therapy with current immunotherapy in a personalized manner.

OUR CHALLENGES

Through our research, we aim to answer the following questions:

1
How do B lymphocytes decide their identity? How is gene silencing established?

2
Why does HDAC7 expression improve the prognosis of some hematopoiesis diseases?

3
Why is HDAC7 underexpressed in pro-B-ALLA and DLBCL?

4
How can we restore HDAC7 expression in pro-B-ALL and DLBCL to impair disease progression?

5
Can we implement 3D organoids from DLBCL patients aimed at drug screening towards a precision medicine strategy and immunotherapy improvement?

KEYWORDS

B lymphocyte development, Epigenetics and transcriptional regulation,HDAC7, B cell acute lymphoblastic leukemia (B-ALL), Diffuse large B-cell lymphoma (DLBCL)

ABOUT US

RESEARCH GROUPS

REGULATORY GENOMICS LED BY TANYA VAVOURI

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OVERVIEW

Regulation of gene expression is the fine-tuning of the synthesis of the functional product of genes and is one of the most fundamental processes in life. It is the process that makes different cell types have different properties and differentiates unhealthy from healthy cells. Gene expression is regulated by internal signals (the activity of other genes, mutations, etc.) and by external signals (diet, temperature, pharmacological therapies, etc.).

OUR RESEARCH

Our research focuses on three main areas:

- Firstly, we study **the effect of the environment on gene expression** changes that are transmitted from parents to their offspring. We want to understand how information about our exposure to different environments may be encoded in molecules - other than DNA - inside germ cells that are transmitted between generations.
- Secondly, we work on **non-coding RNAs and other non-coding elements that influence gene expression.** We are interested in which non-coding elements affect gene expression and how.
- Finally, we want to **understand how epigenetic drugs affect gene expression and chromatin** in different genomic contexts. Epigenetic drugs currently used in the clinic include those for the treatment of patients with acute myeloid leukemia and myelodysplastic syndrome. A more in-depth understanding of the effects of these drugs and how they work may lead to improved or more personalized medicine in the future.

OUR GOALS

Our aim is to contribute to a better understanding of gene regulation and the consequences of drug treatments and inter-individual genetic variation in gene expression. Although most of our research is based on data from animal model organisms or cell lines, we hope that, in the long term, the knowledge acquired will increase our understanding about humans. Extensive aberrant gene expression and genome deregulation are extremely common in cancer, especially hematopoiesis forms, and treatments targeting gene regulation pathways are being used for hematopoiesis malignancies. Last, but not least, we hope that the data we generate and the analysis methods we develop serve as useful tools for the wider research community.

OUR CHALLENGES

We hope that our research sheds light on the following questions:

1. Which epigenetic mechanisms are involved in the transmission of acquired or variable traits between generations in humans and other animals?
2. Which non-coding DNA elements affect gene expression and therefore potentially phenotype?
3. How drugs (such as those used for the treatment of blood cancers) affect gene expression and the function of the non-coding parts of our genome?

KEYWORDS

Bioinformatics, gene regulation, epigenetic inheritance, germline, genomics

ABOUT US

RESEARCH GROUPS

REGULATORY RNA AND CHROMATIN LED BY SÒNIA GUIL

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OVERVIEW

We study the emerging roles of noncoding RNAs as key regulators of gene expression in physiological cellular programmes and at the onset or during progression of human diseases, with a major focus on tumorigenesis and neurodevelopmental diseases. The research carried out by our group combines biochemical, cellular and global genomic approaches to dissect mechanisms of gene expression regulation with the participation of ncRNAs, with the ultimate aim of revealing molecules of therapeutic/biomarker interest for clinical translation.

OUR RESEARCH

The lab focuses on a variety of RNA and RNA-binding protein functions in the context of changing cellular conditions. Our interest concentrates on the noncoding transcriptome, with the main aim of separating the wheat from the chaff to reveal true biologically relevant molecules and to understand how they are connected to broader gene regulatory networks.

Cancer research has led the way in the study of noncoding RNAs, but the abundance and key roles of the noncoding transcriptome in the human brain are being increasingly recognized. Importantly, common dysregulated mechanisms in different pathological contexts and with the involvement of ncRNAs, are emerging.

OUR GOALS

Our research aims to gain a better understanding of the biological relevance of ncRNAs for an informed use in therapeutic strategies. Recently, our group's research has taken advantage of state-of-the-art global transcriptomic approaches to identify ncRNA candidates that act as master regulators of oncofoetal genes, thereby revealing their validity as biomarkers in human cancer.

In addition to our work related to cancer, the group has been developing new experimental tools for research into Rett syndrome, a neurodevelopmental disorder usually caused by loss-of-function mutations in the epigenetic regulator MeCP2.

OUR CHALLENGES

Through our research, we hope to answer the following questions:

1. What is the precise contribution of the noncoding transcriptome to tumor biology?
2. How can we use RNA tools to improve treatment or diagnosis of human disease?
3. How can we better model neurodevelopmental diseases such as Rett syndrome to understand key initial changes in gene expression programmes?

KEYWORDS

Noncoding RNAs, cancer epigenetics, gene expression regulation, stem cells, Rett syndrome.

ABOUT US

RESEARCH GROUPS

EPIGENETIC CONTROL OF HEMATOPOIESIS LED BY JOSÉ LUIS SARDINA

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OVERVIEW

DNA methylation-related genes are among the most frequently mutated in blood malignancies. Traditionally, studies aimed at understanding the effect of aberrant DNA methylation in cancer patients have focused on gene promoters. However, recent findings focus on enhancers as the most important regions in dynamic DNA methylation studies. We aim to understand how aberrant DNA methylation dynamics impact on the chromatin structure at enhancers during blood cancer onset and progression.

OUR RESEARCH

We study how aberrant DNA methylation at distal gene regulatory regions poisons the chromatin to trigger corrupted gene expression signatures in cells, thus eventually leading to the onset and progression of hematopoiesis neoplasms. This line of research has implications for a broad spectrum of patients suffering from blood diseases sharing an abnormal genome-wide DNA methylation landscape.

OUR GOALS

We aim to unravel the different layers of intricated epigenetic information that specify which subsets of genes are expressed in every one of the cells of the hematopoietic system, thereby defining their cellular identity. We hope to apply this knowledge to better understand how and when deleterious transcriptional programs leading to cellular transformation are activated, thus leading to the discovery of new treatments that will potentially end up in improving the quality of life of patients suffering from a wide range of blood diseases.

OUR CHALLENGES

There is an urgent need for novel therapies for acute myeloid leukemia, since barely any drugs introduced in the last decades have increased the overall survival its patients. Hence, our research aims to shed light on the following questions:

1. What is the interplay between DNA (hydroxy) methylation and chromatin dynamics at distal gene regulatory regions during hematopoietic cell fate decisions?

2. What are the molecular mechanisms underlying the role of TET2 in the epigenetic control of the chromatin at distal gene regulatory regions during leukemia onset and progression?

3. What is the role of mRNA methylation-mediated post-transcriptional control in myeloid cell differentiation?

KEYWORDS

NA methylation; TET enzymes; Chromatin; Hematopoiesis malignancies; Stem cells

ABOUT US

RESEARCH GROUPS

TRANSCRIPTIONAL DYNAMICS IN LEUKEMIA LED BY SERGI CUARTERO

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OVERVIEW

Hematopoietic differentiation is a tightly regulated process that maintains blood production throughout life. The transcriptional changes that hematopoietic cells undergo during differentiation are controlled at multiple levels and an accurate integration of all of them is essential to ensure the production of sufficient numbers of blood cells at all stages of differentiation. However, most of the acute myeloid leukemia (AML) cases have mutations in transcriptional regulators and chromatin modifiers. These mutations alter transcriptional dynamics and can impair normal differentiation.

OUR RESEARCH

We study the mechanisms that regulate transcription during hematopoietic differentiation and investigate the leukemogenic potential of mutations in transcriptional regulators and epigenetic modifiers. We are also looking into the role of mutations in proteins that drive the three-dimensional organization of the genome.

OUR GOALS

Our main goals are:

1.

To understand the role of mutations in hematopoietic transcription factors and chromatin regulators in acute myeloid leukemia (AML).

Using genetic models to mimic these mutations, we aim to dissect their impact on gene expression and thus understand how they promote a selective advantage.

2.

To characterize the impact of inflammatory signalling on normal hematopoietic differentiation and during leukemic progression.

We want to understand the impact of inflammation on the progression of myeloid malignancies and how are they linked to the most common mutations.

OUR CHALLENGES

Acute myeloid leukemia (AML) is the one of the most aggressive forms of leukemia, and there is an urgent need to find new treatment options. While we now know **what** genes are recurrently mutated in AML, we still do not understand **why** these mutations are malignant. Through our research, we aim to answer the following questions:

1. What transcriptional mechanisms are deregulated in acute myeloid leukemia?
2. How do inflammatory signals influence leukemic progression?
3. Can we use inflammatory modulation to attenuate the severity of myeloid malignancies?

KEYWORDS

Hematopoiesis, chromatin, AML, MDS, cohesin, inflammation

ABOUT US

RESEARCH GROUPS

CANCER IMMUNOGENOMICS LED BY EDUARD PORTA

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OVERVIEW

Our research lies at the interface of artificial intelligence, molecular biology and medical oncology, and we bring together experts from all three fields. We use computational approaches to study the interaction between genetic variants in cancer genomes and multiple aspects of cancer, ranging from the immune response against tumors to the susceptibility of cancer cells to different treatments.

OUR RESEARCH

Over the last two decades, there has been an explosion of three main types of big data in cancer research. We use three types of data:

- Germline genotypes from cancer patients, this is, the base non-altered genome of individual people being diagnosed with cancer.
- Somatic tumor genomes, being the particular genomes of individual tumors, with its unique set of alterations.
- The amount and composition of cells in tumors coming from single-cell sequencing.

Until now, these three different aspects of tumor immunobiology have mostly been studied on an individual basis. However, it is now evident that the three factors are inextricably linked and should be studied as a whole.

OUR GOALS

Our main goal is to understand how genetic variation influences the immune response against cancer cells and vice versa. Specifically, we are working on the following lines:

1.
Understanding how inherited genetic variants change the immune response against cancer cells.

2.
Understanding how inherited genetic variants interact with biological sex to influence cancer predisposition. We are using the MareNostrum supercomputer to analyze genetic data from hundreds of thousands of cancer patients from this biological sex perspective, to identify genetic variants that predispose to cancer differently depending on gender.

3.
Integration of protein structure and genetic data to identify new cancer-associated mutations.

4.
Creating a molecular and cellular map of the tumor microenvironment in bladder cancer.

OUR CHALLENGES

Through our research, we hope to answer the following questions:

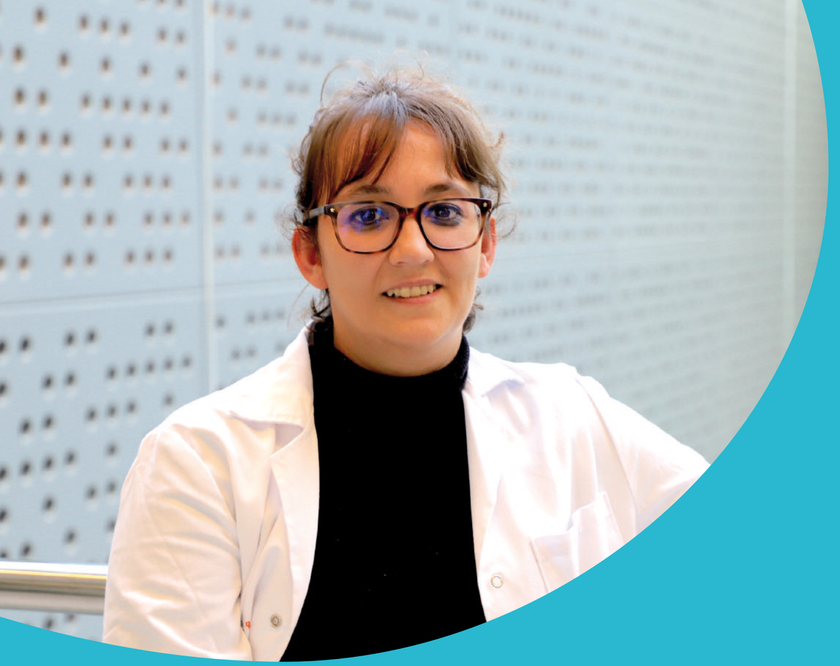
1
Is it possible to use a person's genetic data to predict whether he/she will develop cancer?

2
Which genes play a role in the development of cancer?

3
How do genetic variants change the immune response against cancer cells?

KEYWORDS

Computational biology, cancer genomics, big data, GWAS, bladder cancer



ABOUT US

RESEARCH GROUPS

CANCER HETEROGENEITY AND HIERARCHIES LED BY VERÓNICA RODILLA

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OVERVIEW

Our laboratory studies the key signals governing stem cell and cell fate specification during malignant progression and the mechanisms by which different signaling pathways control cell plasticity in cancer. Specifically, we use in vivo lineage tracing, live imaging, cytometry and expression profile analysis as experimental tools to achieve our goals. Our group combines murine transgenic models, patient-derived xenografts and 3D organoids to unravel cellular hierarchies within tumors, to gain a better understanding of cancer heterogeneity and drug resistance.

OUR RESEARCH

Cancer is a heterogeneous disease with a cellular hierarchical organization that is largely unexplored in many tumor subtypes. Moreover, in some cases hierarchical relationships among stem cells, progenitors and differentiated cells remain unsolved due to the high degree of cellular plasticity, which allows cells to switch between different cellular stages.

OUR GOALS

We are a newly created group passionate about cellular hierarchies and tumor heterogeneity. Our main lines of research and specific goals are:

1.
To illustrate cellular hierarchies within tumors.

We use a well-established hierarchical model to study multipotency in tumors. Now, we are separately monitoring three mammary epithelial compartments to measure the presence of multipotency within breast tumors. Our hypothesis is that breast tumors.

2.
To discover cytotoxic agents for specific cellular subpopulations.

A therapy based on a combination of several drugs to target different cellular populations could eradicate primary tumors, thereby preventing relapse and metastasis. We want to screen for natural compounds that selectively kill specific subsets of cells that are responsible for tumor maintenance and/or intrinsically resistant to current therapies.

3.
To target the tumor niche to prevent the spread of cancer.

One of our main objectives is to generate in vivo tools that will allow us to study new therapeutic targets to prevent relapses in hematological cancer. To that end, our lab works on different strategies, which include murine and human models, to test a panel of drugs currently used as a standard of care for non-Hodgkin's lymphoma (NHL) and explore the role of senescence in tumors cells, as well as in their microenvironment.

Our ultimate mission is to understand the tumor heterogeneity between different patients with a view to improving their treatment of choice by searching for novel and personalized therapeutic strategies.

RESEARCH GROUPS

OUR CHALLENGES

We hope to answer the following questions through our research:

1. How can cellular plasticity improve treatment for cancer patients?
2. Can we achieve truly personalized medicine by identifying single or combinatorial therapies to target different cellular populations at the same time?
3. Can we prevent metastasis and/or relapses by targeting the most frequently colonized tissues?

KEYWORDS

Cellular hierarchies, heterogeneity, cell plasticity, senescence, tumor microenvironment

ABOUT US

RESEARCH GROUPS

LEUKEMIA AND IMMUNO-ONCOLOGY LED BY LAURA BELVER

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OVERVIEW

Juvenile myelomonocytic leukemia (JMML) is a rare and aggressive type of pediatric blood cancer that affects mainly children under two years of age and occurs when bone marrow production of white cells becomes severely dysregulated. Hematopoietic stem cell transplantation (HSCT) is currently the only available treatment for patients, although just two out of three children affected by the disease survive.

OUR RESEARCH

Since 2020, our work has focused on the study of the molecular mechanisms driving JMML and the exploration of alternative therapeutic strategies specifically designed for these patients. To achieve this, we incorporate different methods into epigenetics, systems biology, functional genomics and biochemistry, to help address critical questions about the origin and progression of JMML and to identify new therapeutic targets for the treatment of this disease.

OUR GOALS

The specific goals of our research program are as follows:

1.

To create a centralized JMML sample repository and patient-derived xenograft (PDX) collection.

In collaboration with clinical teams at different hospitals around the country and with the endorsement of the Spanish Society of Pediatric Hematology and Oncology (SEHOP), we are creating a national JMML patient sample repository and PDX collection, that will be instrumental for the development of our research program.

2.

To develop a comprehensive molecular analysis of JMML patients to define accurate diagnostic and stratification criteria.

We aim to explore the non-coding genome of JMML patients to identify new genetic alterations that can drive JMML or contribute to the pathogenesis of the disease by other means. This information will help improve our knowledge of JMML and develop more accurate criteria for the diagnosis and management of JMML patients.

3.

To identify new potential therapeutic targets and develop specific therapies for the treatment of JMML.

HSCT is currently the only effective therapy for the treatment of JMML. However, only two out of three children with this disease survive. Thus, new therapies specifically designed to treat JMML patients are needed. To achieve this, we are collaborating with other research groups and with biotechnology companies to identify specific JMML therapeutic targets and explore strategies for their clinical use for the treatment of JMML patients.

We are confident that our results will have an important impact on the diagnosis and treatment of JMML by increasing knowledge of the disease and expanding the therapeutic options open to these patients. Moreover, our close collaboration with clinical teams in the development of this project will guarantee that our most promising discoveries have a rapid and direct impact on JMML patients.

OUR CHALLENGES

We hope to answer the following questions through our research:

1. What is the relevance of non-coding somatic mutations in the generation and development of JMML?
2. Can non-coding mutations predict the prognosis of JMML patients?
3. What are the best therapeutic targets for the development of JMML-specific treatments?

KEYWORDS

Leukemia, JMML, PTPN11, experimental therapeutics, CAR-T cells, rare diseases, pediatric diseases, sequencing, diagnosis, therapeutic targets, preclinical models, drug discovery

ABOUT US

RESEARCH GROUPS

CELLULAR SYSTEMS GENOMICS LED BY ELISABETTA MEREU

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OVERVIEW

In the interface between genomics, digital pathology and artificial intelligence the **Cellular Systems Genomics** group aims to define the spatiotemporal organization of complex tissues in health and disease, by the identification of key regulatory mechanisms driving heterogeneity in cellular identity and function, particularly in the context of inflammation, inflammatory disorders and autoimmune diseases.

To address these questions, we will adopt a single-cell perspective, enabling the fine-grained and spatially resolved molecular profiling of tissues. We will develop new machine learning approaches and open source tools in order to unlock molecular mechanisms hidden in large-scale datasets. In a short-term perspective, these methods will help understand disease mechanisms, allowing the stratification of patients based on their molecular and cellular characteristics, ultimately providing new therapeutic targets for their treatments.

OUR RESEARCH

Single cell sequencing allows to profile thousands of individual cells per experiment, enabling the unbiased analysis of tissues, organs and even entire organisms at an unprecedented resolution. These data represent a powerful tool for cell biology, with relevant clinical applications including diagnosis and treatment of diseases. Despite the many advantages of this approach, data are noisy and sparse, making the computational analysis challenging. To address these challenges, we apply machine learning and other statistical methods to develop new analytical frameworks and open source tools to analyze, interpret and integrate data coming from single-cell and spatial genomics experiments.

As part of the Human Cell Atlas (HCA) consortium, which aims to create a catalogue of all cell types in our body, we have extensive experience on the systematic comparison of protocols in single cell RNA sequencing (scRNA-seq). In conjunction with the new Single Cell Unit of the Institute, which is equipped with the Chromium controller to perform the single-cell analysis, we will provide support to design new experiments and generate high-quality data and computational analysis.

Beyond transcriptomic profiling with scRNA-seq, different cellular modalities can now be measured, including single-cell epigenetics (scATAC-seq), spatial transcriptomics as well as the joint profiling of chromatin accessibility and transcription on the same cell.

However, the integration of multimodal data poses new analytical challenges and new benchmarking are needed to assess reproducibility and integrity of these methods. We are working on new mathematical frameworks for the integration of multimodal data, enabling the comprehensive characterization of cells in their identity and function.

OUR GOALS

In the European Pancreas Atlas consortium (ESPACE, https://www.espace-h2020.eu), we are working to build a first version of the Human Cell Atlas of the Pancreas, by profiling the transcriptome and epigenome of cells from distinct anatomical regions of the adult pancreas. The integration of distinct single-cell and spatial data types will allow the comprehensive transcriptional and epigenetic landscape of pancreas cell types within their spatial context.

Our experience in single-cell data analysis on healthy and diseased tissues allowed us to build a deep understanding of cell-type structure and plasticity in different research contexts. To accelerate biological discovery and advance science, our group will share user-friendly computational solutions, by promoting open science, diversity and supporting an inclusive and collaborative environment. We welcome proposals for interdisciplinary research collaborations, from both industry and academia.

KEYWORDS

Genomics, inflammation, autoimmunity, single cell, machine learning, computational analysis

ABOUT US

RESEARCH GROUPS

STEM CELLS AND CANCER LED BY ANNA BIGAS

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OVERVIEW

Our research group investigates how to generate and maintain the stem cells in the hematopoietic system under physiological conditions but also how these processes are mimicked by the tumors for their perpetuation. We constantly improve our research by implementing novel technology to understand the process of normal and malignant hematopoietic development. Our research includes basic studies at the molecular level to understand cellular processes in the context of mouse models and human patients.

OUR RESEARCH

Our research comprises from basic biochemical research to the analysis of animal models that reproduce the pathologies of interest and allow us to study the functional relevance of new hypothesis. The ultimate goal is to confirm the importance of the findings and study possible therapeutic applications through the analysis of patient samples. In this sense we have devoted special efforts to understand the regulation of hematopoietic stem cells, as a tool to understand the mechanisms that regulate leukemia initiation and maintenance.

OUR GOALS

The specific goals of our research program are as follows:

1.

Generation of hematopoietic stem cells.

Our current studies are focused on understanding the signals that the embryo uses to form these self-renewing cells that maintain the hematopoietic system throughout the life of the organism.

2.

Understanding T Acute Lymphoblastic Leukemia (T-ALL) development and T-cell lymphoma.

We study the signals that regulate the generation and maintenance of normal and leukemic cells, as well as leukemic stem cells (LSCS). With this aim we have developed in vitro and in vivo experimental models that complement the analysis of patient samples.

3.

GATA2 deficiency syndrome.

We are collaborating in an international consortium to understand the contribution of GATA2 mutations to pediatric Myelodysplastic syndrome and transformation to Acute Myeloid Leukemia (AML). We are developing humanized blood animal models of this syndrome.

4.

Understanding cell transformation.

We work closely with the Research Group for Molecular Mechanisms of Cancer and Stemness directed by Dr. Lluís Espinosa, and we take advantage of our discoveries in hematopoietic cells to understand epithelial tissues and vice versa.

OUR CHALLENGES

Through our research, we aim to understand the following:

1. What signals are imposed in embryonic HSCs that affect the adult hematopoietic system?
2. What are the molecular mechanisms that impose resistance to treatment in T-ALL cells?
3. What are the basic mechanisms that control cell transformation?

KEYWORDS

Embryonic hematopoiesis, T-ALL, CTCL, GATA2, Notch, NFkB, hematopoietic stem cell, leukemic stem cells

ABOUT US

RESEARCH GROUPS

ENDOTHELIAL PATHOBIOLOGY AND MICROENVIRONMENT LED BY MARIONA GRAUPERA

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OVERVIEW

Our research is devoted to study the biology of the endothelium and its role in disease towards the development of therapeutic strategies to target this compartment. Specifically, we aim to untackel the fundamental insigths of vessel growth and function in developmental setting as well as to identify the pathological contexts in which the vasculature plays a critical role either intrinsically, as in vascular anomalies, or extrinsically as in cancer.

OUR RESEARCH

Blood vessels are crucial components of every organ, as they maintain tissue homeostasis by ensuring: (i) transport of gases, nutrients, waste products and circulating cells, (ii) blood coagulation, and (iii) vascular tone and barrier. The endothelium lines the lumen of blood vessels and regulates the dynamic passage of materials and cells, whereas mural cells adhere to the abluminal surface of the endothelium and regulate vessel growth, permability and function. Both excessive and insufficient vascular network is deleterious for organisms and lead to a broad spectrum of pathologies. The overall aim of the Graupera lab is to understand the mechanisms that regulate the vasculature in development, homeostasis and disease. Most of our research has focused on the the endothelium that plays an active role in important physiological processes and diseases such congenital disorders, obesity and cancer. Over the past decade, we have taken advantage of the PI3K pathway as a paradigm to understand how intracellular roads regulate vessel morphogenesis, and how this knowledge can be translated into therapeutic opportunities for diseases with aberrant angiogenesis. For our research, our lab develops unique animal models including, established cell lines, and patient-derived samples. We apply a holistic approach utilising state-of-the-art techniques as high-throughput analysis, next-generation sequencing, single cell RNA sequencing, phospho/proteomics, and high-resolution imaging. Our lab closely collaborates with clinicians to translate our research into the clinic at both the diagnostic and therapeutic levels.

RESEARCH GROUPS

OUR GOALS

The Graupera lab is devoted to 5 main research lines:

1.
Insights on developmental vessel growth and function.

2.
Understanding oncoproteins-related developmental disorders.

3.
To study tumor-stroma interaction.

4.
Identify vascular therapies to treat metabolic disorders.

5.
To study endothelial and hematopoietic cell interface.

KEYWORDS

Endothelium, vascular compartment, homeostasis, next generation sequencing, single cell, high-resolution imaging

ABOUT US

RESEARCH GROUPS

T-CELL LYMPHOMA LED BY LAURA MONDRAGÓN

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OVERVIEW

Our research is focused on the better understanding of the molecular mechanisms leading to T cell lymphomas appearance. We will develop our research by determining possible defective mechanisms during thymopoiesis and, by developing preclinical mice models for the study of T cell lymphomas, such as angioimmunoblastic T cell lymphoma.

With this knowledge we expect to design and validate new therapeutic treatments more specific and effective than the ones currently available in order to improve patient's survival and quality of life.

OUR RESEARCH

T cell lymphomas can be defined as a group of malignancies caused by the uncontrolled proliferation of T cells. They constitute less than 15% of all Non-Hodgkin's lymphomas and within this group frequency can vary enormously. Despite all being caused by T cell defective cell growth little is known about its specific origin. Besides, they present a wide variety of symptoms and clinical characteristics ranging from highly aggressive (fast-growing) lymphomas to subtypes that can develop for years without endangering the patient's life (indolent); presence of enlarged spleen, liver and/or lymph nodes; eczema and skin rash appearance; age appearance and higher incidence in men than in women. As a result, it is often difficult to establish a correct diagnosis of the disease and even more difficult to design an appropriate therapy for its specific treatment.

In this sense, our line of research aims at improving our understanding of the molecular mechanisms leading to the defective behaviour of the T cells originating this type of lymphoma. To develop our objective we will employ immunology, functional genomics, molecular biology and medicinal chemistry techniques.

Specifically, we will make use of potential and already described genetically modified mice models as pre-clinical models for the study of different subtypes of T cell lymphomas and we will:

- Characterize their phenotype once the disease is developed in order to find the specific T cell population inducing its appearance.
- Study thymocytes maturation processes and mature T cells response to antigens to try to determine if lymphoma appearance can be already settled during thymopoiesis or once the T cells leave the thymus.
- Compare by genomic techniques like single cell sequencing the characteristics of defective thymocytes and T cells in mice models and try to find similarities in human patient's samples and databases.

With the information obtained, we aim at finding new therapeutic targets in order to:

- Develop chemical libraries screening assays for drug discovery to modulate the activity of these therapeutic targets.
- Perform structure-activity assays and possible administration in form of nanomedicines to optimize their biological activity and cellular uptake in vitro.
- Validate their use as new therapeutic strategies alone or in combination with other chemotherapeutics employing mice models of the disease.

OUR GOALS

To make available new therapies to treat angioimmunoblastic T cell lymphoma and reduce mortality in those patients. The most important one would be to improve life expectancy of patient's suffering from this type of disease. Although, we would like to apply our research to different types of T cell lymphomas, we are initially focused on the study of molecular mechanisms leading to angioimmunoblastic T cell lymphoma. This type of disease has no specific treatment and all the strategies chosen so far have not improved patient's survival in the last 3 decades. Finding new strategies will for sure improve their chances to recover from this disease and it will significantly improve their quality of life. To unveil the molecular mechanisms leading to T cell lymphoma appearance and to provide new therapeutic targets to design more specific and effective therapeutic treatments to fight these group of hematopoiesis diseases.

OUR CHALLENGES

In summary, our line of research will have the final objective to provide more specific and effective therapies to treat T cell lymphoma in order to find a cure or, if not, to improve the prognosis and quality of life of patients.

- Disease Knowledge deepening
- Drug development

There are some questions we are trying to answer with our research:

1. When does defects in T cells leading to T cell lymphoma appearance start?
2. Which are the specific T cell populations responsible for T cell lymphoma induction?
3. Can we design more specific and effective therapeutic treatments for this type of disease?

KEYWORDS

thymopoiesis, t cell lineage selection, T cell receptor, T cell activation, lymphoma

ABOUT US

RESEARCH GROUPS

NUCLEAR ARCHITECTURE IN LEUKEMIA LED BY GREGOIRE STIK

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OVERVIEW

The main goal of our lab is to understand the molecular mechanisms that induce and control the malignancy of leukemic cells. For that, we combine and integrate state-of-the-art genomics technology, genome-engineering tools, optogenetic and advanced microscopy imaging to study gene regulatory network in human leukemic cells.

We focus particularly in the role of the three-dimensional (3D) genome organization in leukemic phenotype and how fusion protein induced by chromosomal translocation can alter the chromatin organization. Beyond our fundamental discoveries, we aim to uncover new targets and biomedical applications for the treatment of lymphoid malignancies.

OUR RESEARCH

Understanding the mechanisms that control cell identity and gene regulation and whether they can be used therapeutically are fundamental objectives of current biomedical science. Indeed, the precise regulation of gene expression is crucial to guarantee tissue homeostasis and its alteration drives cell disorders and diseases. In addition to transcription factors and chromatin modifiers, the 3D genome organization has recently emerged as an instrumental player of gene regulation.

OUR GOALS

Important efforts have been made to define the basis of acute lymphoblastic leukemia (ALL) and identify the genetic lesions contributing to leukemogenesis. The most common mutations affect transcription factors or chromatin modifiers. Nonetheless, chromosomal translocations that create chimeric transcription factors are often associated also to ALL. These mutations may alter the protein function, modify the transcriptional program and initiate leukemogenesis.

More specifically, the research in our lab develops around the following axes:

1.

Uncovering the biophysical properties of the chimeric E2A-PBX1 oncogene and its role on 3D genome alteration and pathogenesis of B cell acute leukemia

We are developing new research lines to explore the molecular mechanisms driving nuclear organization of cancer cells, focusing on chimeric transcription factors generated by chromosomal translocation and its impact on 3D genome organization and pathogenesis.

2.

Identification and characterization of genome topology alteration in B cell acute lymphoblastic leukemia

Our lab uses a unique model of "cell normalization" of human leukemic cells via transcription factor-mediated transdifferentiation. This process leads to a rewiring of the gene expression pattern, including several oncogenes. We employ genomics technologies on leukemic cells undergoing a conversion to non-tumorigenic macrophages to study the dynamical interplay between oncogenes expression and key epigenetic regulatory mechanisms, including genome topology.

3.

Characterization of transcription factor mutations and their role in 3D genome organization alteration and leukemogenesis

We focus on mutation altering domains essential to the biochemical properties of transcription factors (TFs) and evaluate how these mutations affect their properties and the ability to shape the genome. We aim to precisely profile the aberrant function of TFs and link it to the altered gene regulatory network observed in the disease.

OUR CHALLENGES

The genome is highly organized in the nucleus into various structures including compartments, domains and loops. These structures are crucial to maintain the physical interactions between regulatory regions and gene expression. The comprehensive integration of the 3D genome organization with other layers of the gene regulatory network is therefore crucial to uncover the molecular mechanisms beyond the disease and identify new potential therapeutic targets.

KEYWORDS

Genomics, 3D genome organisation, Acute Lymphoblastic Leukemia, Transcription Factors, Translocations

ABOUT US

RESEARCH GROUPS

ACUTE LYMPHOBLASTIC LEUKEMIA (ALL) LED BY JOSEP Mª RIBERA

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OVERVIEW

Our research focuses on analyzing the genomic and epigenomic landscape of patients with adult ALL (acute lymphoblastic leukemia) to find out genetic alterations that predict patients' response to treatment and to identify new alternative (targeted) therapies to apply to those patients. In this way, we aim to design more personalized treatments to increase the probability of survival of ALL patients.

OUR RESEARCH

The group's current research is divided into two main areas, according to the two main subtypes distinguished in ALL:

- **Precursor B-cell acute lymphoblastic leukemia (BCP-ALL):** BCP-ALL is the most prevalent ALL subtype and accounts for 75% of ALL cases. Although it is a highly heterogeneous disease at genetic level, different cytogenetic subtypes have been identified and, more importantly, their prognosis has been clearly established in many clinical trials. This has allowed clinicians to stratify patients according to their genetic profile to schedule intensive or less intensive treatments.
- **T-cell acute lymphoblastic leukemia (T-ALL):** T-ALL is the least common ALL subtype (25% of adult ALL cases), and the most complex and heterogeneous at genetic level, with a dismal prognosis. to improve the survival rate of patients with T-ALL, we first need to obtain detailed and relevant molecular information to accurately define the risk and thus decide on the treatment.

OUR GOALS

We are convinced that new treatments for ALL patients can be obtained only through basic research. Therefore, our goals are:

1.
To identify the genetic alterations leading to treatment resistance and disease recurrence in adult ALL.

2.
To accurately define the risk of ALL by genetic analysis at diagnosis and relapse in order to decide on the most appropriate treatment.

OUR CHALLENGES

Although ALL is a rare form of cancer, it has a huge impact on patients, their relatives and the health system. To find new therapies and provide new knowledge, our research hopes to:

1. Decipher the genetic complexity of ALL at both diagnosis and relapse.
2. Identify critical genetic lesions in ALL cells that could be targetable with new drugs.

KEYWORDS

Acute lymphoblastic leukemia, adults, genomic analyses, minimal residual disease, treatment resistance

ABOUT US

RESEARCH GROUPS

BARCELONA ENDOTHELIUM TEAM (BET) LED BY ENRIC CARRERAS

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OVERVIEW

Blood vessel endothelium crosses each and every tissue and presents unique structural and functional properties in each vascular bed. This cellular heterogeneity is reflected by the structural and functional heterogeneity of the whole endothelium as a complex system. Due to its location, the endothelium is exposed to all kinds of physiological and pathological stimuli and constitutes the first barrier to many drug interventions.

This organ has the capacity to constantly adapt to environmental changes by modulating vasomotor tone, haemostatic balance and inflammatory reactions, among other responses. Endothelial activation could lead to an irreversible state known as endothelial dysfunction that, in a broad sense, leads to various non-adaptive alterations of the functional phenotype that constitute a net liability to the host.

OUR RESEARCH

Our group has extensive experience in the study of the endothelial dysfunction that develops in association with different vascular pathologies, such as the early complications associated with hematopoietic cell transplantation, obesity, chronic kidney disease, thrombotic microangiopathies and sepsis.

We also explore strategies for the protection of this endothelial dysfunction to improve patient health. In this regard, one of our main interests is to evaluate different compounds that potentially exhibit the capacity to protect the endothelium and to decipher their mechanisms of action.

OUR GOALS

Our main lines of research are:

1.
o characterize the endothelial activation and dysfunction associated with cardiometabolic diseases through in vitro models.

2.
To elucidate the mechanisms that lead to endothelial dysfunction.

3.
To investigate agents with potential protective effects on the endothelium to prevent complications.

4.
To find soluble markers with prognostic and diagnostic value for vascular complications.

5.
To study complement pathways and complement deficiencies in thrombotic microangiopathies.

6.
To assess platelet physiology and alterations of haemostasis by using perfusion devices to explore adhesive and cohesive properties of platelets under flow conditions.

OUR CHALLENGES

Hematopoietic cell transplantation (HCT) has been the major curative therapy for several hematological, metabolic and neoplastic disorders. However, the efficacy of this procedure is limited by life-threatening complications, the most important of which is graft versus host disease (GvHD), which has a high mortality rate. Through our research, we aim to answer the following questions:

1
What are the pathophysiologic mechanisms that characterize endothelial dysfunction?

2
How can we avoid the vascular complications associated with hematopoietic cell transplantation?

3
Which is the role of the complement system in vascular complications?

KEYWORDS

Endothelium, Inflammation, Diagnostic and prognostic markers, Thrombotic microangiopathies (TMA), Drugs

ABOUT US

RESEARCH GROUPS

MYELOID NEOPLASMS
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OVERVIEW

Genetic profiling for hematopoiesis malignancies involves chasing a moving target. Not so long ago, leukemias were stratified based on karyotype abnormalities. In recent years, however, knowledge of molecular genetics in hematology has increased significantly, thus offering new clinical opportunities. It has now been shown that gene expression, mutations and other genetic and epigenetic abnormalities also have diagnostic, prognostic and therapeutic implications.

OUR RESEARCH

Since 2004, our group has been studying MN, with a particular focus on characterizing genetic and epigenetic lesions to find new diagnostic, prognostic and therapeutic markers that could help us better diagnose and treat patients with these diseases. First, we started with karyotype and single nucleotide polymorphism arrays (SNP-A) to help us detect alterations at chromosome level, and we are currently performing studies at gene level (mutational profile studies) and analysing the impact that telomere size could have on the development of the disease.

OUR GOALS

The aim of our group is to apply our research to myeloid neoplasms (MN), specifically three distinct hematopoiesis diseases: chronic myelomonocytic leukemia, myelodysplastic syndromes and myeloproliferative neoplasms (PV, ET, MFP and CML), with a view to finding better tools for diagnosis and prognosis stratification and achieving individualized targeted therapies (personalized medicine). Therefore, our research focuses mainly on the following areas:

1.
Chronic myelomonocytic leukemia (CMML).

The aim of our research is to characterize the type, frequency and prognostic impact of mutations and cytogenetic alterations detected by SNP arrays in patients with low-risk CMML and, then, study its epigenetic changes (DNA methylation and miRNAs expression).

2.
The classification and prognosis of the group of diseases termed myelodysplastic syndromes (MDS).

This knowledge will also contribute to a better understanding of MDS biology and a better stratification of the prognosis of these patients, which would also help with the selection of the most appropriate treatment for each one.

3.
Chronic myeloid leukemia (CML).

The aim of our research is to determine whether or not we can find any genetic marker at CML diagnosis that could explain a patient's toxicity to tyrosine kinase inhibitors, or identify which patients will achieve a molecular response. We are also seeking a technique with higher sensitivity than QRT-PCR.

4.
BCR-ABL1 negative classic myeloproliferative neoplasms (MPNs).

We study several genomic changes in an attempt to associate them with cytological subtypes, laboratory parameters, clinical complications and probability of transformation to either MF or AML.

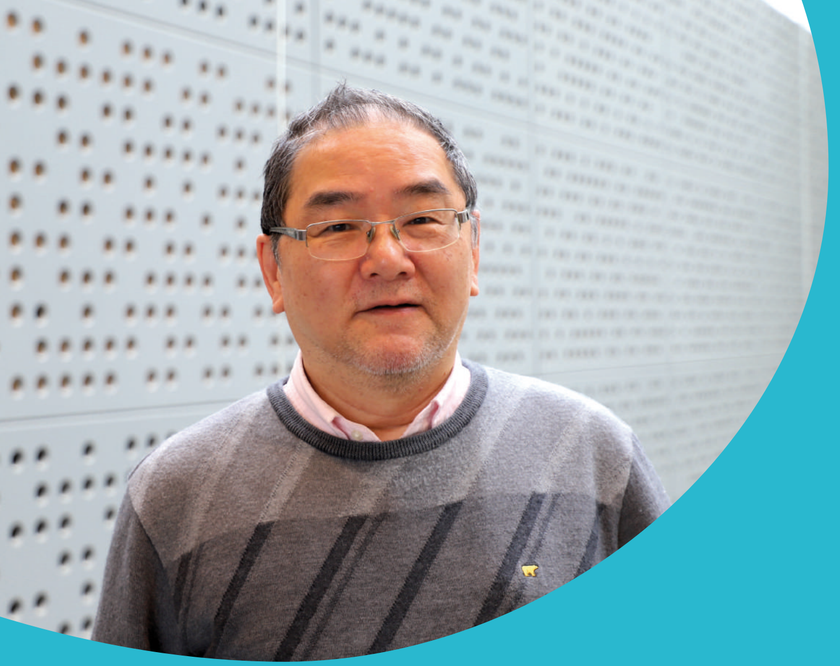
OUR CHALLENGES

Our research is highly socially relevant because we promote capacity building, advancing knowledge, help in making informed decisions and improve the health in general terms, with economic benefits for the whole society. Through our research, we aim to answer the following questions:

1. How can we improve diagnosis, prognosis stratification, treatment response and life expectancy in chronic myelomonocytic leukemia?
2. How can we improve diagnosis, prognosis stratification, treatment response and life expectancy in myelodysplastic syndromes?
3. How can we improve diagnosis, prognosis stratification, treatment response and life expectancy in myeloproliferative neoplasms?

KEYWORDS

Myeloproliferative neoplasms, chronic myeloid leukemia, myelodysplastic syndromes, MPN/MDS, acute myeloid leukemia



ABOUT US

RESEARCH GROUPS

IMMUNOHEMATOLOGY AND GLYCOBIOLOGY LED BY FUMIICHIRO YAMAMOTO

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OVERVIEW

The ABO blood group system is one of the most important blood group systems in transfusion medicine. The ABO polymorphism consists of A and B glycan antigens on red blood cells (RBC) and antibodies against A and B antigens in the sera of individuals who do not express these antigens. In humans, A and B antigens are also present on epithelial and endothelial cells, depending on the ABO genotype/phenotype of the individual. Because of this, ABO matching is also crucial in cell, tissue and organ transplantation.

OUR RESEARCH

We study the molecular genetic mechanisms for the expression of genetically incompatible glycan antigens and have thus far revealed several potential mechanisms, including the appearance of FORS1 induced by the deletion of exon 3 or 4 of the AT mRNA. Because altered splicing is a hallmark of cancer, this mechanism may be responsible, at least partially, for FORS1 expression in group A and AB individuals.

We also investigate the potential mechanism by which incompatible A antigens appear in group O individuals through complementation by recombination of DNA or trans-splicing of RNA and also expression of FORS1 due to changes in specificity resulting from incorrect intra-Golgi localization of modified glycosyltransferases.

OUR GOALS

Cancer growth indicates that the cancer cell-killing activities of natural immunity against genetically incompatible and/or cryptic glycans are ineffective and insufficient. However, they can be improved through active and/or passive immunization. Therefore, our goals are:

1.
To investigate the use of genetically incompatible and/or cryptic glycan antigens as molecular targets for medical intervention.

2.
To explore the possibility of using forced expression of genetically incompatible glycans to make cancer cells susceptible to natural immunity.

OUR CHALLENGES

If successful, the active immunization we advocate for could revolutionize the cancer treatment landscape, thereby dramatically reducing the financial burdens on patients and society. Through our research, we aim to answer the following questions:

1
What is the molecular genetic/epigenetic basis of glycan alterations in cancer?

2
Can we use cancer-specific glycans as molecular targets for cancer detection and immunotherapy?.

3
Does the minitransfusion/injection of mismatched red blood cells expressing genetically incompatible and/or cryptic glycans improve humoural and cellular immunity against cancer cells expressing cancer-specific glycans?

KEYWORDS

Genetically incompatible glycan antigens, cryptic glycan antigens, cancer immunotherapy, disease susceptibility, ABO polymorphism

ABOUT US

RESEARCH GROUPS

LEUKEMIA STEM CELL LED BY RUTH RISUEÑO*

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OVERVIEW

Many leukemias originate in a population of leukemic stem cells, which are responsible for initiating and maintaining the disease. Our group investigates the biology and sensitivity of stem cells that sustain tumors such as acute myeloid leukemia (AML), myelodysplastic syndromes (MDS), chronic myelomonocytic leukemia (CMML) and acute lymphoblastic leukemia T (ALL-T).

OUR RESEARCH

Our work focuses on searching new therapeutic targets for blood disorders enabling us to identify new biomarkers that can be used for prognostic and/or diagnostic purposes, thereby gaining greater insight into the biology behind leukemic processes. New knowledge will help us develop new drugs that specifically attack the population of leukemic stem cells.

Due to the similarities between leukemic stem cells and healthy hematopoietic stem cells, our research group is striving to develop therapies that trigger the terminal differentiation of the population of leukemic stem cells, which eliminates their capacity to initiate and maintain the disease and enhances its chemosensitivity.

OUR GOALS

Our main goal is to understand the biology of leukemic stem cells and identify pharmacological mechanisms that will enable us to modulate their functionality and eliminate them selectively. To achieve this, we adopt a dual approach: on the one hand, we study the underlying biological mechanisms responsible for these neoplasms and, on the other, we develop new therapeutic approaches with the potential for clinical application.

* The Leukemia Stem Cell Group left the Josep Carreras Institute on October 31, 2022

OUR CHALLENGES

Due to the biological properties of leukemic stem cells, understanding how they work and identifying the differences between these and healthy hematopoietic stem cells is essential to design new more efficient and selective therapies that can be personalized for each patient. Therefore, our research aims to:

1. Understand how leukemic stem cells work.
2. Identify therapeutic targets and develop new drugs for leukemic stem cells.
3. Determine the prognostic and diagnostic value of these therapeutic targets as biomarkers for leukemia.
4. Describe the differences between leukemic stem cells and healthy haemopoietic stem cells.

KEYWORDS

Leukemia, leukemic stem cell, drug development, hematopoiesis, differentiation therapies

ABOUT US

RESEARCH GROUPS

LYMPHOID NEOPLASMS LED BY TOMÁS NAVARRO

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OVERVIEW

Our research is focused on the study of rare lymphomas, such as those that affect immunosuppressed patients, in terms of both clinical and genetic aspects. We have made important contributions to this field and our current objective is to reveal genetic and epigenetic characteristics of lymphoid neoplasms that occur mainly in immunosuppressed individuals. The purpose is to identify markers to improve the accuracy with which these patients are managed. Furthermore, we aim to implement liquid biopsy as a tool for diagnosis and follow-up of aggressive lymphomas.

OUR RESEARCH

Our group focuses mainly on the research of AIDS-related lymphomas (ARLs). The most frequent ARLs are diffuse large B-cell lymphoma (DLBCL), Burkitt lymphoma (BL) and Hodgkin lymphoma (HL). Plasmablastic lymphoma (PBL) and primary effusion lymphoma (LEP) are less frequent, but typically present in immunosuppressed individuals. We also study other hematopoiesis disorders with an increased incidence in the HIV-positive population such as Castleman disease (CD).

Our main areas of research are:

- **Genetic studies on HIV-related lymphomas.** Although HIV-infected patients are treated with the same regimens as HIV-negative individuals, their survival rate is lower due to the higher susceptibility to infections and secondary neoplasms.
- **Liquid biopsy in aggressive lymphomas.** This technique could be useful to diagnose DLBCL earlier, and in a more comprehensive and accurate manner than with tissue biopsy alone.
- **Genetic studies on plasmablastic lymphoma.** Plasmablastic lymphoma (PBL) is a rare B-cell lymphoid neoplasm that especially affects immunocompromised individuals and has a poor.

OUR GOALS

We believe that genetic and epigenetic profiles will help clarify the mechanisms involved in lymphomagenesis and identify potential biomarkers, thus allowing cases to be classified more effectively. The possible diagnostic and/or prognostic impact of these markers could pave the way for the design of new targeted therapies, thus leading to new treatment approaches and improving the outcome of patients suffering from the lymphomas on which our research focuses, i.e. AIDS-related lymphomas, plasmablastic lymphoma and Castleman disease.

OUR CHALLENGES

We expect that the results of our studies will lead to changes in the management of these rare lymphoid neoplasms and improve the poor prognosis of some lymphoid malignancies, such as plasmablastic lymphoma. Through our research, we hope to answer the following questions:

1. What genetic and epigenetic mechanisms are involved in the development of HIV-related lymphomas?
2. Which biomarkers can be used for an earlier diagnosis of lymphoid neoplasms in populations at high risk of developing these disorders, such as HIV-infected and transplanted patients?
3. How can we apply liquid biopsy in the diagnosis and follow-up of aggressive lymphomas?

KEYWORDS

Non-Hodgkin's lymphoma, Hodgkin lymphoma, HIV, Epstein-Barr virus, diagnosis, prognosis, marker, treatment, targeted therapy, early detection

ABOUT US

RESEARCH GROUPS

MULTIPLE MYELOMA LED BY ALBERT ORIOL

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OVERVIEW

Multiple myeloma is a cancer of plasma cells, a type of white blood cell that accumulates in the bone marrow and interferes with normal blood precursors and bone remodelling, thus causing anemia, bone lesions, renal insufficiency and recurrent infections. Between four and five out of 100,000 people are diagnosed every year. Despite the fact that treatments and prognosis have greatly improved in recent years, multiple myeloma is not yet curable. It is a recurrent disease that can leave important sequelae after each relapse.

OUR RESEARCH

Our clinical research team participates in the main international collaborative phase I to phase III trials establishing the current standards of care, with a particular focus on the optimal combinations of agents with clinically relevant synergies.

Active trials are already focusing on the efficacy of next-generation combinations, including antibody-drug conjugates, T-cell engagers and CAR-T cells. We are interested in the identification of subjects unlikely to respond to optimized first-line strategies and, therefore, of ideal candidates for such trials with novel immunotherapeutic approaches.

OUR GOALS

We believe that the drug combinations currently being evaluated can cure a proportion of patients with multiple myeloma. Furthermore, we believe that it should be possible to predict patients in whom such combinations are not curative so that we can promote early interventions with alternative agents, mainly based on immunotherapeutic approaches to prevent the clinical consequences of full-blown relapse and maintain a symptom-free response in patients.

On this regard, our main goals are:

1.
To define standards of treatment that provide a long-lasting response in most individuals.

2.
To identify patients who will probably be cured and will safely remain treatment-free.

3.
To identify patients who are unlikely to be disease-free for long with current treatments and search for alternative treatment options that can be applied before recurring disease causes organic damage.

OUR CHALLENGES

Full-blown multiple myeloma has devastating consequences that severely reduce patients' quality of life and autonomy and represent a huge burden for caregivers and families. Therefore, the diagnosis of multiple myeloma has a dramatic impact on individuals and society. Through our research, we hope to answer the following questions:

1
What patients are unlikely to obtain prolonged benefits from current standards?

2
Would they benefit from early intervention with alternative agents?

3
Can we identify patients who will potentially be cured or are unlikely to relapse and safely spare them the burden of continuous therapy?

KEYWORDS

Multiple myeloma, synergistic combinations, immuno-drug conjugates, T-cell engagers, CAR-T cells.

ABOUT US

RESEARCH GROUPS

MYELODYSPLASTIC SYNDROMES LED BY FRANCESC SOLÉ

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OVERVIEW

Myelodysplastic syndromes (MDS) are a heterogeneous group of hematopoiesis stem cell disorders that result in ineffective hematopoiesis, blood cytopenia, myelodysplasia and a significant risk of progression to acute myeloid leukemia (AML). MDS is one of the most common hematopoiesis malignancies in the elderly and the severity of the disease depends on a variety of biological factors that translate into a spectrum of symptoms with a profound impact on the patient's quality of life and survival; a third of MDS patients will progress to AML and the remaining two thirds will suffer from a combination of chronic anemia, recurrent infections and bleeding episodes.

OUR RESEARCH

Our research focuses on unravelling the heterogeneity of myelodysplastic syndromes (MDS), mainly using genomic techniques. We study MDS patients who harbor a specific cytogenetic alteration: the deletion of the long arm of chromosome 5. Our aim is to improve the genetic characterization of these patients by studying the impact of adjunct cytogenetic abnormalities on their prognostic stratification; how cytogenetics and mutations can influence the response to lenalidomide treatment; the molecular landscape of MDS through next-generation sequencing techniques; and, finally, intratumoral heterogeneity before and after lenalidomide treatment using single-cell techniques.

OUR GOALS

Through our research, we intend to contribute to a better understanding of MDS from a genomic point of view, contributing to refine the current criteria to diagnose this disease and predict patient outcomes to select the best possible treatment. Hence, our research addresses the following lines:

1.
Evaluating the feasibility of using peripheral blood samples to perform genetic analyses (SNP-A and NGS) in MDS.

2.
Monitoring mutational burden in low-risk MDS patients through the use of sequential peripheral blood samples to minimize invasive techniques on these patients.

3.
Genetic characterization of myelodysplastic syndromes / myeloproliferative neoplasms (MDS/MPN) to define the genetic changes that could contribute to the differential diagnosis and prognostic stratification of these patients.

4.
Genetic characterization of therapy-related myeloid neoplasms.

5.
Mechanisms of progression from clonal hematopoiesis to MDS.

OUR CHALLENGES

Our research can translate into a more efficient use of public healthcare resources and improve the quality of life for patients. Therefore, we want to shed light on the following questions:

1
How might genomic techniques contribute to refining the current criteria for MDS diagnosis, prognostic stratification and treatment response?

2
Can peripheral blood samples be useful to monitor MDS patients through next-generation sequencing?

3
Could single-cell studies help us better understand intratumoral heterogeneity and clonal evolution from CHIP to MDS and TRMN (therapy-related myeloid neoplasms)?

KEYWORDS

Myelodysplastic syndromes, chronic myelomonocytic leukemia, intratumoral heterogeneity, myelodysplasia, cytopenias, CHIP, TRMN

ABOUT US

RESEARCH GROUPS

STEM CELL BIOLOGY, DEVELOPMENTAL LEUKEMIA AND IMMUNOTHERAPY LED BY PABLO MENÉNDEZ

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OVERVIEW

Our group is interested in understanding the cellular origin, etiology and pathogenesis of childhood leukemia. We aim to ascertain the cell in which mutations occur and we strive to discover which cells are responsible for triggering relapses. Furthermore, we work to identify new therapeutic targets and develop more targeted, less toxic therapies. To achieve this, our laboratory uses various approaches, including genetic studies, epigenetic techniques and animal models, as well as adoptive cell immunotherapy tools.

OUR RESEARCH

Given that acute childhood leukemia (including the B, T and myeloid variants), and childhood cancer in general, are relatively uncommon illnesses, with around 500 cases in Spain each year, it does not represent a priority target for the pharmaceutical industry. As a result, there is a serious lack of active programs that aim to identify medicines to target childhood cancer. Our group has been investigating the origin of this diseases in utero, as well as its etiological causes and physiopathological mechanisms. In 2016, we began researching non-toxic, targeted adoptive cellular immunotherapies for these children with the aim of preventing the long-term effects of current chemotherapy.

OUR GOALS

Our group is currently involved in various lines of research in pursuit of the following objectives:

1.

To understand the aetiology and pathogenesis of leukemia in breastfeeding infants.

To do so, we use primary samples taken from patients and develop different animal and cellular models based on prenatal (embryonic, foetal) and postnatal (neonatal and adult) stem cells.

2.

To gain a better understanding of the role of bone marrow (BM) stroma in chemoresistance in acute myeloid leukemia (AML)

and identify new therapeutic targets for AML, which is the most common form of leukemia in adults and whose prevalence increases with age.

3.

To improve adoptive cellular immunotherapies against ALL-B, ALL-T and AML.

To achieve this, we are searching for new therapeutic targets and developing new CARs (chimeric antigen receptors) for the different types of acute leukemia.

Our overall goal is to contribute towards curing 100% of childhood leukemias or convert them into chronic conditions, without generating lifelong toxicities.

OUR CHALLENGES

Childhood diseases have an enormous emotional impact on the patient's whole family and everybody around them. Moreover, we must not forget that children are the future of our society, so investing in their health will benefit the future of our society enormously. Through our research, we aim to:

1

Identify the cellular origin, cellular and molecular mechanisms, and the genetic and epigenetic composition of ALL-B in breastfeeding infants.

2

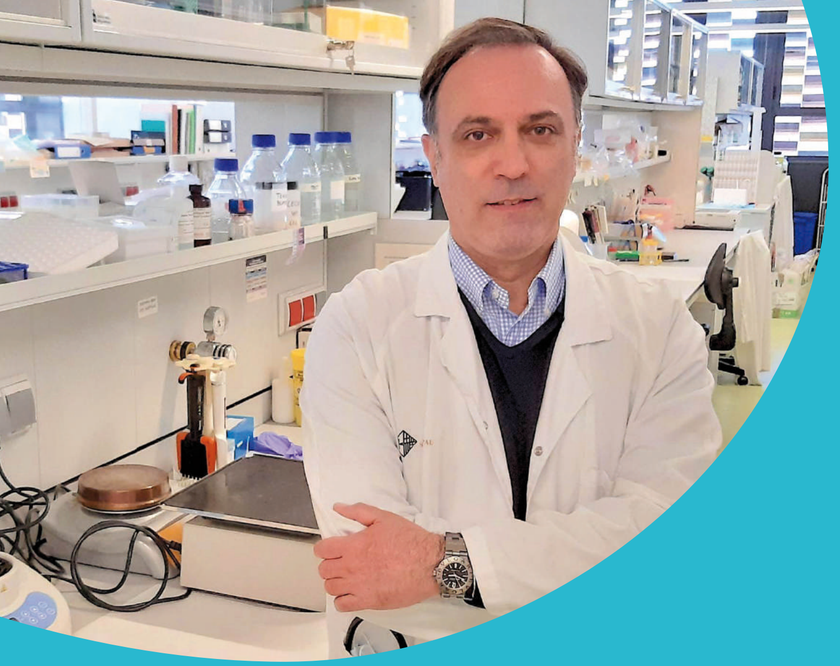
Contribute to the development of new therapeutic strategies in AML targeted towards reducing the resistance mediated through the BM microenvironment and that are particularly effective against LICs.

3

Develop adoptive cellular immunotherapies against ALL-B, ALL-T and AML using allogeneic T-cells without genome editing to eliminate TCR, CD3 and other molecules that play a role in immunological synapse.

KEYWORDS

Pediatric leukemia, stem cells, immunotherapy, MLL rearrangements, PDX models



ABOUT US

RESEARCH GROUPS

CELLULAR IMMUNOTHERAPY AND GENE THERAPY LED BY JAVIER BRIONES

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OVERVIEW

The Cellular Immunotherapy and Gene Therapy Group is focused on the study of genetically modified T-cells expressing chimeric antigen-specific receptors (CARs) and their clinical application in patients with blood cancer.

OUR RESEARCH

Blood malignancies, like leukemia and lymphoma, are challenging diseases affecting one or more blood cell lineages. Traditional treatments include chemotherapy to deplete altered cells as much as possible, followed by bone marrow transplantation from a compatible donor, when appropriate.

The group currently focuses on studying T-cells genetically modified with chimeric antigen-specific receptors (CARs) and their clinical application in patients with blood cancer. Its current lines of research concentrate on the following aspects of cellular immunotherapy:

- Functional antitumor research into subtypes of memory T-cells.
- Study of the antitumor efficacy of memory stem T-cells genetically modified with CARs.
- Development of new CARs targeted against hematopoiesis malignancies.
- Development of clinical immunotherapy trials with CAR T-cells on patients with lymphoid neoplasms.

OUR GOALS

Recently, a new and powerful immune cell type has been discovered called memory stem T cells. These are scarce but very special and, despite being roughly a 1% of the cells in our blood, they can find, attack and destroy cancer cells very efficiently. We have developed mechanisms to expand memory stem T cells in the lab and grow them in greater numbers, to make their action more potent and long-lasting.

Also, the genetic modification of patient-derived memory stem T cells to make them express CAR receptors, the so-called CAR-T therapy, is proving an enhanced anti leukemic potential in the clinical trials.

KEYWORDS

PCAR-T; T-Cells; Lymphoid Neoplasms

ABOUT US

RESEARCH GROUPS

STEM CELL TRANSPLANTATION AND CELLULAR IMMUNOTHERAPY LED BY ÁLVARO URBANO-ISPIZUA

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OVERVIEW

We conduct research into cell immunotherapy treatments for patients with advanced malignant blood disorders, who tend to have a very short life expectancy. To treat such patients, we develop CAR-T and CAR-NK therapies based on adding chimeric antigen receptors (CAR) to cells of the immune system, such as T-lymphocytes and NK cells, respectively. CARs help recognize and attack tumor cells exclusively, specifically and effectively, thereby preventing an autoimmune response and reducing secondary effects on healthy cells.

OUR RESEARCH

Former research from our group focused on the cytotoxicity mechanisms of CB-NK cells when they come into contact with MM tumor cells, in order to fight them. We discovered that they were able to regulate the cytotoxic or attack mechanisms depending on the characteristics of each tumor cell.

Later on, we started developing CAR-T cells against BCMA to treat MM patients and achieved good results. We are currently improving cell immunotherapy treatments by combining CAR-T with CB-NK cells. Moreover, we are studying how and why some tumor cells develop resistance to these therapies, with the aim of suppressing this resistance capacity.

OUR GOALS

We are studying what happens at a molecular level between CAR-T and CB-NK cells throughout the process of recognizing, contacting and attacking tumor cells in order to identify which proteins and defence strategies are used by CAR-T, CB-NK and other cells in the immune system. Moreover, we are examining what happens within the environment of the cells when they meet tumor cells. This knowledge will help us develop better strategies to improve the efficacy of these therapies.

Through our research, we aim to achieve the best possible scenario: to cure patients and ensure that they do not relapse.

OUR CHALLENGES

If we manage to enhance the efficacy of the CAR-T therapy and its permanence in patients to protect them from relapses, this breakthrough could be applied to patients with types of cancer other than MM. Therefore, through our research we hope to answer the following questions:

1. How are tumor cell resistance mechanisms against immune cells developed?
2. How can these tumor cell resistance mechanisms against immune cells be avoided?
3. How can the persistence and efficacy of CAR-T cell treatment be increased?

KEYWORDS

Multiple myeloma, B-cell malignancies, chimeric antigen receptors, T lymphocytes, NK cells, cord blood-derived NK cells, hematopoiesis malignancies, B-cell maturation antigen

ABOUT US

RESEARCH GROUPS

EPIGENETIC THERAPIES LED BY MARÍA BERDASCO

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OVERVIEW

Epigenetic therapies aim to modify the epigenome, the set of molecular processes that regulate gene expression without altering the DNA sequence and can change the course of a disease and its phenotype. There are now examples of epigenetic drugs for treating hematopoiesis malignancies approved by the United States Food and Drug Administration (FDA). However, the volume of promising preclinical evidence far exceeds the number of epigenetic research projects that have resulted in clinical applications to patients. Therefore, more translational studies that may lead to the development of more specific epigenetic drugs and more robust biomarkers are required.

OUR RESEARCH

Our research aims to ascertain the therapeutic benefit of targeting epigenetic alterations in cancer together with the epigenetic-based stratification of patients to predict therapy response. To achieve this, we develop research based on three specific aims:

- Identification of the epigenetic alterations that act as drivers of tumor progression ("druggable epigenetic alterations").
- Validation of epidrugs that can efficiently revert aberrant epigenomes in cancer.
- Stratification of patients based on their epigenetic profile to predict response to immunotherapy.

OUR GOALS

We aim to develop a translational research line that focuses on elucidating the epigenetic alterations that are druggable targets in a tumor, and the means to exploit them therapeutically within the framework of precision medicine.

OUR CHALLENGES

Through our research, we aim to help answer the following questions:

1

Which epigenetic alternations represent targets for drugs to treat cancer?

2

How can we efficiently treat tumors caused by epigenetic alternations?

3

Who could benefit from therapeutic strategies based on epigenetics?

KEYWORDS

Epigenetic drug, epigenetic editing, epidrug, hematopoiesis malignancies, targeted therapies

ABOUT US

RESEARCH GROUPS

LYMPHOMA TRANSLATIONAL LED BY GAËL ROUÉ

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OVERVIEW

Our research is centered on the development of innovative preclinical models of B-cell lymphoma that can be used to unravel the complex role of tumor-lymphoma crosstalk during the development of the disease and the acquisition of refractoriness in current regimens. To that end, we intend to reproduce the original composition and architecture of tumors in the laboratory to carry out a complete transcriptomic and proteomic analysis and develop new pharmacological entities in collaboration with academic experts and clinical-level pharmaceutical companies, all with a view to fostering the bench-to-bedside transfer of new and tailored therapeutic strategies.

OUR RESEARCH

We develop in vitro and animal models with the intention of more accurately recreating the context of onset and progression of the lymphoma in each patient. In this regard, we take account of fundamental parameters that until now have been largely unexplored in preclinical research, such as the architecture of the original tumor, the components of the immune system that accompany it and the defects in the regulation of the proteins – not only of the genes – that characterize it.

OUR GOALS

Our main areas of research are

1.

Development of a patient-derived xenograft platform for the evaluation of new targeted therapies in aggressive B-cell lymphomas.

To confirm the efficacy, safety and translationality of these studies, we propose that standard in vitro assays be associated with innovative in vivo models (PDX) to allow us, firstly, to work directly with primary tumor cells to validate the most effective therapies and the most relevant biological effects while taking into account the role of the tumor microenvironment; and, secondly, to guarantee that these therapies can be translated into clinical trials with molecular and genetic determination of the factors that condition the response to the best therapies tested in each patient.

2.

Modulation of the lymphoid microenvironment by intrinsic protein homeostasis in aggressive B-cell lymphoma.

We will characterize the ubiquitome of malignant B cells through proteomic profiling and correlate these intracellular complexes with the immunological pattern of each tumor model to validate the impact of tumor protein homeostasis on the development of B-cell lymphoma and the intratumoral infiltration of immune cells.

OUR CHALLENGES

Through our research, we aim to understand the following:

1. To what extent intrinsic protein homeostasis can regulate the complex tumor-stroma crosstalk in different models of aggressive B-cell lymphoma.
2. How germinal center-derived lymphoma can be sensitized to immune checkpoint blockade therapy.
3. How multiomics analysis of paired treatment-naïve and therapy-refractory B-cell lymphoma can help in the design of efficient and personalized therapies.

KEYWORDS

B-cell non-Hodgkin's lymphoma (NHL), tumor modelling, proteostasis, tumor microenvironment, immunotherapy

ABOUT US

RESEARCH GROUPS

DESCRIPTIVE EPIDEMIOLOGY, GENETICS AND CANCER PREVENTION LED BY RAFAEL MARCOS GRAGERA

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OVERVIEW

One of the main lines of research of the group is the epidemiology of hematological neoplasms, with the aim of determining the incidence, prevalence and survival of this type of cancer. The results obtained aim to provide useful and reliable information to design and/or improve the appropriate health resources and describe the population trends of this group of diseases.

OUR RESEARCH

Hematological neoplasms are a large group of diseases with a wide variability derived mainly from the type of cells that cause it. Also, the evolution and prognosis of each histological subtype of neoplasia is very diverse and it is necessary to deepen the knowledge of each of them. In this sense, population cancer records are a reference tool for establishing the incidence and survival of each type.

OUR GOALS

Specifically, our research objectives aim at:

1.
Establishing the prevalence, incidence and survival of myeloid, lymphoid and histiocytosis neoplasms globally and according to the respective subtypes.

2.
To analyze the temporal trend of the incidence and survival of hematological neoplasms in the context of the evolving therapeutic background.

3.
Determine epidemiological parameters based on sex and age.

4.
Carry out etiological studies of hematological neoplasms according to each of the histological subtypes.

5.
To study the genetic and environmental risk factors related to hematological neoplasms.

6.
Describe the risk factors and epidemiology of multiple myeloma based on its precursor cells.

7.
Analyze the associations between comorbidity and the survival of lymphoid and myeloid neoplasms.

8.
Evaluate the population effectiveness of new therapies in a real population and the impact on survival.

9.
dentify changes in the classification, definition and coding of hematological neoplasms and establish working protocols to have homogeneous tools that allow epidemiological comparisons at the international level.

OUR CHALLENGES

Through our research, we aim to understand the following:

1
What is the incidence of hematological neoplasms in the territory?

2
What is the survival of each of the histological subtypes of neoplasia?

3
How have changes in the coding of hematological neoplasms over time affected the epidemiological determinants of this group of diseases?

KEYWORDS

Incidence, survival, mortality, lymphoid neoplasms, myeloid neoplasms, histiocytosis, hematological neoplasms

ABOUT US

RESEARCH GROUPS

ONCOGENESIS AND ANTITUMOR DRUGS LED BY RAMON MANGUES

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RUEDA MATAS, ARIANA
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Lab Technician

HUACA MANCHEGO, VANESSA
Lab Technician

SEIRA ORIACH, CLARA
Lab Technician

MIRANDA TOVAR, EVA
Lab Technician

NAVAS JIMENEZ, LUIS CARLOS
Lab Technician

OVERVIEW

Current treatments lack selectivity towards cancer cells, which induces insufficient anticancer activity and produces severe adverse effects that limit their dosage. We are developing self-assembling protein-based nanoparticles for the treatment of hematological and solid cancers that are highly selective in targeting receptors overexpressed in cancer stem cells. They display a wide therapeutic window by avoiding renal clearance while internalizing into and selectively eliminating cancer target cells and enhancing the uptake of the payload drug into cancer tissues, with negligible uptake or toxicity in normal tissues.

We have achieved high antitumor and antimetastatic effects using apoptotic, genotoxic or microtubule inhibitor drugs as payloads, and we are now testing novel payloads that use non-apoptotic cell death mechanisms.

OUR RESEARCH

We generate novel protein-based nanomedicines with a high therapeutic window that tackle unmet treatment needs in acute myeloid leukemia (AML), diffuse large B-cell lymphoma (DLBCL) and colorectal cancer (CRC) using preclinical models and clinical translation, through:

- The development of animal cancer models that are resistant to current therapy or disseminated for the study of the molecular mechanisms of cancer stem cell involvement in these processes, especially in cancer cells that overexpress the chemokine receptor CXCR4.
- The development of drug nanoconjugates or protein-only nanoparticles for intravenous injection that use receptor-mediated targeted delivery of cytotoxic agents to cancer stem cells and incorporate novel drugs or polypeptide domains that exploit the higher capacity for apoptotic induction, or the triggering of cell death mechanisms as an alternative to apoptosis.
- The development of artificial amyloid bodies for subcutaneous injection with capacity for the sustained release of therapeutic protein nanoparticles in the bloodstream that reach cancer tissues.

OUR GOALS

Our aim is to develop nanomedicines that can effectively render cancers that have disseminated or relapsed sensitive to therapy by acquiring resistance to current therapy. In doing so, we expect to increase the cure and complete response rates, thereby leading to longer survival times.

An additional goal is to ensure that the repeated administration of these novel nanomedicines induces potent anticancer activity, while maintaining low or absent toxicity in normal tissues, associated with a lack of, or tolerable, side effects.

Finally, we also aim to develop a formulation of amyloid structured inclusion bodies whose capacity for the sustained release of therapeutic nanoparticles into the blood could be subcutaneously administered once a month. Their development will circumvent the need to administer the nanoparticles twice a week by intravenous injection, thus allowing patients to stay at home during treatment and avoid hospitalization.

OUR CHALLENGES

Ninety percent of cancer patients die of metastases that do not respond to current treatments. Therefore, patients who develop metastases are considered incurable. Through our research, we aim to answer the following questions:

1

Is the selective elimination of cancer stem cells a relevant clinical target to improve therapy in different cancer types with acquired resistance and disseminated disease?

2

Will protein-based targeted nanoparticles that incorporate non-apoptotic and immunogenic cell death polypeptides increase cure, response and survival rates while reducing side effects once tested in patients?

3

What are the underlying mechanisms that dictate the highly selective accumulation of protein nanoparticles targeting the CXCR4 receptor we observe in cancer tissues?

KEYWORDS

Biotechnology, nanomedicine, targeted drug delivery, oncotherapy, metastases

ABOUT US

RESEARCH GROUPS

CHRONIC LYMPHOCYTIC LEUKEMIA LED BY CAROLINA MORENO

GROUP MEMBERS

MORENO ATANASIO, CAROLINA
Group Leader

MORA RAYA, ALBA
Postdoctoral Researcher

CUELLAR GARCIA, CAROLINA
Attending Physician

JARA BUSTAMANTE, PAOLA
Lab Technician

OVERVIEW

Chronic lymphocytic leukemia (CLL), the most common adult B-cell malignancy in Western countries. It is characterized by the accumulation of monoclonal CD5+ B cells with a characteristic immunophenotype in peripheral blood, bone marrow, and lymphoid tissues. The clinical course of the disease is extremely heterogeneous and, as a result, the individual life-expectancy ranges from a few years to a virtually normal lifespan. Despite the important progress in its therapy, CLL is still considered an incurable disease.

OUR RESEARCH

Our group is constantly making hard efforts to gain further insights into the characteristics of the leukemic compartment and deepen on the impact of the tumor microenvironment. This knowledge can contribute to better understand the biologic events involved in the disease development and be used to develop new strategies for clinical management and therapy and eventually to improve the quality of life of patients with CLL.

Our group has been involved in several pivotal clinical trials that led to the approval of new drugs for CLL therapy. Also, we are involved in several projects focused on the characterization of residual leukemic cells, autoimmunity and mechanisms at work in BCR signaling.

OUR GOALS

Our group is constantly making hard efforts to gain further insights into the characteristics of the leukemic compartment and deepen on the impact of the tumor microenvironment. This knowledge can contribute to better understand the biologic events involved in the disease development and be used to develop new strategies for clinical management and therapy and eventually to improve the quality of life of patients with CLL. Through our research, we aim to:

1.
Make available better diagnosis, prognosis and therapeutical strategies in CLL.

2.
Extend the knowledge of molecular and cellular events implicated in CLL development.

3.
Prevent clinical relapses in CLL.

4.
Design feasible approaches to monitor MRD in CLL.

5.
Find a cure to CLL patients.

OUR CHALLENGES

Inherent to the disease, CLL patients have a deregulated immune system, which predispose them to have disease complications, including recurrent infections and autoimmune phenomena. The understanding of how the immune cells of tumor microenvironment contribute to maintain the survival of leukemic cells is essential to design future therapeutic strategies aimed, not only at eradicating leukemic cells, but also at restoring the immune system.

In the last years our group has started some projects aimed at describing the immune status and functional characteristics of B and T cell populations during the evolution of the disease and after therapy with targeted therapies.

KEYWORDS

Chronic Lymphocytic Leukemia, B-cell receptor signaling, minimal residual disease, liquid biopsy, tumor microenvironment

ABOUT US

RESEARCH GROUPS

HEMATOLOGY RESEARCH LED BY DAVID GALLARDO

GROUP MEMBERS

GALLARDO GIRALT, DAVID
Group Leader

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MORET PUIG, CARLA
Attending Physician

MOSTACEDO MARASOVI, SILVIA
Attending Physician

RONCERO VIDAL, JOSEP
Attending Physician

SANTOS CARVAJAL, NAZLY
Attending Physician

SITGES ARRIAGA, MARTA
Attending Physician

VILA BOU, JORDI
Attending Physician

LLOPIS PUIGMARTÍ, FRANCISCA
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GONZÁLEZ BÁRTULOS, MARTA
Lab Technician

CASADO PUERTAS, LORENA
Administrative Assistant

OVERVIEW

The research group in hematology is devoted to clinical and translational trials in hematology, focused on diagnosis, prognosis and the development of new therapies to treat hematological malignancies such as leukemias and myelomas.

OUR RESEARCH

The group uses a variety of approaches such as genetic studies of the immune response, pharmacogenomics in response to treatment, analysis of polymorphisms as disease predictors and the study of cell populations using flow cytometry for the characterization of residual disease.

RESEARCH GROUPS

OUR GOALS

Through our research, we aim to:

1.
Investigate biological, clinical, and epidemiological aspects of hematological diseases.

2.
Carry out translational research projects focused on finding prognostic factors or treatment response predictors.

3.
Carry out clinical research, promoting participation in clinical trials for hematological diseases and participating in national and international cooperative groups.

KEYWORDS

Hematology, myeloblastic leukemia, multiple myeloma, chronic lymphatic leukemia, residual disease

ABOUT US

RESEARCH GROUPS

MYELOID NEOPLASMS (CLÍNIC) LED BY JORDI ESTEVE

GROUP MEMBERS

ESTEVE REYNER, JORDI
Group Leader

OVERVIEW

Myeloid neoplasms are a group of diseases in which the bone marrow produces an abnormal quantity of precursors for red blood cells, platelets or certain types of white blood cells. This leads to a variety of symptoms and from fatigue to bones fragility and, eventually, to a higher risk of developing Acute Myeloid Leukemia (AML).
Despite the advances produced during the last decades, not all those diagnosed benefit from efficient therapies. Advancing in the knowledge of myeloid neoplasms is, therefore, paramount to increase both prognosis and survival of patients.

OUR RESEARCH

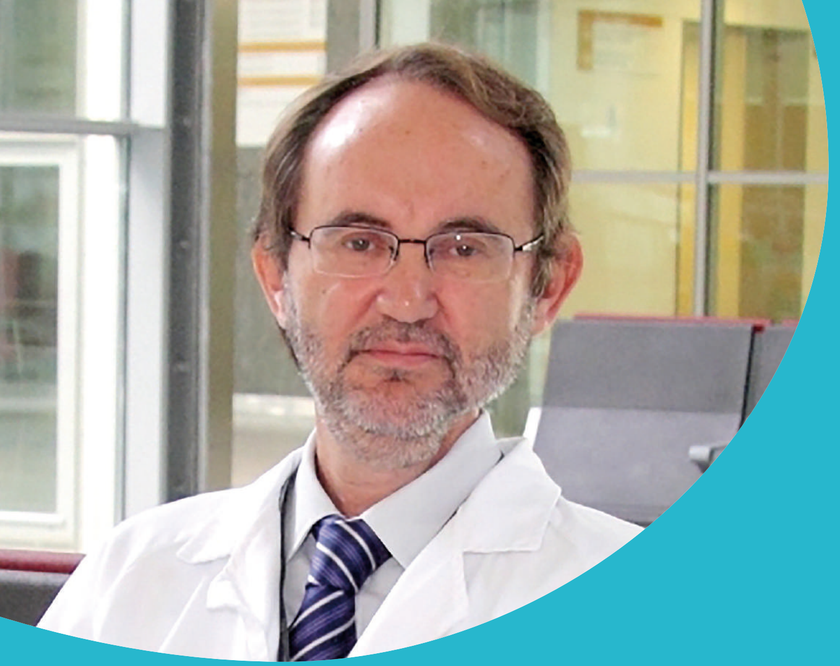
Our research group is searching for key molecular features of myeloid neoplasms that could be used as therapeutic targets. In particular, we are focusing our efforts towards:

- Myeloma and other monoclonal gammopathies
- Mechanisms of progression in monoclonal gammopathies
- Myeloid neoplasmas
- Lymphoid neoplasms

Also, we are seeking to improve the overall knowledge of the neoplasm microenvironment, the conditions where malignant cells live and proliferate, as well as how the body defenses respond to it.

KEYWORDS

Pediatric leukemia, stem cells, immunotherapy, MLL rearrangements, PDX models



ABOUT US

RESEARCH GROUPS

HEMATOLOGICAL DISEASES, TRANSPLANT AND CELL THERAPY LED BY JORDI SIERRA

GROUP MEMBERS

SIERRA SIERRA, JORDI
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MARTINO BOFARULL, RODRIGO
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GARCÍA CADENAS, IRENE
Attending Physician

SAAVEDRA GEROSA, SILVANA
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PhD Student

ARGÜELLO DE TOMAS, MIGUEL
Junior Researcher

REDONDO VELAO, SARA
Junior Researcher

ESQUIROL SANFELIU, ALBERT
Lab Technician

ABOUT US

RESEARCH GROUPS

OUR RESEARCH

Our research focuses on the molecular and cellular physiopathology of blood cancers, particularly on acute myeloid leukemia (LMA) and chronic lymphatic leukemia (CLL) where we seek to find new treatment options targeting molecular features. Also, we study the prognostic value of clinical and biological features in malignant hemopathies, like LMA and CLL.

We study the transplant of hematopoietic progenitors and its complications and develop new academic CAR-T cells enriched in T-memory stem cells to treat T and B Hodgkin lymphomas.

OUR GOALS

The main goals of our research are:

1.
Identify new prognostic parameters for risk and therapeutic stratifications.

2.
Molecularly characterize acute myeloid leukemia and determine the prognostic value of known genes and other genes of uncertain significance.

3.
Evaluate targeted therapy in cell lines and animal models (together with Dr. Mangues' group).

4.
Reduce toxicity and increase the availability of allogeneic transplants.

5.
Preclinical (mouse) and clinical studies on immunotherapy for lymphoproliferative diseases. Development of non-commercial CAR-T cell therapies.

Our overall goal is to contribute towards curing 100% of childhood leukemias or convert them into chronic conditions, without generating lifelong toxicities..

OUR CHALLENGES

It is paramount that we improve the prognosis of hematological patients by using new more precise therapies, and less toxic. Therefore, through our research, we aim to:

1
Improve the genotypic and immunophenotypic characterization of AML and CLL, to identify new prognostic factors and administer targeted therapy.

2
Improve the safety and effectiveness of hematopoietic transplantation and expand the number of patients who can benefit from it.

3
Develop new CAR-T products that enhance the currently commercially available ones.

KEYWORDS

Hematopoietic transplantation, CAR-T cells, Immunotherapy, Acute Myeloid Leukemia, Chronic Lymphocytic Leukemia

ABOUT US

RESEARCH GROUPS

HEMATOLOGICAL DIAGNOSIS LED BY JOSEP NOMDEDÉU

GROUP MEMBERS

NOMDEDÉU GUINOT, JOSEP
Group Leader

ABOUT US

RESEARCH GROUPS

OUR RESEARCH

In our lab, we focus on both malignant and non-malignant hemopathologies to offer better diagnostics, understand its biological characteristics, and develop new treatments. We study acute leukemias multi-omics and platelet pathologies like thrombocytopenias, thrombocytopathies and thrombosis.

MALIGNANT HAEMOPATHOLOGIES

- Diagnostic activities: morphological, immunophenotyping, cytogenetic and molecular characterization of acute leukemias).
- Biological characteristics responding to therapy.
- New treatments using cell line models aimed at molecular targets.
- Genomic (microarrays) and proteomic platforms for diagnosis.
- Murine models development.

NONCANCEROUS HAEMOPATHOLOGIES

- Diagnosis and characterization of thrombocytopenia, thrombocytopathies and other platelet pathologies.
- In the framework of the GAIT-2 project (genetic analysis of idiopathic thrombophilia, phase 2), to seek new phenotypes that favor the development of thrombosis, specifically related to the structure and function of platelets and other blood cells.

OUR CHALLENGES

Through our research, we aim to:

1. Consolidate the characterization of hemato-logical tumors and complex, rare, and genetic noncancerous hematopathology's.
2. Include the results of mass-analysis genomic and proteomic platforms in diagnostic algo-rithms and establish prognostic factors for hematological disorders.
3. Develop functional cell culture and animal (murine) models.
4. Consolidate cooperation with the GAIT-2 project, especially regarding platelet and other blood cell participation in thrombosis generation.

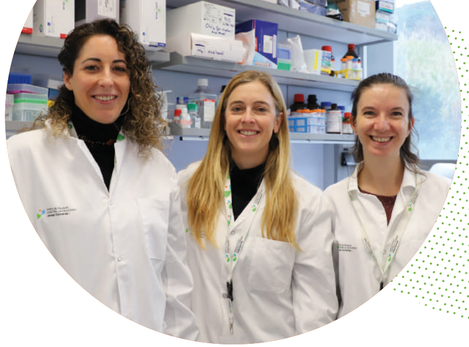
KEYWORDS

Malignant hemopathologies, thrombocytopathies, thrombosis

ABOUT US

CORE FACILITIES

SINGLE CELL UNIT



The Single Cell Unit aims to provide scientific services to the Josep Carreras Institute community and is equipped with cutting-edge technology to apply single-cell technology to basic and translational genomic and transcriptomic studies. Single Cell approaches allow us to identify cell populations that are impossible to isolate with less resolute technologies previously used, such us bulk sequencing, allowing to characterize cell populations and thus identify differences at the genetic and phenotypic level within tumor tissues.

These techniques can, therefore, reveal the cellular heterogeneity of tumors and help identify cells resistant to standard treatments or more prone to proliferate.

The Single Cell Unit is equipped with 2 Chromium Controller (10x Genomics), for single cell analysis at the transcriptomic level, a Nikon ECLIPSE Ti Series inverted microscope and a CytAssit equipment (10X Genomics), for spatial transcriptomics at tissue level and a Tapestri platform (Mission Bio), for single cell analysis at the genomic and proteomic level.

These technologies provide a comprehensive, scalable solution for cell characterization, gene expression profiling and DNA sequencing of up to tens of thousands of cells.

Services:

ChromiumTM Controller (10x Genomics):

- Single-cell RNA-seq (gene expression)
- Single-cell RNA-seq (gene expression) + Feature Barcoding
- Single-cell Immune profiling (GEX 5’ + TCR/BCR)
- Single-cell ATAC-seq
- Single-cell Multiome (ATAC + GEX)

Spatial Transcriptommics (10X Genomics):

- Visium Spatial Gene Expression for Fresh-Frozen (FF) tissues
- Visium Spatial Gene Expression FFPE tissues
- Visium CytAssist Spatial Gene Expression for FF & FFPE tissues

Tapestri Platform (Mission Bio):

- Single-cell targeted DNA-seq (mutation analysis)
- Single-cell targeted DNA-seq (mutation analysis + CNV analysis)
- Single-cell DNA-seq + cell-surface protein analysis

GROUP MEMBERS

MATA GARCIA, CATERINA
Core Facility Leader

LÓPEZ JIMÉNEZ, LIDIA
Core Facility Technician

ABOUT US

CORE FACILITIES

GENOMICS UNIT

The Genomics Unit at IJC aims to provide scientific services to the IJC community as well as to external researchers. Several technologies have been implemented in the Unit to assess relevant genomic and epigenomic features in order to unravel basic molecular mechanisms of disease and contribute to discover therapeutic targets and biomarkers.

The Unit is equipped with cutting-edge technology to offer solutions on both basic and translational epigenomic and genomic studies in many types of samples (primary cells, cell lines, frozen and paraffin-embedded tissues, etc.). We have long standing experience in arrays-based genome-wide DNA methylation analysis, and we also perform pyrosequencing for DNA analysis. We use next-generation sequencing (NGS) technology to investigate subsets of genes or specific genome regions.

1.

Infinium MethylationEPICTM BeadChip technology (Illumina):
Infinium MethylationEPIC BeadChip Kit, allows interrogation of over 850,000 methylation sites quantitatively across the genome at single-nucleotide resolution. It provides a Comprehensive Genome-Wide Coverage as interrogated sites include CpG sites outside of CpG islands, Non-CpG methylated sites identified in human stem cells (CHH sites), differentially methylated sites identified in tumor versus normal, FANTOM5 enhancers, ENCODE open chromatin and enhancers, DNase hypersensitive sites and miRNA promoter regions. High throughput is supported by chip analysis performed using Illumina HiScanTM SQ fluorescent scanner and the Freedom EVO® platform.

2.

Infinium Mouse Methylation BeadChip (Illumina):
The mouse methylation kit is ideal for genome-wide DNA methylation studies with a large number of samples, as it allows the interrogation with sample high throughput of > 285k methylation sites per sample at single-nucleotide resolution. It provides balanced coverage of CpG islands, translation start sites, enhancers, imprinted loci, and other regions.

3.

MySeqTMDx NGS sequencer (Illumina):
The MiSeqDx technology is used for targeted sequencing of DNA libraries from human genomic DNA extracted from peripheral whole blood or formalin-fixed, paraffin-embedded (FFPE) tissue. It can be used for basic research purposes or for in vitro diagnostic (IVD) assays. It reaches a throughput of 1–96 samples/run depending on the assay, and gives a read length of up to 2 × 300 bp.

4.

PyroMarkTMQ48 (Qiagen):
Pyrosequencing is a sequence-based platform, that integrates detection and quantitative real-time data for the analysis of targeted short DNA sequences. It is used to characterize single nucleotide polymorphisms (SNPs), insertion-deletions (indels), and unknown sequence variants, and DNA methylation levels at both CpG and non-CpG (CpN) sites.

GROUP MEMBERS

ALVAREZ ERRICO, DAMIANA
Core Facility Leader

ARRIBAS JORBA, CARLES
Core Facility Technician

ALEDÓN ANDÚJAR, LAILA
Core Facility Technician

ABOUT US

CORE FACILITIES

CELL IMMORTALIZATION UNIT

The Cell Immortalization Unit of the Josep Carreras Leukaemia Research Institute offers Infection of B-cells with Epstein–Barr virus (EBV) leads to more and subsequent immortalization. This is considered as the method of choice for generating lymphoblastoid cell lines (LCLs). Cell culture is an essential tool to study the fundamentals of genetic background variables. With the development of personalized medicine, this applies increasingly to the development and safety testing of drugs. Infection of B-cells with Epstein–Barr virus (EBV) leads to more and subsequent immortalization. This is considered as the method of choice for generating lymphoblastoid cell lines (LCLs). After successfully production of LCLs, different parameters including temperature, serum concentration, type of culture medium, and CO2 concentration must be evaluated on EBV-transformed B-cells. Our unit can produce LCLs and optimize condition.

Applications

- This immortalization technology enables rapid, efficient, and reliable production of unlimited numbers of personalized cells.
- To produce control material for rare genetic disorders.
- Lymphocyte Immortalization technique let to preserve of DNA, RNA, and proteins samples, that appears to be a valid strategy for further studies.
- To determine optimized condition for reliable and reproducible LCLs from different sources.
- Testing drugs analysis.
- Allows us to have enough biological sample without having to access the patient again.

GROUP MEMBERS

DE LA TORRE GÓMEZ, CAROLINA
Core Facility Leader

SETIÉN BARANDA, ESTEBAN FERNANDO
Core Facility Technician

PROTEOMICS UNIT

The Proteomics Unit of the Josep Carreras Leukaemia Research Institute, part of the Carlos III Health Institute (ISCIII) and the Proteomics Network ProteoRed, offers mass spectrometry services to the academic and to the private sector under request.

The unit's main activity is to promote the incorporation of proteomics as a key tool for the development of clinical and basic projects at our institution. Our main work consists of offering innovative, high-quality proteomic and peptidomic services that allow the best therapeutic and human health solutions to be selected.

How do we support you?

- By providing scientific and technological support to high-level research projects in the field of proteomics according to international standard procedures.
- By providing researchers with scientific advice, from the project's planning and experimental design stage to the execution phase, processing of samples and interpretation of results, and support during presentations and writing of results for publication.
- Through dissemination and training for researchers on the methodology and applications of the techniques offered.
- By contributing to the promotion of innovation in health technologies and the transfer of the knowledge generated to the public health service, and supporting genetic, epigenetic and pharmacogenetic diagnosis.

Our services:

Proteomic analysis is a very powerful approach to addressing key challenges in clinical and health research. This approach can be used for different aspects of clinical and health sciences, such as biomarker discovery, drug target identification and food technology.

Biomarker discovery from a wide range of samples to improve precision medicine at different levels: a) early diagnostic, and b) prognosis to predict disease progression and guide treatment selection.

GROUP MEMBERS

DE LA TORRE GÓMEZ, CAROLINA
Core Facility Leader

BECH SERRA, JOAN JOSEP
Core Facility Technician

MARTINEZ LASTRA, CARLOTA
Core Facility Technician

TRIGUERO OLMO, CARLA
Core Facility Technician

CUCURULL PREIXENS, BERNAT
Core Facility Technician

RINCON RIVEROS, WALTER ANDRES
Core Facility Technician

ABOUT US

CORE FACILITIES

BIOINFORMATICS UNIT

The Bioinformatics Unit at IJC provides both internal and external researchers with high-quality computational analysis services covering all project aspects related to clinical and biological data. This includes experimental design and data analysis for microarray experiments and Next Generation Sequencing, statistical consulting, data integration, interpretation and reporting, as well as software development and data management.

The unit further provides training workshops on different bioinformatics related topics, such as working in a Linux environment, the use of high-performance computing (HPC) resources, the R programming language, working with containers, best practices, etc., and supervises students.

General services:

- Data analysis, including consulting on experimental design and selection of the appropriate workflow and tools, data visualizations, report generation
- Custom analyses and tailored software development
- Data management, transfer/submission from/to public repositories (GEO, SRA, EGA)
- Support for grant and project proposal writing
- Supervision of students, bioinformatics training

Genomics:

- Genotyping and variant calling from whole-exome sequencing (WES), whole genome sequencing WGS, amplicon sequencing and SNP microarrays,

Transcriptomics:

- Differential expression analysis from RNA-seq (polyA, totalRNA) and miRNA, mRNA microarrays, target prediction
- Analysis of alternative splicing from RNA-seq
- Variant calling (e.g., RNA editing) from RNAs-eq

Epigenomics:

- Analysis of 5mC and 5hmC DNA methylation by microarray (450K, EPIC, mouse), or NGS (whole-genome bisulfite sequencing (WGBS), reduced-representation bisulfite sequencing (RRBS))
- Chromatin analysis by ChIP-seq, DNAse-seq, ATAC-seq

Epitranscriptomics:

- Analysis of RNA Protein binding by CLIP-seq (binding and motif prediction)

GROUP MEMBERS

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Core Facility Leader

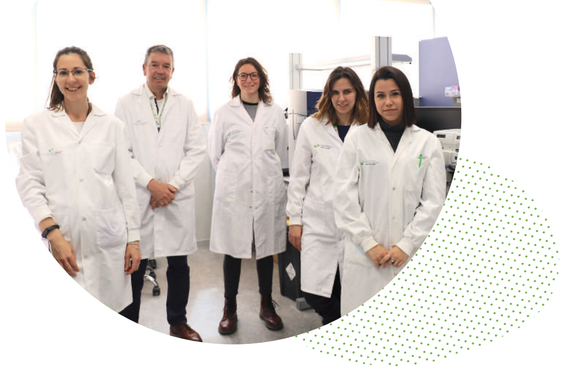
DE VILLASANTE LLAQUET, IZAR
Bioinformatician

BECCHI, LORENZO
Bioinformatician

FERNANDEZ REBOLLO, IRENE
Junior Researcher

ABOUT US

CORE FACILITIES



MICROARRAYS UNIT

The Microarrays Unit (UM) is a service focused on DNA and RNA microarray solutions towards a personalized medicine and participates in the Cytogenetic European Quality Assessment (CEQA).

MOLECULAR CYTOGENETICS

Microarray studies can offer various solutions for cytogenetic applications:

- Detection of whole genome gains and losses at a high resolution.
- Analysis of whole genome absences of heterozygosity.
- SNPs genotyping and genome-wide association studies.
- RNA Analysis Solution
- Gene expression profile studies on either human or mouse are suitable for:
- Detection of genes and pathways involved in diseases, treatment responses and biological processes.
- Predictive models based on gene expression profiles.
- Pharmacogenomics and toxicogenomics studies.
- Alternative splicing detection.
- Classification of samples on gene signatures.
- Analysis of miRNA.
- Microarray analysis on compromised samples with degraded and/or low quantity samples.
- Quality sample analysis.

Microarray studies can offer various solutions for cytogenetic applications:

- Detection of whole genome gains and losses at a high resolution.
- Analysis of whole genome absences of heterozygosity.
- SNPs genotyping and genome-wide association studies.
- RNA Analysis Solution
- Gene expression profile studies on either human or mouse are suitable for:
- Detection of genes and pathways involved in diseases, treatment responses and biological processes.

HIGH THROUGHPUT QPCR

The Biomark HD system is a high throughput qPCR that runs IFCs in either real-time or end-point read modes, bringing PCR solutions to a range of applications. The 48x48 Dynamic Array combines up to 48 samples and 48 assays, generating 2304 different assays in one single run. The 96x96 Dynamic Array combines up to 96 samples and 96 assays, generating 9216 different assays. In addition, the FLEXsix IFC incorporates six 12x12 partitions that can be organized in any configuration, in up to six separate experimental runs.

Applications

- Genotyping.
- Targeted Gene expression.
- Digital PCR.

Equipment

- Affymetrix/Thermofisher Research Platform: GCS3000 with autoloader.
- Agilent Bioanalyzer 2100.
- NanoDrop 2000 Spectrophotometer.
- Saphyr (Bionano) equipment for OGM. Renting.

GROUP MEMBERS

SOLE RISTOL, FRANCESC
Core Facility Coordinator

MALLO FAJULA, MARIA DEL MAR
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CUERO QUEZADA, IDALID
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SANTANA HERNÁNDEZ, JENNIFER
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DE HARO CAMPS, NURI
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COLETO MARTIN, PAULA
Core Facility Technician

TIJERO SANTOS, JESSICA
Core Facility Technician

ABOUT US

CORE FACILITIES

CYTOGENETICS UNIT

The Cytogenetics Unit in collaboration with the Hematology Service from Hospital ICO-Germans Trias i Pujol (Badalona) is responsible for analytical tests belonging to the Hematology Service from samples of whole blood, serum, plasma, urine, body fluids, bone marrow, lymph nodes, spleen, and tumor masses. The available analysis include Cytogenetics (karyotype), FISH and SNP-microarrays (in collaboration with Unit of Microarrays from IJC).

The Cytogenetics Unit includes the Laboratory of Cytogenetics of the Institut Català d'Oncologia (ICO) from Badalona. The Unit process more than 3000 samples per year from ICO Badalona, from ICO Girona and ICO Bellvitge.

Services

- Conventional cytogenetics culture and karyotype performance.
- Fluorescence in situ hybridization (FISH) with commercial probes.
- QF-PCR.

GROUP MEMBERS

SOLE RISTOL, FRANCESC
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ORIOL PUIG, LAIA
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CISNEROS SALA, ADELA
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SANTAFÉ COLLADO, ENCARNACIÓ
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SAMPLE HANDLING CIRCUIT UNIT

The Josep Carreras Leukaemia Research Institute (IJC) Can Ruti Location houses the Germans Trias I Pujol Hospital and Institute (IGTP-HUGTP) Sample Handling Circuit Unit, which manages the processing and storage of voluntarily donated samples of hematological neoplasms. The samples are stored in the collection entitled "IJC Leukemia and other blood disease Sample Collection. The IJC Sample Banking Unit receives the bulk of its samples from the Catalan Institute of Oncology at the Germans Trias i Pujol Hospital (ICO-HUGTP, Badalona). Samples received from other hospitals are processed in an identical way.

The technical staff of the IJC have created a database of patients, donors and samples received and processed according to required specifications for the tracking of each sample in the collection. The staff verify the quality, security and tracking of the data and samples throughout the process and starting at extraction. Every year the Unit process and cryopreserve approximately 1000 samples from patients with hematologic cancers.

GROUP MEMBERS

SOLE RISTOL, FRANCESC
Core Facility Coordinator

RUIZ CORTÉS, ROCÍO
Core Facility Technician

ARANDA CEBRIAN, JESSICA
Core Facility Technician

SILVERIO AYALA, AIDA
Core Facility Technician

ABOUT US

MANAGEMENT UNITS

STRATEGY AND ACTING MANAGING DIRECTOR

Garrido Anglada, Ana

Together with Dr. Manel Esteller, Ana Garrido is part of the management team that contributes to the development of general policy and strategic planning, enabling, and translating scientific vision and strategic objectives into a clearly articulated operational strategy.

The main objectives of the Strategy and Managing Director are:

- Strategic and operational organization of the Institute.
- Management, in accordance with the marked guidelines of the governing bodies, of human resources, hiring staff, incidents, separation and termination of employment contracts, HR organizational policies, as well as the management of scholarships and grants, encouraging policies of Corporate Social Responsibility and attracting and retaining talent.
- Detection of the needs derived from the activity of the Institute Research Groups and the rest of the units from the economic and management point of view.
- Implementation of an agile model focused on researchers that favors their performance and the best concentration in scientific activity.

Group members

GARRIDO ANGLADA, ANA
Strategy Director and Acting Managing Director

ABOUT US

MANAGEMENT UNITS

Research Grants Unit

Its objective is the attraction of public and private competitive funding, both nationally and internationally, as well as the proactive management of the granted research projects. We support researchers throughout the life cycle of projects, from the detection of opportunities, the preparation of proposals and the training of research consortiums, to the management of projects in all areas beyond the economic.

Group members

MORALES CUÑADO, CRISTINA
Project Manager

PADIAL MELIÁN, VERÓNICA
Project Manager

LAGUNAS VILA, LAIA
Project Manager

GIL GUIÑON, ESTEL
Project Manager

VILLANUEVA DELGADO, ANAÍ NOEMÍ
Project Manager

MARTINEZ ESCRIBANO, BEATRIZ
Project Manager

GARCIA GALAN, MARIA JESUS
Project Manager

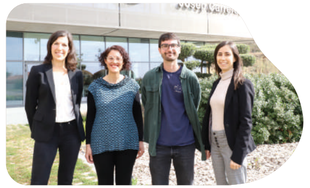
RODRIGUEZ AYUSO, NURIA PILAR
Project Manager

MANCUSO PONCE, CHIARA
Project Manager

DOLSET VILLALOBOS, SARAI
Project Support Officer

GARCIA MONTERO, ADRIÀ
Project Support Officer

ÁLVAREZ RIU, GUILLEM
Projects Assistant



Innovation Unit

In order to promote, maintain and invigorate knowledge and technology transfer at the Institute the main objectives of the Innovation Unit are: to establish a culture of innovation, valorization and translation of results among professionals; to promote the effective transfer of the research results for the benefit of health especially for leukemia patients, and to align the technology produced with the market and the industry.

To this end, the Innovation Unit explores the development of collaborative projects with centers and companies; providing specific training actions to research staff and management to improve and enhance the efficiency of public-private co-operation; systematizing communication both internally and externally; giving support to research in terms of intellectual property and innovation-related competitive calls, developing regulations according to current legislation, and promoting the diffusion and commercialization of its technology portfolio.

Group members

RIERA GUERRA, ANNA
Innovation Manager

FARRÉS ÀLVAREZ, CLARA
Innovation Officer

MARTIN TARIN, ELVIRA
Innovation Officer

MIALET RIU, MARC
Innovation Officer

Enocomic Management Unit

The Economic Development Manager manages and coordinates the financial control of the Institute. Its main functions are linked to the control and supervision of finances and, ultimately, it acts as a link between the Institute's management and accounting.

Group members

BOIX MONTEMAYOR, HEURA
Economic Development Manager

ME Business Development Unit

The ME Business Development Manager is in charge of the Institute's growth strategy in the Middle East Region. She analyzes and identifies new opportunities for the Institute to expand by developing partnerships with key international actors in the health and research sector of the ME region.

Group members

EL-GHAUCHE EL-HALLAK, RANIA
International Business Development Manager

Human Resources Unit

Its mission is to plan, organize and execute all processes related to the professional development of the staff and their commitment with the organization, within the framework of current regulations, including training opportunities and occupational risk prevention.

Group members

CHICO GENEROSO, LETICIA B.
HR Manager

VARGAS SOLETO, BRIAN
HR Officer

LATORRE REQUELME, IRENE
HR Officer

SOUIRJI GOMEZ, SOFIA
HR Officer

ROMERO JIMENEZ, MARTA
HR Officer

Finance Unit

Its mission is to keep track of the actual finance situation of the Institute, rigorously and transparently, to support data driven strategic decision making in the short, medium, and long run.

Group members

CALONGE CORTÉS, MARIA CRISTINA
Finance Manager

FINESTRES MARTINEZ, XAVIER
Finance Officer

VILANOVA CUADRA, YAIZA
Finance Officer

MURE FERNANDEZ, MIREIA
Finance Officer

GARCIA SEGUER, ANA CARINA
Finance Officer

MATOS SILVA, AWILDA
Finance Officer

Purchasing Unit

The unit's aim is to optimize purchasing at the institutional level according to the legal framework for public research bodies, to be more efficient, fast, and agile, avoid unnecessary costs and save resources for research.

Group members

REYES IBORRA, LAIA
Purchasing Manager

VERGÉS COLOMINAS, ANNA
Purchasing Officer

MONTSERRAT SANCHEZ, QUIQUE
Purchasing Officer

NIUBÓ BALCELLS, NURIA
Purchasing Officer

IT Unit

The unit's objective is to support the Institute's staff in the use and purchasing of IT components -hardware, software, and systems- and keep the institutional IT systems online and secure while ensuring its efficiency.

Group members

JUBANY LÓPEZ, MARC
IT Manager

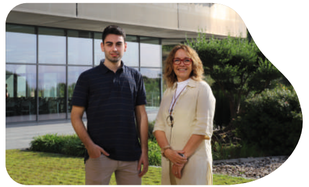
CONTRERAS PEÑA, FRANCISCO
IT Technician

ALCANTÁRA RUIZ, JOSE ANTONIO
IT Technician

GALLARDO PEREZ, DIANA
IT Technician

DIAZ LOPEZ, SERGI
IT Assistant

BOLAÑOS, ABRAHAM
IT Assistant



Data Management Unit

Aimed at delivering and maintaining all necessary infrastructure to efficiently keep institutional data available at all levels: strategic, technical, administrative and for transparent accountability in front of local or international management bodies.

Group members

CARRIO REIG, MARTA
Data Manager

DE HIGES ALBERICH, PAU TADAYUKI
Data Officer

Communication Unit

Passionate about spreading the latest discoveries of our scientists and bring their research efforts closer to society, in any form. The unit strives to keep our partners closer and updated, and to foster the staff's sense of belonging.

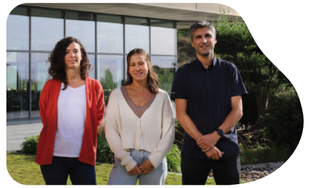
Group members

DÍAZ LÓPEZ, HELENA
Communication Manager

BADAL SOLER, MARTI
Communication Officer

BERZOSA FERNÁNDEZ, BEATRIZ
Communication Officer

OLMO GONZÁLEZ, AINOA
Communication Officer



Lab Management Unit

The aim is to support researchers in their daily work in laboratories so that their research can be carried out with the best equipment, in the best state and with maximum safety.

Group members

PEREZ LADAGA, ALBERT
Lab Manager

GARCIA FERRAN, ALBA
Lab Technician

MORENO ZAMBRANA, ELISABET
Lab Technician

Legal Services

Its mission is to support the Institute in all legal matters, particularly in establishing the collaboration framework with partner institutions, ongoing clinical trials, and innovation aspects in biomedical research.

Group members

LAFARGA TRAVER, JOSEP LLUIS
Legal Manager

Facilities Unit

This Unit assures the proper functioning of all the facilities of the Institute. It coordinates facility maintenance and janitorial services and guarantees efficient and effective delivery of logistics for on-site activities.

Group members

ARQUE COMAS, CARLES
Facilities Manager

CARREÑO PEREZ, JUAN
Facilities Officer

PEREZ GARCIA, MARIA ISABEL
Receptionist

FERNÁNDEZ GARCÍA, SANDRA
Receptionist

Support Unit

The Support Unit supports the governing bodies of the Institute in daily administrative tasks, agendas as well as in travel procedures.

Group members

MARIN MANZANERA, ESPERANZA
Management Assistant

IZQUIERDO SÁNCHEZ, IRMA
Management Administrative Assistant

FARRÉ VIADER, LAURA
Management Assistant

Travel Unit

The Travel Unit facilitates the international mobility of the Institute's researchers by managing flights and hotels bookings as well as any other travel-related costs in an effective and cost-efficient manner.

Group members

AMADO BALLANO, ERIKA
Travel Administrative Assistant

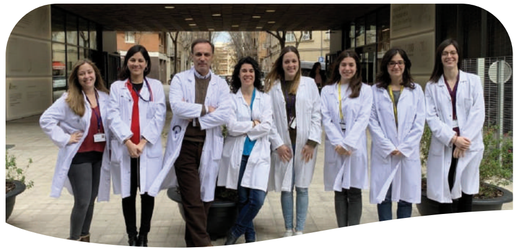
MATOS BERGADA, LAURA
Travel Administrative Assistant

COMMUNICATION

SELECTED PRESS RELEASES

APRIL

The hidden cells behind B-cell acute lymphoblastic leukemia relapse spotlighted for the first time. A previously unnoticed population of pre-leukemic cells might be responsible for some relapses in B-cell acute lymphoblastic leukemia (B-ALL), according to a new research paper published by a team led by Dr. Pablo Menéndez, group leader at the Josep Carreras Leukaemia Research Institute, in close collaboration with Oxford University and clinicians from Sant Joan de Déu Hospital, Hospital Clinic y Universidad de Salamanca, as well as the Spanish group Pethema.



APRIL

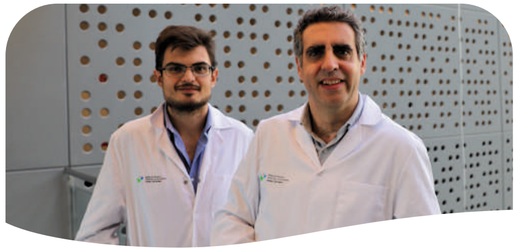
Phase I of Europe's first self-produced CAR-T30 clinical trial for Hodgkin's and non-Hodgkin's lymphoma ends. The first trial with a CAR-T immunotherapy drug produced in Sant Pau, a pioneer in Europe, for the treatment of classic Hodgkin's lymphoma and non-Hodgkin T lymphoma CD30 + in relapse or refractory, has successfully completed its Phase I. The project is led by Dr. Javier Briones, Head of the Clinical Hematology Unit of the Hematology Service of the Hospital de Sant Pau and Head of the Cell Immunotherapy and Gene Therapy Research Group of the Research Institute of the Hospital de Sant Pau - IIB-Sant Pau and the Josep Carreras Leukaemia Research Institute.

COMMUNICATION

SELECTED PRESS RELEASES

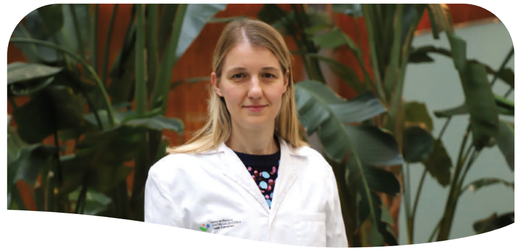
MAY

The Josep Carreras Leukaemia Research Institute has been selected by the National Cancer Institute for the Cancer Proteome Project with the support of the Spanish Ministry of Science and Innovation. The Josep Carreras Institute becomes part of the Cancer Proteome project, an international initiative for the study of malignant tumors. It has the support of the Spanish Ministry of Science and Innovation, whose Minister, Diana Morant, visited the Josep Carreras Institute on Thursday and announced a grant of one million euros to promote the project.



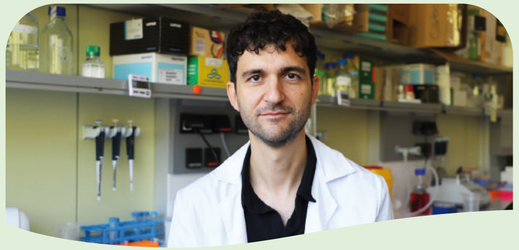
JUNE

A mechanism is found explaining how cancer cells turn into normal harmless ones. A new research describes how highly proliferative leukemia cells end up becoming normal cells that no longer multiply, by changing the chemical modifications -the so-called epigenetics- of a type of its genetic material: the messenger RNA. The article, published in the high-impact journal Leukemia, is authored by Alberto Bueno-Costa, researcher at the group of Dr. Manel Esteller, supervisor of the research and Director of the Josep Carreras Leukaemia Research Institute, ICREA Researcher and Professor at the University of Barcelona.



JULY

Researchers at the Josep Carreras Institute will study a rare T-cell lymphoma thanks to the Leukemia Research Foundation. Dr. Laura Mondragón, "T-cell lymphoma" group leader at the Josep Carreras Leukaemia Research Institute, has been granted a new project to fight against angioimmunoblastic T cell lymphoma (AITL). The project, starting October 1st 2022, is funded by the Leukemia Research Foundation based in Northfield, Illinois (USA) and aims to exploit the latest generation of animal models for AITL, to better understand this type of adult lymphoma and open the door to new therapeutic approaches.

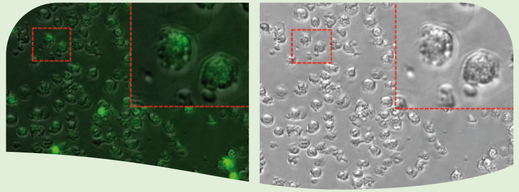


JULY

A new project aims to shed new light on a frequent form of leukemia in Down Syndrome children. The American Society of Hematology (ASH) has selected a project led by Dr. Sergi Cuartero, researcher at the Josep Carreras Leukaemia Research Institute, to receive the 2022 ASH Global Research Award. Dr. Cuartero is one of 13 talented early-career investigators selected for this honor. The project aims to better understand the molecular basis of myeloid leukemia of Down Syndrome (ML-DS) and contribute to the identification of novel actionable targets for therapeutic use in ML-DS.

COMMUNICATION

SELECTED PRESS RELEASES



AUGUST

Leukemia vulnerability discovered causing drug sensitivity. The article, published in the journal Redox Biology by the group of Dr. Manel Esteller, shows that epigenetic changes prevent iron-associated programmed cell death in leukemia and show a new target for treatment with experimental drugs.

DECEMBER

Eight researchers from the Josep Carreras Institute among the most relevant on an international level according to Elsevier. Manel Esteller, Josep Maria Ribera, Esteban Ballestar, Alejandro Vaquero, Montse Sánchez-Cespedes, María Berdasco, Fumiichiro Yamamoto and Ciril Rozman, together with other nearly twenty researchers and medical staff from Can Ruti Campus institutions, are included in Elsevier's Scopus list, composed by the 200,000 most renowned researchers worldwide.

DECEMBER

Epigenetics breaks into the clinical practice of cancer. Dr. Manel Esteller and Dr. Verónica Dávalos, researchers at the Josep Carreras Leukaemia Research Institute, describe in a new article the impact of epigenetics on cancer treatment and how it has become a crucial tool to improve early detection, predict disease progression and become a target for new treatments.

DECEMBER

World's first clinical trial with CAR-T technology for patients with T-cell leukaemia subtype authorized. OneChain Immunotherapeutics (OCI), spin-off founded by Dr. Pablo Menéndez and the Josep Carreras Leukaemia Research Institute, has obtained authorization for the CARxALL clinical trial to evaluate a new CAR-T therapy for patients who have T-cell leukemia without therapeutic alternatives.

COMMUNICATION

SCIENTIFIC DISSEMINATION

The Josep Carreras Leukaemia Research Institute is a public institution with a strong commitment towards society. On this regard, the Institute is constantly seeking for new ways of disseminating the results and impacts of our research. It is precisely for this reason that bringing the research of the Josep Carreras Institute closer to the public is one of our fundamental values.

2022 has seen a major upgrade of our outreach activities, with the opening of new channels (Instagram) and the strengthening of our social media outputs with a new dedicated community manager. As a result, the Institute has grown in social base, impact and recognition in a measurable way, helping position the Josep Carreras Research Institute as a pole of scientific excellence among the public.

New initiatives have been developed and implemented on social media, like science-based threads on special days, encouraging our followers to ask questions to our researchers and the creation of a set of cartoon characters that will help us explain basic concepts to a wider and younger audience in an easy and understandable way.

In addition, we participate in the main scientific dissemination events of Barcelona and Catalonia through talks and workshops. These actions are indicated in the list below:

- **UB Science Festival,** organized by the University of Barcelona, where Dr. Esteller is chairman of genetics, we presented a workshop aimed at understanding the vulnerabilities of cancer cells.
- **Barcelona Science Festival,** organized by the City Council through the Barcelona Institute of Culture (ICUB): talk by Dr. Lucas Pontel and a workshop on CAR-T cells.
- **European Research Night,** organized by the Catalan Association for Scientific Communication with funding from the European program MSCA: a workshop on the basics of leukemia translational research, by the Dissemination Specialist of the Institute.
- **#100tifiques, organized by the FCRi and the BIST:** networking and talks in schools by researchers from the center on the day of Women and Girls in Science, February 11th.

In addition, all the Conferences and Seminars organized by the Institute have been transformed into hybrid events with online streaming. These talks are delivered by national and international speakers and have the aim to facilitate access to the latest developments in leukemia research.

The Josep Carreras Leukemia Research Institute has maintained its collaboration with the Josep Carreras Foundation, with whom we have close ties. Every year, the Josep Carreras Foundation celebrates the Unstoppable Day and the Week against Leukemia, organizing activities for patients, relatives and civil society. The 2022 edition, still due to the restrictions imposed by the COVID-19 pandemic, all the events went fully online in the form of TV shows. IJC researchers participated with a rich program of presentations, experiences, testimonials and other online activities through their social networks. A few of the shows were held at the institute's auditorium, with the one corresponding to science in society being conducted by the communication unit's staff.

FACTS & FIGURES

INNOVATION

The **Josep Carreras Institute** is committed to bringing the cure of leukemia and other haematological malignances to patients. To do so and contribute to society and country's progress, it fosters the generation of new knowledge together with the development of new therapies and technologies.

INDICATORS 2022

In 2022, the results of our research in terms of innovation were as follows:

69
Active Innovation Projects

2
Active Spin-Offs: Leukos Biotech and One Chain Immunotherapeutics

15
Patent families Portfolio (47% Licensed)

13
Proof of Concept Competitive calls granted*

1,7M€
1.7M€ in granted PoC calls

79
New innovation-related agreements signed in 2022

854K€
854K€ raised through Agreements

* ERC Proof of Concept Grant (European Commission), Proyectos estratégicos orientados a la transición ecológica y a la transición digital, TED (MICIN); Líneas estratégicas (MICIN); Prueba de Concepto (MICIN), AJUTS D'INDÚSTRIA DEL CONEIXEMENT, PRODUCTE i AJUTS PER A PROJECTES DE VALORITZACIÓ I TRANSFERÈNCIA DE CONEIXEMENT, INNOVADORS (AGAUR), Gínjol (i-CERCA), i4KIDS Valorisation Program, Programa de Apoyo a la Innovación (ITEMAS), and additional competitive research calls that have funded proof of concept projects: RETT Syndrome Innovation Award, Deutsche José Carreras Leukämie Stiftung, Ayudas Merck de Investigación, Hollis Brownstein Research Grants Program (Leukemia Research Foundation)

FACTS & FIGURES

INNOVATION

The Josep Carreras Institute has consolidated ...

In 2020 the Josep Carreras Institute made a strong commitment to innovation launching its own unit which has resulted in 2022 with more than 2,6 million euros raised only through innovation activity, a 60% increase respect 2021. The Institute closed 2022 with almost 70 active projects, 15 patents (almost half of them already licensed), 3 new licenses in 2022 and 2 spin-off companies, Leukos and OneChain Immunotherapeutics. This last company, founded only two years ago, has obtained authorisation for clinical trial to evaluate a new CAR-T therapy against T-cell leukaemia.

Up to 10 Institute's research projects have been granted with more than 1,7 million euros of competitive calls in order to get a proof of concept needed to attract private investors and pharma/biotech industry interest and ultimately reach patients. The quality and impact of these 10 projects have been validated by, among others, the European Comission (through ERC Proof of Concept call), the Spanish Ministry of Science and Innovation (trough TED, Líneas estratégicas and Prueba de concepto calls), the Catalan Government by the Agency for Management of University and Research Grants (through Producte and Innovadors calls).

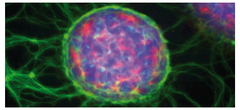
On the other hand, the Institute has strenghtened the alliance with international biotech and Pharma companies in order to codevelop new therapeutic and diagnostic approaches, which has led to 465.000 euros of private investment in research projects.

Finally, in 2022 the Institute has consolidated a disruptive tool of fundraising, called the Economic Interest Grouping (AIE). Thanks to the collaboration with consulting companies, the Institute raised more than €300,000 in 2021 (in a research project valued at more than €1M) and in 2022 €388,000 (in a 1,3M€ valued project). In 2022, with the patronage of AXA Insurance company, and the collaboration of Adastra Capital and LKS Next, the project "Proteogenomic profiling of myelodysysplastic syndromes with micromegakaryocytes", led by Dr Gaël Roué, was performed successfully.

Teaching and training

INTERNATIONAL CONGRESSES

In 2022, the Institute co-organized, together with the Centre for Genomic Regulation (CRG) and the Institute for Research in Biomedicine (IRB), the 1st Single Cell Genomics Symposium, which took place at the PRBB. This event aimed to bring together researchers from Barcelona working in Single-Cell Genomics and global leaders of the field in order to foster new local and international research alliances.



March. 24th & 25th, 2022
"Single Cell Genomics Symposium" Congress.

YOUNG RESEARCHERS SEMINARS

(29)

The Young Researchers Seminars are 20-min talks given by our PhD Students and young Postdoctoral Investigators, in which they explain an aspect of their research to their IJC fellows and respond to their questions. This is the perfect opportunity for them to practice an activity that they will have to face not only in their thesis defense, but also on numerous occasions throughout their research career.

THESIS READ

(7)

CURRENT DOCTORAL THESIS

(64)

FACTS & FIGURES

TEACHING AND TRAINING

Distinguished and Invited lectures

The Institute has the pleasure to receive national and international well-renowned researchers in the cancer research-related field. They deliver a 1-hour lecture on their research, career, and findings, which is open to all the Institute and the scientific community.

LECTURES
At the Josep Carreras Leukaemia Research Institute

1st SEMESTER 2022

4 FEB, 15h — DISTINGUISHED LECTURE
BENJAMIN EBERT
Dana-Farber Cancer Institute, USA
"Targeted protein degradation for the treatment of cancer"

11 FEB, 12h — INVITED LECTURE
CRISTINA MAYOR
Institute for Research in Biomedicine (IRB Barcelona), Spain
"Targeted protein degradation: genetic determinants and drug discovery opportunities"

29 APR, 12h — INVITED LECTURE
TONI CELIÀ-TERRASSA
Hospital del Mar Medical Research Institute (IMIM), Spain
"Stem cell properties in breast cancer immunotherapy resistance"

6 MAY, 12h — INVITED LECTURE
Mª JOSÉ ALONSO
USC CIMUS Research Institute, Spain
"Nanotechnology has been critical in the development of mRNA COVID vaccines"

13 MAY, 12h — INVITED LECTURE
LUIS PAZ-ARES
Hospital Universitario 12 de Octubre, Spain
"Are we curing in Lung Cancer?"

27 MAY, 12h — DISTINGUISHED LECTURE
JAN COOLS
VIB-KU Leuven Center for Cancer Biology, Belgium
"Oncogene cooperation in T-cell acute lymphoblastic leukemia"

17 JUN, 12h — DISTINGUISHED LECTURE
JANE SKOK
NYU Langone Medical Center, USA
"Architectural proteins and their role in chromatin organization and gene regulation"

DISTINGUISHED & INVITED LECTURES
Josep Carreras Leukaemia Research Institute

2nd SEMESTER 2022

SEP 16 — **Dr. FLORIAN HEIDEL**
University of Greifswald, Germany
"Signaling landscape define dependencies of JAK2-mutated clones"

OCT 21 — **Dr. JONATHAN LICHT**
The University of Florida Health Cancer Center, USA
"Aberrant histone methylation in lymphoid malignancies"

OCT 28 — **Dr. MARIA CASANOVA-ACEBES**
Spanish National Cancer Centre (CNIO), Spain
"Redefining Myeloid cell Functions in the Tumor Microenvironment"

NOV 04 — **Dr. LAURA M. LECHUGA**
Catalan Institute of Nanoscience and Nanotechnology (ICN2), Spain
"Nanobiosensor devices for the early diagnosis of cancer"

NOV 18 — **Dr. DOMINIQUE BONNET**
The Francis Crick Institute, UK
"The Bone marrow niche: A supportive ecosystem for haematopoiesis and malignant cell growth"

NOV 25 — **Dr. JESÚS SAN-MIGUEL**
Universidad de Navarra, Spain
"The Pathway to cure Myeloma: A long and winding road"

Distinguished Lecture Invited Lecture

OPEN LECTURES
Josep Carreras Leukaemia Research Institute

2nd SEMESTER 2022

SEP 19 — **Dr. ROLF MARSCHALEK**
Institute of Pharmaceutical Biology, Goethe University. Frankfurt am Main, Germany
"MLL/KMT2A leukemia - insights into the disease pathology"

OCT 11 — **Dr. AMER ZEIDAN**
Yale University School of Medicine. New Haven, USA
"Immune checkpoint inhibition for AML and MDS: Is there a way forward?"

OCT 25 — **Dr. PAU CREIXELL**
Cancer Research UK. Cambridge Institute. Cambridge University. Cambridge, UK
"Pro- and Anti-oncogenic Tyrosine Kinase-driven Signaling in Leukemia"

OCT 28 — **Dr. VERA PANCALDI**
INSERM Cancer Research Center of Toulouse (CRCT). Toulouse, France. Barcelona Supercomputing Center. Barcelona, Spain
"Describing and modelling the tumour microenvironment using spatial multi-omics approaches"

NOV 2 — **Dr. BERNHARD PAYER**
Centre for Genomic Regulation (CRG). Barcelona, Spain
"Epigenetic reprogramming linked to pluripotency and germ cell fate"

NOV 16 — **Dr. JOHN DICK**
Princess Margaret Cancer Centre, University Health Network. Toronto, Canada
"Development of a hierarchy-based classification system in human leukemia"

DEC 1 — **Dr. ALEX DE MENDOZA**
School of Biological and Behavioural Sciences, Queen Mary University of London. London, UK
"Deciphering the roles of cytosine DNA methylation through epigenome engineering and evolutionary approaches"

COURSES AND SEMINARS

TRAINING COUSES

Researchers from the Institute periodically offer highly-specialized sciences courses.

JUNE 14-16

- **Cytogenetics and Molecular Cytogenetics of Hematological Neoplasms**

NOVEMBER 25

- **Preceptorship. Advances in Acute Lymphoblastic Leukemia**

SEMINARS

FEBRUARY

- **Distinguished Lecture: "Targeted protein degradation for the treatment of cancer"** Dr. Benjamin Ebert, Harvard Medical School; Dana-Farber Cancer Institute; Howard Hughes Medical Institute. Boston, USA.
- **Invited Lecture: "Targeted protein degradation: genetic determinants and drug discovery opportunities"** Dr. Cristina Mayor-Ruiz, Institute for Research in Biomedicine (IRB). Barcelona, Spain.

FACTS & FIGURES

COURSES AND SEMINARS

APRIL

- **Invited Lecture: "Stem cell properties in breast cancer immunotherapy resistance"** Dr. Toni Celià-Terrassa, Cancer Stem Cells & Metastasis Dynamics Lab Hospital del Mar Medical Research Institute (IMIM) Barcelona Biomedical Research Park (PRBB). Barcelona, Spain.

MAY

- **Open Lecture: "Computational proteomics of cancer cell signalling"** Dr. Pedro Rodríguez-Cutillas, Centre for Genomics and Computational Biology, Barts Cancer Institute, Queen Mary University of London. London, UK
- **Invited Lecture: "Nanotechnology has been critical in the development of mRNA COVID vaccines"** Dr. María José Alonso, CIMUS Research Institute. Campus Vida - University of Santiago de Compostela. Santiago de Compostela, Spain
- **Invited Lecture: "Are we curing in Lung Cancer?"** Dr. Luís Paz-Ares, Hospital 12 de Octubre. Madrid, Spain
- **Distinguished Lecture: "Oncogene cooperation in T-cell acute lymphoblastic leukemia"** Dr. Jan Cools, VIB-KU Center for Cancer Biology. Leuven, Belgium.

JUNE

- **Open Lecture: "Snapshots of Divergent Evolution Towards Sex Chromosome-Specific Gene Regulation"** Dr. Peter Becker, Ludwig-Maximilians-University of Munich. Munich, Germany.
- **Distinguished Lecture: "The impact of cancer associated CTCF mutations and CTCFL on chromatin architecture and gene regulation"** Dr. Jane Skok, Sandra and Edward Meyer Professor Department of Pathology. Perlmutter Cancer Center, New York University School of Medicine. New York, USA.
- **Open Lecture: "Leveraging the tumor microenvironment to combat cancer"** Dr. Hind Medyouf, Institute for Tumor Biology and Experimental Therapy "Georg-Speyer-Haus". Frankfurt, Germany.

SEPTEMBER

- **Distinguished Lecture: "Signaling landscape define dependencies of JAK2-mutated clones"** Dr. Florian Heidel, University of Greifswald. Greifswald, Germany.
- **Open Lecture: "MLL/KMT2A leukemia - insights into the disease pathology"** Dr. Rolf Marschalek, Institute of Pharmaceutical Biology, Goethe University. Frankfurt am Main, Germany.

OCTOBER

- **Open Lecture: "Immune checkpoint inhibition for AML and MDS: Is there a way forward?"** Dr. Amer Zeidan, Yale University School of Medicine. New Haven, USA.
- **Distinguished Lecture: "Aberrant histone methylation in lymphoid malignancies"** Dr. Jonathan Licht, University of Florida Health Cancer Center. Gainesville (Florida), USA.
- **Open Lecture: "Pro- and Anti-oncogenic Tyrosine Kinase-driven Signaling in Leukemia"** Dr. Pau Creixell, Cancer Research UK. Cambridge Institute. Cambridge University. Cambridge, UK.
- **Open Lecture: "Describing and modelling the tumour microenvironment using spatial multi-omics approaches"** Dr. Vera Pancaldi, INSERM Cancer Research Center of Toulouse (CRCT). Toulouse, France. Barcelona Supercomputing Center. Barcelona, Spain
- **Invited Lecture: "Redefining Myeloid cell Functions in the Tumor Microenvironment"** Dr. Maria Casanova-Acebes, Spanish National Cancer Centre (CNIO). Madrid, Spain.

NOVEMBER

- **Open Lecture: "Epigenetic reprogramming linked to pluripotency and germ cell fate"** Dr. Bernhard Payer, Centre for Genomic Regulation (CRG). Barcelona, Spain.
- **Workshop: Mission Bio Symposium, User Group Meeting** Mission Bio
- **Invited Lecture: "Nanobiosensor devices for the early diagnosis of cancer"** Dr. Laura M. Lechuga, Catalan Institute of Nanoscience and Nanotechnology (ICN2). Bellaterra, Spain.
- **Workshop: "10 working days to get your NGS data in BGI"** Zhengyu Xiao, BGI
- **Open Ad-Hoc Lecture: "Development of a hierarchy-based classification system in human leukemia"** Dr. John Dick, Princess Margaret Cancer Centre, University Health Network. Toronto, Canada.
- **Distinguished Lecture: "The bone marrow niche: A supportive ecosystem for haematopoiesis and malignant cell growth"** Dr. Dominique Bonnet, The Francis Crick Institute. London, UK.
- **Invited Lecture: "The Pathway to cure Myeloma: A long and winding road"** Dr. Jesús San-Miguel, University of Navarra. Pamplona, Spain.

DECEMBER

- **Open Lecture: "Deciphering the roles of cytosine DNA methylation through epigenome engineering and evolutionary approaches"** Dr. Alex de Mendoza, School of Biological and Behavioural Sciences, Queen Mary University of London. London, UK.

FACTS & FIGURES

INSTITUTIONAL EVENTS

MANAGEMENT RETREAT

OCTOBER 6, 2022

The Josep Carreras Institute's Management Retreat served to create a shared vision among all staff categories within the organization. At this retreat, which was held at the Masia Cal Riera, our management staff collaborated with colleagues, engaged in meaningful leadership activities, and built management team excellence.

SCIENTIFIC RETREAT

OCTOBER 13, 2022

The Institute's Scientific Retreat was held at the Museu de la Ciència CosmoCaixa, and participants talked about the past, present, and future of the Josep Carreras Institute. It was the perfect opportunity for our researchers to share their insights about the work done during the year and gain a deeper knowledge of the projects of their closest colleagues.

FINANCIAL DATA

The Institute's pioneering mixed-funding model is partially financed by the Josep Carreras Foundation. It also receives core funding from the Catalan government and is reliant on competitive funding for its research activities.

In 2022, there was a **15,34%** increase in income from public funds and the provision of services.

With respect to spending, this one increased by **19,30%** compared to the previous year.

	2021	2022	
INCOMES	16.292.754	18.792.033	15,34%
CONTRIBUTIONS FROM THE GENERALITAT	3.587.060	4.089.268	
OTHER TRANSFERS (FIJC)	750.000	1.000.000	
SERVICES	3.033.238	3.263.095	
PROJECT	7.417.512	9.103.001	
OVERHEADS	1.504.945	1.336.669	
OPERATIONAL EXPENSES	14.349.820	17.119.712	19,30%
STAFFING COSTS	4.026.115	4.461.440	
INFORMATION TECHNOLOGIES SERVICES	161.536	153.875	
COMMUNICATION	31.938	35.403	
BUILDING MAINTENANCE	949.738	1.158.743	
LABORATORIES MAINTENANCE	214.312	262.241	
RESEARCH SUPPORT (travels, catering...)	26.273	105.120	
PROJECT	7.846.698	9.830.449	
SCIENTIFIC-TECHNICAL SERVICES (Platforms)	363.967	595.845	
BIOBANK	16.565	13.669	
MANAGEMENT SUPPORT SERVICES	263.925	224.037	
OTHER	67.841	129.047	
VAT PRORATA	80.454	97.827	
EXPENDITURE ON INVESTMENTS PENDING ACTIVATION			
HERITAGE	2.342		
REIMBURSEMENT OF SUBSIDIES AND OTHER MANAGEMENT LOSSES	298.116	52.018	
RESULT OF THE ACTIVITY	1.942.934	1.672.321	
EXTRAORDINARY RESULT	0	0	
OPERATING INCOME	1.942.934	1.672.321	
FINANCIAL PERFORMANCE	-1.107.638	-1.369.742	
RESULT BEFORE AMORTIZATION	835.296	302.579	
Amortization	-1.527.277	-1.933.451	
RESULT	**-691.982**	**-1.630.872**	

COMPETITIVE GRANTS AWARDED AND ACTIVE PROJECTS

Cancer Epigenetics led by Manel Esteller

Type: HR
2020 Ministerio de Ciencia e Innovación, AYUDAS JUAN DE LA CIERVA FORMACIÓN 2020
ESTELLER BADOSA, MANEL
Reference: FJC2020-044658-I
Title: **Single cell analysis of clonal heterogeneity in myelodysplastic syndromes treated with azacitidine**
Start Date: 01/07/2022 - **End Date:** 30/06/2024
Granted amount: 52.600,00€

Type: Project
2021 Fundació "La Caixa", CAIXARESEARCH HEALTH 2022
ESTELLER BADOSA, MANEL
Reference: HR22-00732
Title: **Somatic mutations and clonal hematopoiesis as predictors and drivers of heart failure progression**
Start Date: 01/10/2022 - **End Date:** 30/09/2025
Granted amount: 300.000,00€

Type: Project
2021 Ministerio de Ciencia e Innovación, PROYECTOS DE GENERACIÓN DE CONOCIMIENTO 2021
ESTELLER BADOSA, MANEL
Reference: PID2021-125282OB-I00
Title: **Uso de aproximaciones de célula única para decifrar la epigenómica del cáncer y las epidrogas**
Start Date: 01/09/2022 - **End Date:** 30/08/2025
Granted amount: 471.900,00 €

Type: Project
2021 Ministerio de Ciencia e Innovación, AYUDAS A PROYECTOS ESTRATÉGICOS ORIENTADOS A LA TRANSICIÓN ECOLÓGICA Y A LA TRANSICIÓN DIGITAL 2021
MUSULÉN PALET, EVA
Reference: TED2021-131248B-I00
Title: **AlgoRitmoS de aprEndizaje Profundo en el diagnóstico de adenomas y del cáNcer cOlorrectal precoz**
Start Date: 01/12/2022 - **End Date:** 30/11/2024
Granted amount: 280.140,00€

Type: HR
2021 European Commission, MSCA COFUND 2021
ESTELLER BADOSA, MANEL
Reference: 101081298

FACTS & FIGURES

COMPETITIVE GRANTS AWARDED AND ACTIVE PROJECTS

Title: **FCAECC Fellowship programme for talented researchers in cancer**
Start Date: 01/09/2023 - **End Date:** 31/08/2028

Type: Project
2022 Agència de Gestió d'Ajuts Universitaris i de Recerca, AJUTS D'INDÚSTRIA DEL CONEIXEMENT PER A L'ANY 2021 (LLAVOR I PRODUCTE)
ESTELLER BADOSA, MANEL
Reference: 2021 PROD 00020
Title: **Development and validation of a DNA methylation signature for predicting the response to chimeric antigen receptor (CAR)-T cell therapy**
Start Date: 19/10/2022 - **End Date:** 18/04/2024
Granted amount: 100.000,00€

Type: Project
2022 Agència de Gestió d'Ajuts Universitaris i de Recerca, AJUTS PER A PROJECTES DE VALORITZACIÓ I TRANSFERÈNCIA DE CONEIXEMENT DESENVOLUPATS PER INNOVADORS EN ESTADES EN ENTITATS DEL SISTEMA DE RECERCA I INNOVACIÓ DE CATALUNYA (INNOVADORS) PER A L'ANY 2021
ESTELLER BADOSA, MANEL
Reference: 2021 INNOV 00011
Title: **Creation of a spin-off dedicated to the study of genetic and rare diseases through the immortalization of lymphocytes and derived biological material.**
Start Date: 07/12/2022 - **End Date:** 06/06/2024
Granted amount: 84.000,06€

Type: HR
2022 Fundación Científica de la Asociación Española Contra el Cáncer, PRÁCTICAS DE LABORATORIO AECC VERANO 2022
ESTELLER BADOSA, MANEL
Start Date: 01/08/2022 - **End Date:** 30/09/2022
Granted amount: 2.000,00€

Type: Project
2022 Ministerio de Ciencia e Innovación, PRUEBA DE CONCEPTO 2022
ESTELLER BADOSA, MANEL
Reference: PDC2022-133476-I00
Title: **Study of validation and valorisation to the market of EPICART, a signature for predicting the response to CAR T-cell therapy (EPICART2M)**
Start Date: 01/12/2022 - **End Date:** 30/11/2024
Granted amount: 148.005,00€

Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS PARA CONTRATOS RAMÓN Y CAJAL 2021
ESTELLER BADOSA, MANEL
Reference: RYC2021-032395-I
Title: **The role of metabolism in disease aetiology**
Start Date: 01/01/2023 - **End Date:** 31/12/2027
Granted amount: 236.350,00€

Type: HR
2022 Instituto de Salud Carlos III, Sello de excelencia ISCIII-HEALTH-Acciones individuales MSCA
ESTELLER BADOSA, MANEL
Reference: IHMC22/00035
Title: **Epitranscriptomic regulation of DNA methylation in Acute myeloid leukemia**
Granted amount: 163.728,68€

Title: Project
2021 Ministerio de Ciencia e Innovación, PLAN COMPLEMENTARIO DE BIOTECNOLOGÍA APLICADA A LA SALUD DEL PLAN DE RECUPERACIÓN, TRANSFORMACIÓN Y RESILIENCIA
PONTEL, LUCAS BLAS
Title: **Precision Medicine in FA: drug screening to identify a mutation specific drug**
Start Date: 31/01/2023 - **End Date:** 31/12/2024
Granted amount: 73.075,00€

Cancer Genetics led by Montse Sanchez-Cespedes

Type: HR
2021 Ministerio de Universidades, CONTRATOS PREDOCTORALES PARA LA FORMACIÓN DE PROFESORADO UNIVERSITARIO-FPU 2021
SANCHEZ CESPEDES, MONTSE
Reference: FPU21/00047
Title: **Inactivación genética de moléculas involucradas en la represión transcripcional: análisis funcional y papel en el desarrollo del cáncer de pulmón**
Start Date: 01/01/2023 - **End Date:** 30/04/2025
Granted amount: 79.705,00€

Chromatin Biology led by Alex Vaquero

Type: HR
2021 European Commission, MSCA POSTDOCTORAL FELLOWSHIPS 2021
VAQUERO GARCÍA, ALEJANDRO
Reference: 101065013
Title: **Role of the SIRT7-NPM-c-Myc pathway in lung cancer**
Start Date: 01/09/2023 - **End Date:** 31/08/2025
Granted amount: 165.312,96€

Type: HR
2022 Agència de Gestió d'Ajuts Universitaris i de Recerca, Ajuts per a la contractació de personal investigador novell (FI-2022)
VAQUERO GARCÍA, ALEJANDRO
Reference: 2022 FI_B 00924
Title: **Role of sirtuins in epigenetic regulation and genome integrity in stress respnse and their implication in cancer and aging**
Start Date: 01/07/2022 - **End Date:** 30/06/2025
Granted amount: 69.508,40€

Chromatin, Metabolism and Cell Fate led by Marcus Buschbeck

Type: HR
2022 Fundación Científica de la Asociación Española Contra el Cáncer, INVESTIGADOR AECC 2022
BUSCHBECK, MARCUS
Reference: INVES223200DIES
Title: **Un enfoque funcional para acelerar el desarrollo de terapias farmacológicas combinatorias**
en cánceres de la sangre
Start Date: 01/11/2022 - **End Date:** 31/10/2025
Granted amount: 150.000,00€

Type: Project
2021 Ministerio de Ciencia e Innovación, PROYECTOS DE GENERACIÓN DE CONOCIMIENTO 2021
BUSCHBECK, MARCUS
Reference: PID2021-126907NB-I00
Title: **Regulación de potenciadores de la expresión génica y detección de metabolitos por parte de variantes de histonas**
Start Date: 01/09/2022 - **End Date:** 31/08/2025
Granted amount: 338.800,00€

FACTS & FIGURES

COMPETITIVE GRANTS AWARDED AND ACTIVE PROJECTS

Type: Project
2022 Fundación Científica de la Asociación Española Contra el Cáncer, PROYECTOS GENERALES AECC 2022
BUSCHBECK, MARCUS
Reference: PRYGN222668BUSC
Title: **Re-educación epigenética del estroma en el microambiente de la médula ósea como enfoque terapéutico en la prevención de cáncer de sangre (EPISTROMA)**
Start Date: 01/12/2022 - **End Date:** 30/11/2025
Granted amount: 300.000,00€

Type: HR
2021 European Commission, MSCA COFUND 2021
BUSCHBECK, MARCUS
Reference: 101081347
Title: **Carreras Postdoc Program Empowering Future Leaders to Fight Blood Cancers**
Start Date: 01/01/2023 - **End Date:** 31/12/2027
Granted amount: 2.292.480,00€

Type: HR
2021 European Molecular Biology Organization, EMBO POSTDOCTORAL FELLOWSHIPS 2021 (Spring evaluation)
BUSCHBECK, MARCUS
Reference: ALTF 81-2022
Title: **Elucidating the role of the histone variant macroH2A1.2 as a metabolic sensor in cell fate**
Start Date: 01/08/2022 - **End Date:** 31/07/2024
Granted amount: 108.000,00€

3D Chromatin Organization led by Biola M. Javierre

Type: Project
2021 Ministerio de Ciencia e Innovación, PROYECTOS DE GENERACIÓN DE CONOCIMIENTO 2021
JAVIERRE MARTINEZ, BIOLA M
Reference: PID2021-125277OB-I00
Title: **Descifrando el papel y la regulación de la arquitectura del genoma espacio-temporal en la linfomagénesis de células B**
Start Date: 01/09/2022 - **End Date:** 31/08/2025
Granted amount: 302.500,00€

Type: Mobility
2022 European Molecular Biology Organization, SCIENTIFIC EXCHANGE GRANTS
FANLO ESCUDERO, LUCIA
Title: **Dynamic 3D Chromatin Organization in Human B-cell lymphopoiesis: description of novel genes associated with B-cell acute lymphoblastic leukaemia**
Start Date: 08/11/2022 - **End Date:** 14/03/2023
Granted amount: 6.350,00€

Epigenetics and Immune Disease led by Esteban Ballestar

Type: Project
2021 Fundació ""La Caixa"", CAIXARESEARCH HEALTH 2022
BALLESTAR TARIN, ESTEBAN
Reference: HR22-00668
Title: **Uncovering the Differentiation Determinants and Dynamics of Congenital Susceptibility to Infections**
Start Date: 01/09/2022 - **End Date:** 31/08/2025
Granted amount: 477.722,19€

Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS CONTRATOS PREDOCTORALES PARA FORMACIÓN DOCTORES (FPI)
BALLESTAR TARIN, ESTEBAN
Reference: PRE2021-098003
Title: **Entendiendo el papel de la comunicacion celular en el sistema inmune en la desregulacion epigenetica en inflamacion**
Start Date: 01/09/2022 - **End Date:** 30/08/2025
Granted amount: 100.860,00€

Lymphocyte Development and Disease led by Maribel Parra

Type: Project
2021 Deutsche José Carreras Leukämie Stiftung, DJCLS PROJECT CALL 2021
PARRA BOLA, MARIA ISABEL
Reference: DJCLS 07 R/2022
Title: **Precision medicine in infant acute lymphoblastic leukemia: Modulating specific histone deacetylases to improve prognosis**
Start Date: 01/10/2022 - **End Date:** 30/09/2025
Granted amount: 134.100,00€

Regulatory Genomics led by Tanya Vavouri

Type: HR
2022 Fundació "la Caixa", BECAS DE DOCTORADO INPHINIT INCOMING 2022
VAVOURI, TANYA SOULTANA
Reference: 120917
Title: **The effect of transposable elements on gene regulation in mammals**
Start Date: 01/11/2022 - **End Date:** 31/10/2025
Granted amount: 122.592,00€

Regulatory RNA and Chromatin led by Sònia Guil

Type: Project
2022 International Rett Syndrome, RETT SYNDROME INNOVATION AWARD 2022
GUIL DOMÈNECH, SÒNIA
Reference: 996834
Title: **Study of MeCP2 RNA targets involved in autophagy. Leveraging the RNA binding activity of MECP2 to improve gene replacement therapy for Rett syndrome**
Start Date: 15/12/2022 - **End Date:** 14/12/2024
Granted amount: 195.610,14€

Transcriptional Dynamics in Leukemia led by Sergi Cuartero

Type: Project
2021 American Society of Hematology, ASH GLOBAL RESEARCH AWARD
CUARTERO BETRIU, SERGI
Title: **Understanding the role of 3D genome organization in myeloid leukemia of Down Syndrome**
Start Date: 01/07/2022 - **End Date:** 30/06/2025
Granted amount: 142.626,00€

Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS CONTRATOS PREDOCTORALES PARA FORMACIÓN DOCTORES (FPI) - **CUARTERO BETRIU, SERGI**

FACTS & FIGURES

COMPETITIVE GRANTS AWARDED AND ACTIVE PROJECTS

Reference: PRE2021-097862
Title: **DESCIFRANDO EL ROL DE LAS MUTACIONES EN EL COMPLEJO DE LAS COHESINAS Y LA ESTRUCTURA 3D DEL GENOMA EN LEUCEMIA MIELOIDE**
Start Date: 01/08/2022 - **End Date:** 31/07/2026
Granted amount: 100.860,00€

Cancer Immunogenomics led by Eduard Porta

Type: HR
2021 Fundació ""La Caixa"", BECAS DE DOCTORADO INPHINIT RETAINING 2022
PORTA PARDO, EDUARD
Reference: 118772
Start Date: 16/10/2022 - **End Date:** 15/10/2025
Granted amount: 122.592,00€

Type: HR
2021 Fundació ""La Caixa"", BECAS DE DOCTORADO INPHINIT RETAINING 2022
PORTA PARDO, EDUARD
Reference: 122913
Start Date: 16/10/2022 - **End Date:** 15/10/2025
Granted amount: 122.592,00€

Type: Project
2021 European Commission, A COMPETITIVE HEALTH-RELATED INDUSTRY 2022
PORTA PARDO, EDUARD
Reference: 101095717
Title: **Scaling Up secure Processing, Anonymization and generation of Health Data for EU cross border collaborative research and Innovation**
Start Date: 01/01/2023 - **End Date:** 31/12/2025
Granted amount: 230.000,00€

Type: Project
2022 Asociación Española de Investigación sobre el Cáncer, III AYUDA DE INVESTIGACIÓN EN CÁNCER FERO-ASEICA
PORTA PARDO, EDUARD
Title: **Mapping the activity of Cancer Hallmarks to predict the success of cancer treatments**
Start Date: 01/01/2023 - **End Date:** 31/12/2024
Granted amount: 80.000,00€

Cancer Heterogeneity and Hierarchies led by Verónica Rodilla

Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS JUAN DE LA CIERVA FORMACIÓN
RODILLA BENITO, VERÓNICA
Reference: FJC2021-047741-I
Title: **Heterogeneidad tumoral y plasticidad celular del cáncer de mama**
Start Date: 01/01/2023 - **End Date:** 31/12/2024
Granted amount: 64.800,00€

Leukemia and Immuno-Oncology led by Laura Belver

Type: HR
2022 Agència de Gestió d'Ajuts Universitaris i de Recerca, Ajuts per a la contractació de personal investigador novell (FI-2022)
BELVER MIGUEL, LAURA
Reference: 2022 FI_B 00595
Title: **Analysis of the functional impact of non-coding mutations in Juvenile Myelomonocytic Leukemia**
Start Date: 01/04/2022 - **End Date:** 31/03/2025
Granted amount: 69.169,42€

Stem Cells and Cancer led by Anna Bigas

Type: HR
2021 European Commission, MSCA POSTDOCTORAL FELLOWSHIPS 2021
BIGAS SALVANS, ANNA
Reference: 101068212
Title: **Identification and characterization of long non-coding RNAs as drivers of stemness in hematopoietic stemcells and leukemia.**
Start Date: 01/01/2023 - **End Date:** 31/12/2024
Granted amount: 165.312,96€

Type: Project
2021 Deutsche José Carreras Leukämie Stiftung, DJCLS PROJECT CALL 2021
BIGAS SALVANS, ANNA
Reference: DJCLS 14 R/2022
Title: **Establishment of preclinical models of the juvenile myelomonocytic leukemia to develop new therapeutic approaches for high risk patients**
Start Date: 01/10/2022 -**End Date:** 30/09/2025
Granted amount: 84.546,00€

Endothelial Pathobiology and Microenvironment led by Mariona Graupera

Type: Project
2021 Fundació ""La Caixa"", CAIXARESEARCH HEALTH 2022
GRAUPERA GARCIA - MILA, MARIONA
Reference: HR22-00316
Title: **Understanding and promoting the growth and regenerative functions of blood vessels in heart disease**
Start Date: 01/12/2022 - **End Date:** 30/11/2025
Granted amount: 290.025,54€

Type: HR
2022 Institució Catalana De Recerca i Estudis Avançats, ICREA SENIOR CALL 2022
GRAUPERA GARCIA - MILA, MARIONA
Title: **Icrea Senior Call 2022**

Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS CONTRATOS PREDOCTORALES PARA FORMACIÓN DOCTORES (FPI)
GRAUPERA GARCIA - MILA, MARIONA
Reference: PRE2021-099260
Title: **LAS VARIANTES DE PIK3CA IN PROS: DESCIFRANDO EL CODIGO DE PATOGENESIS**
Start Date: 01/09/2022 - **End Date:** 30/08/2025
Granted amount: 100.860,00€

T-Cell Lymphoma led by Laura Mondragón

Type: Project
2022 Leukemia Research Foundation, Hollis Brownstein Research Grants Program - New Investigator Blood Cancer Research Grant Program (Leukemia, Lymphoma, Myeloma, MDS)
MONDRAGÓN MARTÍNEZ, LAURA
Reference:

FACTS & FIGURES

COMPETITIVE GRANTS AWARDED AND ACTIVE PROJECTS

Title: **Nova diana terapèutica (TCRalpha) en AITL (Angioimmunoblastic T-cell lymphoma)**
Start Date: 01/10/2022 - **End Date:** 30/09/2023
Granted amount: 101.220,00€

Acute Lymphoblastic Leukemia (ALL) led by Josep Mª Ribera

Type: Project
2021 Deutsche José Carreras Leukämie Stiftung, DJCLS PROJECT CALL 2021
GENESCA FERRER, EULALIA
Reference: DJCLS 08 R/2022
Title: **Development of innovative therapy strategies to overcome therapy resistance in the Primary therapy for adult T-cell acute lymphatic leukemia (T-ALL)**.
Start Date: 01/11/2022 - **End Date:** 31/10/2025
Granted amount: 155.745,64€

Type: Project
2021 Deutsche José Carreras Leukämie Stiftung, DJCLS PROJECT CALL 2021
GENESCA FERRER, EULALIA
Reference: DJCLS 08 R/2022
Title: **Development of innovative therapy strategies to overcome therapy resistance in the Primary therapy for adult T-cell acute lymphatic leukemia (T-ALL).**
Start Date: 01/11/2022 - **End Date:** 31/10/2025
Granted amount: 155.745,64€

Barcelona Endothelium Team (BET) led by Enric Carreras

Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS JUAN DE LA CIERVA FORMACIÓN
CARRERAS PONS, ENRIC
Reference: FJC2021-048123-I
Title: **Deepening in the pathophysiology of the endothelial damage in various pathologies**
Title: 01/01/2023 - **End Date:** 31/12/2024
Granted amount: 64.800,00€

Myeloid Neoplasms led by Lurdes Zamora and Blanca Xicoy

Type: HR
2022 Lady Tata Memorial Trust, INTERNATIONAL AWARDS 2022
ZAMORA PLANA, LURDES
Reference: 3436
Title: **Dissection of clonal evolution and diversification in secondary and therapy-related acute myeloid leukaemias**
Start Date: 01/10/2022 - **End Date:** 30/09/2023
Granted amount: 41.231,05€

Myelodysplastic Syndromes led by Francesc Solé

Type: HR
2022 Fundación Española de Hematología y Hemoterapia, BECAS DE INVESTIGACIÓN FEHH
ACHA GONZÁLEZ, PAMELA
Reference:

Title: **Monitorización de la carga mutacional en pacientes con síndrome mielodisplásico de bajo riesgo en muestras de sangre periférica secuenciales**
Start Date: 01/01/2023 - **End Date:** 31/12/2024
Granted amount: 78.000,00€

Stem Cell Biology, Developmental Leukemia and Immunotherapy led by Pablo Menéndez

Type: Project
2021 European Commission, TOOLS AND TECHNOLOGIES FOR A HEALTHY SOCIETY 2021
MENÉNDEZ BUJÁN, PABLO
Reference: 101057250
Title: **RNA PROCESSING FOR ANTI-CANCER IMMUNOTHERAPY**
Start Date: 01/06/2022 - **End Date:** 31/05/2025
Granted amount: 350.500,00€

Type: HR
2021 European Commission, MSCA POSTDOCTORAL FELLOWSHIPS 2021
MENÉNDEZ BUJÁN, PABLO
Reference: 101068558
Title: **Contribution of Lipid Droplets to the pathogenesis and chemoresistance of Acute Myeloid Leukemia**
Start Date: 01/09/2023 - **End Date:** 31/08/2025
Granted amount: 226.441,20€

Type: Project
2022 Fundación Merck Salud, AYUDAS MERCK DE INVESTIGACIÓN 2022
SÁNCHEZ MARTÍNEZ, DIEGO
Reference:
Title: **Desarrollo de una innovadora inmunoterapia adoptiva de células CAR-T para sacorma de Ewing**
Start Date: 10/07/2022 - **End Date:** 15/06/2025
Granted amount: 30.000,00€

Type: Project
2021 European Science Foundation, FIGHT KIDS CANCER 2021-2
MENÉNDEZ BUJÁN, PABLO
Reference: 20
Title: **Finding a cure for MLL-rearranged infant acute lymphoblastic leukemia**
Start Date: 01/01/2023 - **End Date:** 31/12/2025
Granted amount: 60.000,00€

Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS JUAN DE LA CIERVA FORMACIÓN
MENÉNDEZ BUJÁN, PABLO
Reference: FJC2021-046789-I
Title: **Next generation T-cell redirected immunotherapy for acute lymphocytic leukemia**
Start Date: 01/01/2023 - **End Date:**31/12/2024
Granted amount: 64.800,00€

Type: HR
2022 Lady Tata Memorial Trust, INTERNATIONAL AWARDS 2022
MENÉNDEZ BUJÁN, PABLO
Reference: 3465
Title: **Contribution of Lipid Droplets to the pathogenesis and chemoresistance of Acute Myeloid Leukeamia**
Start Date: 01/10/2022 - **End Date:** 30/09/2023
Granted amount: 41.231,05€

Type: Project
2022 Fundación Uno Entre Cien Mil , IX BECA PARA LA INVESTIGACIÓN EN EL ÁREA DE LA LEUCEMIA INFANTIL 2022
BUENO UROZ, CLARA
Title: **Novel and innovative therapeutic strategies for patients with childhood B acute lympho-**

COMPETITIVE GRANTS AWARDED AND ACTIVE PROJECTS

blastic leukemia harboring MLL rearrangements
Start Date:: 26/07/2022 - **End Date:** 25/07/2024
Granted amount: 100.000,00€

Type: Project
2021 European Commission, ERC PROOF OF CONCEPT GRANT 2022-1
MENENDEZ BUJAN, PABLO
Reference: 101100665
Title: **Byspecific CAR T-cells for the treatment of CD22/CD19 positive cancer**
Start Date: 01/07/2023 - **End Date:** 31/12/2024
Granted amount: 150.000,00€

Type: Project
2022 Ministerio de Ciencia e Innovación, PROYECTOS DE I+D+i EN LÍNEAS ESTRATÉGICAS, EN COLABORACIÓN PÚBLICO-PRIVADA 2022
MENÉNDEZ BUJÁN, PABLO
Reference: PLEC2022-009416
Title: **Tailored adoptive CAR T-cell Immunotherapy for Ewing Sarcoma**
Start Date: 01/11/2022 - **End Date:** 31/10/2025
Granted amount: 389.715,00€
Type: HR
2022 Agència de Gestió d'Ajuts Universitaris i de Recerca, AJUTS A DOCTORATS INDUSTRIALS
MENÉNDEZ BUJÁN, PABLO
Reference: 2022 DI 43
Title: **Development of new CAR-T treatments for glioblastoma multiforme**
Start Date: 01/10/2022 - **End Date:** 30/09/2025
Granted amount: 33.960,00€

Lymphoma Translational led by Gaël Roué

Type: Project
2021 Ministerio de Ciencia e Innovación, PROYECTOS DE GENERACIÓN DE CONOCIMIENTO 2021
ROUÉ, GAËL
Reference: PID2021-123039OB-C21
Title: **Generación de una colección de esferoides organotípicos 3D y de modelos PDX de LDCG completamente anotados para la evaluación preclínica de disruptores del link tumor-estroma**
Start Date: 01/09/2022 - **End Date:** 31/08/2025
Granted amount: 108.900,00€

Hematological Diseases, Transplant and Cell Therapy led by Jordi Sierra

Type: HR
2022 Fundación Científica de la Asociación Española Contra el Cáncer, INVESTIGADOR AECC 2022
SIERRA SIERRA, JORGE
Reference: INVES223069VELA
Title: **Identificación de nuevas dianas terapéuticas para evitar la recaída y quimioresistencia en Leucemia Mieloide Aguda.**
Start Date: 01/12/2022 - **End Date:** 30/11/2025
Granted amount: 150.000,00€

Proteomics Unit led by Carolina de la Torre

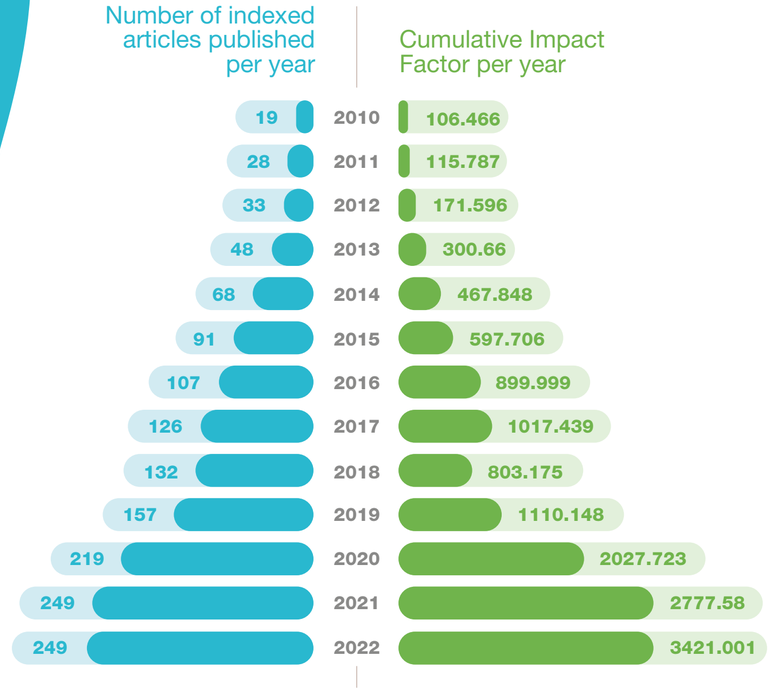
Type: HR
2021 Ministerio de Ciencia e Innovación, AYUDAS PARA PERSONAL TÉCNICO DE APOYO 2021
DE LA TORRE GÓMEZ, CAROLINA
Reference: PTA2021-020842-I
Title: **Técnico de apoyo a la plataforma de proteómica para dar soporte cientifico-tecnico a proyectos de investigación para la comunidad científica general**
Start Date: 01/01/2023 - **End Date:** 31/12/2025
Granted amount: 42.600,00€

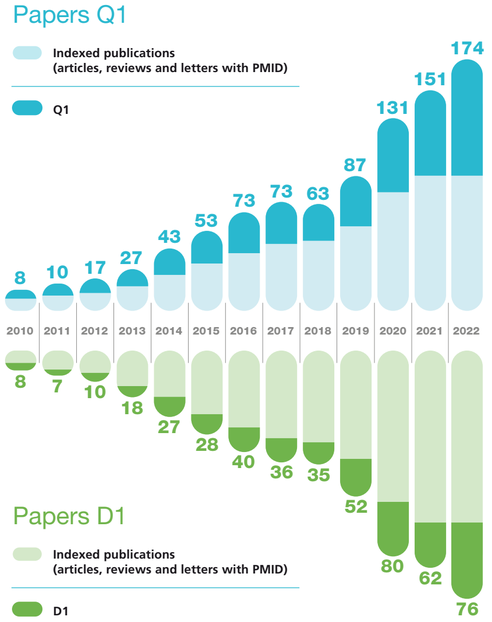
Single Cell Unit led by Caterina Mata

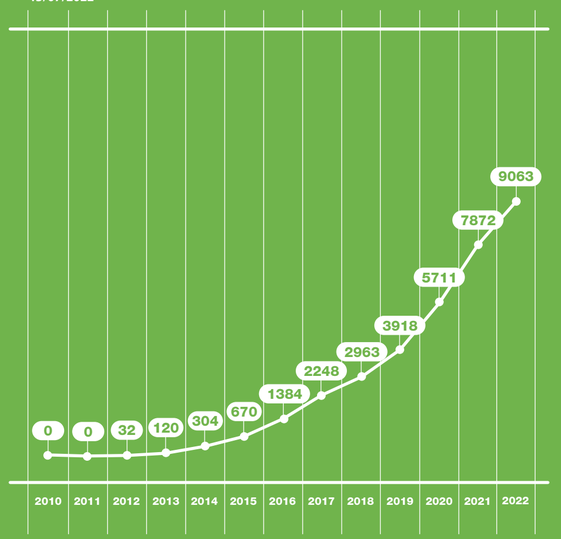
Type: Project
2022 Fundació la Marató de TV3, MARATÓ TV3: SALUT MENTAL
MATA GARCIA, CATERINA
Reference: 202235-31
Title: **Brain and blood coexpression networks using DDR1 as a seed gene in bipolar disorder. Identification of new biomarkers.**
Start Date: planned for 2023 - **End Date:** 3 years

PUBLICATIONS

INDICATORS







2022 PUBLICATIONS

Cancer Epigenetics led by Manel Esteller

Arribas AJ, Napoli S, Cascione L, Sartori G, Barnabei L, Gaudio E, Tarantelli C, Mensah AA, Spriano F, Zucchetto A, Rossi FM, Rinaldi A, **De Moura MC**, Jovic S, Bordone-Pittau R, Di Veroli A, Stathis A, Cruciani G, Stussi G, Gattei V, Brown JR, **Esteller M**, Zucca E, Rossi D, Bertoni F.
Resistance to PI3kδ inhibitors in marginal zone lymphoma can be reverted by targeting the IL-6/PDGFRA axis
Haematologica. 2022 Nov 1;107(11):2685-2697. doi: 10.3324/haematol.2021.279957.
Impact Factor: 11,047 - Q1
PMID: 35484662
Citations: 0 [Web of Science -23/02/2023]

Berdasco M, Esteller M.
Towards a druggable epitranscriptome: Compounds that target RNA modifications in cancer Br J Pharmacol. 2022 Jun;179(12):2868-2889. doi: 10.1111/bph.15604. Epub 2021 Jul 27.
Impact Factor: 9,473 - Q1
PMID: 34185879
Citations: 8 [Web of Science -23/02/2023]

Blecua P, Davalos V, de Villasante I, Merkel A, Musulen E, Coll-SanMartin L, Esteller M.
Refinement of computational identification of somatic copy number alterations using DNA methylation microarrays illustrated in cancers of unknown primary
Brief Bioinform. 2022 Sep 20;23(5):bbac161. doi: 10.1093/bib/bbac161
Impact Factor: 13,994 - Q1
PMID: 35524475
Citations: 0 [Web of Science -23/02/2023]

Bueno-Costa, A; Pineyro, D; Garcia-Prieto, CA; Ortiz-Barahona, V; Martinez-Verbo, L; Webster, NA; Andrews, B; Kol, N; Avrahami, C; Moshitch-Moshkovitz, S; Rechavi, G; **Esteller, M**
Remodeling of the m(6)A RNA landscape in the conversion of acute lymphoblastic leukemia cells to macrophages Leukemia. 2022 Aug;36(8):2121-2124. doi: 10.1038/s41375-022-01621-1
Impact Factor: 12,883 - Q1
PMID: 35681051
Citations: 1 [Web of Science -23/02/2023]

Cao X, Li W, Wang T, Ran D, **Davalos V**, Planas-Serra L, Pujol A, **Esteller M**, Wang X, Yu H.Accelerated biological aging in COVID-19 patients
Nat Commun. 2022 Apr 19;13(1):2135. doi: 10.1038/s41467-022-29801-8.
Impact Factor: 17,694 - Q1
PMID: 35440567
Citations: 20 [Web of Science -23/02/2023]

Carrier A, Desjobert C, Lobjois V, Rigal L, Busato F, Tost J, Ensenyat-Mendez M, Marzese DM, Pradines A, Favre G, Lamant L, Lanfrancone L, Etievant C, Arimondo PB, Riond J.
Epigenetically regulated PCDHB15 impairs aggressiveness of metastatic melanoma cellsClin Epigenetics. 2022 Nov 28;14(1):156. doi: 10.1186/s13148-022-01364-x.
Impact Factor: 7,259 - Q1
PMID: 36443814
Citations: 0 [Web of Science -23/02/2023]

Carrier A, Desjobert C, Ponger L, Lamant L, Bustos M, Torres-Ferreira J, Henrique R, Jeronimo C, Lanfrancone L, Delmas A, Favre G, Daunay A, Busato F, Hoon DSB, Tost J, Etievant C, Riond J, Arimondo PB
DNA methylome combined with chromosome cluster-oriented analysis provides an early signature for cutaneous melanoma aggressiveness
Elife. 2022 Sep 20;11:e78587. doi: 10.7554/eLife.78587.
Impact Factor: 8,7132 - Q1
PMID: 36125262
Citations: 1 [Web of Science -23/02/2023]

Casado-Pelaez M, Bueno-Costa A, Esteller M.
Single cell cancer epigenetics
Trends Cancer. 2022 Oct;8(10):820-838. doi: 10.1016/j.trecan.2022.06.005. Epub 2022 Jul 9.
Impact Factor: 19,161 - Q1
PMID: 35821003
Citations: 3 [Web of Science -23/02/2023]

Cristalli, C; Manara, MC; Valente, S; Pellegrini, E; Bavelloni, A; De Feo, A; Blalock, W; Di Bello, E; **Pineyro, D; Merkel, A; Esteller, M**; Tirado, OM; Mai, ATEL; Scotlandi, K
Novel Targeting of DNA Methyltransferase Activity Inhibits Ewing Sarcoma Cell Proliferation and Enhances Tumor Cell Sensitivity to DNA Damaging Drugs by Activating the DNA Damage Response
Front Endocrinol (Lausanne). 2022 May 31;13:876602. doi: 10.3389/fendo.2022.876602. eCollection 2022.
Impact Factor: 6,055 - Q1
PMID: 35712255
Citations: 2 [Web of Science -23/02/2023]

Cullell N, Soriano-Tárraga C, Gallego-Fábrega C, Cárcel-Márquez J, Torres-Águila NP, Muiño E, Lledós M, Llucià-Carol L, **Esteller M, Castro de Moura M**, Montaner J, Fernández-Sanlés A, Elosua R, Delgado P, Martí-Fàbregas J, Krupinski J, Roquer J, Jiménez-Conde J, Fernández-Cadenas I.
DNA Methylation and Ischemic Stroke Risk: An Epigenome-Wide Association Study
Thromb Haemost. 2022 Oct;122(10):1767-1778. doi: 10.1055/s-0042-1749328
Impact Factor: 6,681 - Q1

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Citations: 0 [Web of Science -23/02/2023]

Davalos V, **García-Prieto CA, Ferrer G**, Aguilera-Albesa S, Valencia-Ramos J, Rodríguez-Palmero A, Ruiz M, Planas-Serra L, Jordan I, Alegría Í, Flores-Pérez P, Cantarín V, Fumadó V, Viadero MT, Rodrigo C, Méndez-Hernández M, López-Granados E, Colobran R, Rivière JG, Soler-Palacín P, Pujol A, **Esteller M.**
Epigenetic profiling linked to multisystem inflammatory syndrome in children (MIS-C): A multicenter, retrospective study
EClinicalMedicine. 2022 Jun 25;50:101515. doi: 10.1016/j.eclinm.2022.101515. eCollection 2022 Aug.
Impact Factor: 17,033 - Q1
PMID: 35770252
Citations: 0 [Web of Science -23/02/2023]

Davalos V; Esteller M
Cancer epigenetics in clinical practice
CA Cancer J Clin. 2022 Dec 13. doi: 10.3322/caac.21765. Online ahead of print.
Impact Factor: 286,13 - Q1
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Citations: 0 [Web of Science -23/02/2023]

Fernández-Figueras MT; Carrato C; Saenz-Sardà X; **Musulén E**; Fuente MJ; Puig L.
MicroRNA31 and MMP-1 contribute to the differentiated pathway of invasion -with enhanced epithelial-to-mesenchymal transition- in squamous cell carcinoma of the skin
Arch Dermatol Res. 2022 Oct;314(8):767-775
Impact Factor: 3,033 - Q2
PMID: 34647185
Citations: 3 [Web of Science -23/02/2023]

Ferrer G, Álvarez-Errico D, Esteller M.
Biological and Molecular Factors Predicting Response to Adoptive Cell Therapies in Cancer
J Natl Cancer Inst. 2022 Jul 11;114(7):930-939. doi: 10.1093/jnci/djac088.
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Garcia-Prieto CA, Álvarez-Errico D, Musulen E, Bueno-Costa A, N Vazquez B, Vaquero A, Esteller M.
Validation of a DNA methylation microarray for 285,000 CpG sites in the mouse genomeEpigenetics. 2022 Dec;17(12):1677-1685. doi: 10.1080/15592294.2022.2053816
Impact Factor: 4,861 - Q1
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Citations: 4 [Web of Science -23/02/2023]

Garcia-Prieto CA, Martínez-Jiménez F, **Valencia A, Porta-Pardo E.**
Detection of oncogenic and clinically actionable mutations in cancer genomes critically depends on variant calling tools
Bioinformatics. 2022 Jun 13;38(12):3181-3191. doi: 10.1093/bioinformatics/btac306
Impact Factor: 6,931 - Q1
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Garcia-Prieto CA; Villanueva L; Bueno-Costa A; Davalos V; González-Navarro EA; Juan M; **Urbano-Ispizua Á**;Delgado J; Ortíz-Maldonado V; Del Bufalo F; Locatelli F; Quintarelli C; Sinibaldi M; **Soler M; Castro de Moura M**; **Ferrer G**; Urdinguio RG; Fernandez AF; Fraga MF; Bar D; Meir A; Itzhaki O; Besser MJ; Avigdor A; Jacoby E; **Esteller M.**
Epigenetic Profiling and Response to CD19 Chimeric Antigen Receptor T-Cell Therapy in B-Cell Malignancies
J Natl Cancer Inst. 2022 Mar 8;114(3):436-445. doi: 10.1093/jnci/djab194.
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DNA methylome in visceral adipose tissue can discriminate patients with and without colorectal cancer
Epigenetics. 2022 Jun;17(6):665-676
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Jan Binkowski, Olga Taryma-Lésniak, Karolina Łuczkowska, Anna Niedzwied´z, Kacper Lechowicz, Dominik Strapagiel, Justyna Jarczak, Veronica Davalos, Aurora Pujol, **Manel Esteller**, Katarzyna Kotfis, Bogusław Machalinski, Miłosz Parczewsk, Tomasz K. Wojdacz
Epigenetic activation of antiviral sensors and effectors of interferon response pathways during SARS-CoV-2 infection Biomed.
Pharmacother. 2022 Sep;153:113396. doi: 10.1016/j.biopha.2022.113396. Epub 2022 Jul 11.
Impact Factor: 7,419 - Q1
PMID: 36076479
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Ji X, Lin L, Fan J, Li Y, Wei Y, Shen S, Su L, Shafer A, Bjaanaes MM, Karlsson A, Planck M, Staaf J, Helland Å, **Esteller M**, Zhang R, Chen F, Christiani DC.
Epigenome-wide three-way interaction study identifies a complex pattern between TRIM27, KIAA0226, and smoking associated with overall survival of early-stage NSCLC
Mol Oncol. 2022 Feb;16(3):717-731. doi: 10.1002/1878-0261.13167. Epub 2022 Jan 7.
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Joshi RS, Rigau M, García-Prieto CA, **Castro de Moura M, Piñeyro D, Moran S, Davalos V**, Carrión P, Ferrando- Bernal M, Olalde I, Lalueza-Fox C, Navarro A, Fernández-Tena C, Aspandi D, Sukno FM, Binefa X, Valencia A, **Esteller M**.
Look-alike humans identified by facial recognition algorithms show genetic similarities
Cell Rep. 2022 Aug 23;40(8):111257. doi: 10.1016/j.celrep.2022.111257.
Impact Factor: 9,995 - Q1
PMID: 36001980
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Cancers (Basel). 2022 Jan 11;14(2):349. doi: 10.3390/cancers14020349.
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Oldoni E, Saunders G, Bietrix F, Garcia Bermejo ML, Niehues A, 't Hoen PAC, Nordlund J, Hajduch M, Scherer A, Kivinen K, Pitkänen E, Mäkelä TP, Gut I, Scollen S, Kozera Ł, **Esteller M**, Shi L, Ussi A, Andreu AL, van Gool AJ.
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Front Mol Biosci. 2022 Oct 13;9:974799. doi: 10.3389/fmolb.2022.974799. eCollection 2022.
Impact Factor: 6,113 - Q1
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Citations: 0 [Web of Science -23/02/2023]

Oriol-Tordera B, Esteve-Codina A, **Berdasco M**, Rosás-Umbert M, Gonçalves E, Duran-Castells C, Català-Moll F, Llano A, Cedeño S, Puertas MC, Tolstrup M, Søgaard OS, Clotet B, Martínez-Picado J, Hanke T, Combadiere B, Paredes R, Hartigan-O'Connor D, **Esteller M,** Meulbroek M, Calle ML, Sanchez-Pla A, Moltó J, Mothe B, Brander C, Ruiz-Riol M.
Epigenetic landscape in the kick-and-kill therapeutic vaccine BCN02 clinical trial is associated with antiretroviral treatment interruption (ATI) outcome
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Pignata, L; Cecere, F; Verma, A; Mele, BH; Monticelli, M; Acurzio, B; Giaccari, C; Sparago, A; Mora, JRH; Monteagudo-Sanchez, A; **Esteller, M**; Pereda, A; Tenorio-Castano, J; Palumbo, O; Carella, M; Prontera, P; Piscopo, C; Accadia, M; Lapunzina, P; Cubellis, MV; de Nanclares, GP; Monk, D; Riccio, A; Cerrato, F
Novel genetic variants of KHDC3L and other members of the subcortical maternal complex associated with Beckwith- Wiedemann syndrome or Pseudohypoparathyroidism 1B and multi-locus imprinting disturbances
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Impact Factor: 10,787 - Q1
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Citations: 0 [Web of Science -23/02/2023]

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Genetically predicted telomere length and Alzheimer's disease endophenotypes: a Mendelian randomization study Alzheimers Res Ther. 2022 Nov 7;14(1):167. doi: 10.1186/s13195-022-01101-9.
Impact Factor: 8,823 - Q1
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Citations: 0 [Web of Science -23/02/2023]

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Impact Factor: 7,259 - Q1
PMID: 35810318
Citations: 1 [Web of Science -23/02/2023]

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Citations: 2 [Web of Science -23/02/2023]

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PLoS One. 2022 Jun 3;17(6):e0269501. doi: 10.1371/journal.pone.0269501. eCollection 2022.
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PMID: 35657981
Citations: 0 [Web of Science -23/02/2023]

Cancer Genetics led by Montse Sanchez-Cespedes

Cucurull M, Notario L, **Sanchez-Cespedes M**, Hierro C, Estival A, Carcereny E, Saigí M.
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PMID: 35096591
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Impact Factor: 17,906 - Q1
PMID: 36494864
Citations: 0 [Web of Science -23/02/2023]

Saigí M, Carcereny E, Morán T, Cucurull M, Domènech M, Hernandez A, Martinez-Cardús A, **Pros E, Sanchez-Cespedes M.**
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Impact Factor: 13,608 - Q1
PMID: 35777135
Citations: 0 [Web of Science -23/02/2023]

Chromatin Biology led by Alex Vaquero

Garcia-Prieto CA, Álvarez-Errico D, Musulen E, Bueno-Costa A, N Vazquez B, Vaquero A, Esteller M.
Validation of a DNA methylation microarray for 285,000 CpG sites in the mouse genomeEpigenetics. 2022 Dec;17(12):1677-1685. doi: 10.1080/15592294.2022.2053816
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Chromatin, Metabolism and Cell Fate led by Marcus Buschbeck

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Transcriptional Dynamics in Leukemia led by Sergi Cuartero

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Cellular Systems Genomics led by Elisabetta Mereu

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*The Leukemia Stem Cell Group left the Josep Carreras Institute on October 31, 2022

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Individually we are strong. **Together we are unstoppable!**

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