COFUND

CURE HEART & BRAIN

CNIC Research Lines

CNIC Program: Myocardial homeostasis & cardiac injury

Coordinator: Enrique Lara Pezzi – Clinical leader: Borja Ibáñez

CNIC Group 1: Molecular Regulation of Heart Failure

PI: Enrique Lara-Pezzi

Research Line: New translational approaches in dilated cardiomyopathy and heart failure: from molecular mechanisms to therapy

Description: The group investigates the molecular mechanisms that underlie inherited cardiomyopathies and heart failure by combining expertise in advanced non-invasive imaging, pathophysiology and molecular biology in translational animal models. Dilated cardiomyopathy (DCM) is a heart disease characterized by thinning and stretching of the ventricles, which grow larger, making it harder for the heart to pump blood. The mechanisms leading to dilatation of the left ventricle are not completely understood. Furthermore, current therapies are inefficient, as they follow a 'one-size-fits-all' approach, disregarding the specific pathology of the disease. The research in our group focuses on the identification of cellular and molecular mechanisms driving ventricular dilatation, cardiac dysfunction and heart failure in pig and mouse models of dilated cardiomyopathy. More specifically, the group investigates the contribution of different immune cell populations to DCM, based on recent single-nuclei RNA-seq analyses in our laboratory. Following the identification of potential targets, the group will develop new gene therapy products for the treatment of this disease.

CNIC Group 2: Translational Laboratory for Cardiovascular Imaging and Therapy

PI: Borja Ibáñez

Research Line: Novel metabolic interventions to prevent cancer therapy related cardiovascular toxicities

Description: By combining complementary experimental approaches in small and large animal models, our aim is to identify novel therapies able to prevent/treat cardiac metabolic alterations occurring in patients with cancer treated with cytotoxic agents. Within several tools, cardiac magnetic resonance will be key across the execution of this research.

CNIC Group 3: Molecular Mechanics of the Cardiovascular System

PI: Jorge Alegre-Cebollada

Research Line: Integrated sarcomere mechanics in familial and acquired cardiomyopathy

Description: The group Molecular Mechanics of the Cardiovascular System specializes in the multiscale characterization of myocardial mechanics, from the single-protein to the tissue levels. The research of the group focuses on understanding how the dysfunction of sarcomeres contributes to heart failure including the crosstalk with defective activity of other organelles like the mitochondria or adhesion complexes.

CNIC Group 4: Heart Failure and Pulmonary Hypertension translational research

PI: Ana García

Research Line: Heart failure and pulmonary hypertension translational research

Description: Our laboratory focuses on the study of myocardial diseases leading to heart failure (HF) and pulmonary hypertension (PH) from a translational perspective ranging from molecular studies and experimental models to multicenter clinical trials. We are a multidisciplinary team that includes cardiologists and cardiac surgeons and closely collaborate with experts in molecular biology, proteomics, metabolomics and genetics. We have developed and deeply characterized four different models of PH or RV pressure overload in pigs, induced by different mechanisms. These models have been used for the development of new diagnostic algorithms based on cardiac magnetic resonance, to identify early RV involvement, and to evaluate the mechanisms underlying the beneficial effects of current and novel treatments. Some of these investigations have been carried forward to the clinical arena and evaluated as multicenter clinical trials. In the last years, our efforts are centered on understanding the underlying mechanisms of RV dysfunction in PH through the integration of advanced imaging and omics (proteomics, metabolomics and genomics), from studies in our experimental models and in patients with various types of PH. The research seeks to decipher the mechanisms that lead to RV dysfunction in the presence of PH and various cardiomyopathies with a translational vision that moves from basic research to the clinics and vice versa, all with an open and friendly dialogue between professionals with different scientific background.

CNIC Group 5: Inherited Cardiomyopathies

PI: Pablo García-Pavía

Research Line: Identification of genetic mechanisms and new biomarkers in cardiomyopathies

Description: The group is focused on the study of the genetic mechanisms involved in the development of inherited heart diseases and in the identification of new biomarkers in TTR cardiac amyloidosis. It is intriguing why despite the advances in genetic field, a genetic cause is still only detected in less than 40% of the families with Hypertrophic cardiomyopathy (HCM) and Dilated cardiomyopathy (DCM), the two main types of cardiomyopathies. Therefore, our research is focused on the identification of new genes and new mutations that could explain the cause of the disease in the unsolved group of patients. Furthermore, the group has a strong interest in TTR cardiac amyloidosis, a progressive and often fatal disease that is a frequent cause of heart failure. In this area, the group is conducting several studies to understand how patients with this disease respond to available therapies along with studies to identify new diagnostic and prognostic biomarkers that would improve the diagnosis and the follow-up of these patients.

CNIC Programme: Cardiovascular risk factors & brain function

Coordinator: María Ángeles Moro - Clinical leader: Valentín Fuster

CNIC Group 6: Neurovascular Pathophysiology

PI: María Ángeles Moro

Research Line: Vascular-driven cognitive impairment and dementia

Description: The Neurovascular Pathophysiology Group, led by Dr. María Ángeles Moro, is focused the pathophysiology of cerebrovascular disease (stroke and vascular cognitive impairment), since they are a leading cause of death and disability, with an increasing prevalence due to the ageing of the population, and represent an unmet therapeutic and diagnostic need.

The group made seminal contributions for the identification of several therapeutic and diagnostic targets in acute ischemic stroke (JEM 2006; Circulation 2007, 2008, 2014; Stroke 2012, 2017; Neuroimage 2011, JCBFM 2016, JCBFM 2021), some in the path to reach the patients in the short-term (Mol Ther 2018, 2022; JAMA Neurol 2023). Now, by applying new models of vascular-driven dementia (post-stroke, cerebral hypoperfusion, pre-hypertension, atherosclerosis), the group explores novel mechanisms in chronic cerebrovascular disease, including maladaptive hippocampal neurogenesis (JCI 2019; Stroke 2023; Front Cell Neurosci 2023), neutrophil-related immunothrombosis (Stroke 2013, Science 2014, Stroke 2019a,b, Front Immunol 2022) and its circadian regulation (Immunity 2019, Nat Immunol 2020, Stroke 2021). These research endeavours have been consistently supported by numerous competitive projects funded by national and international agencies (Spanish Research Agency, Fundación La Caixa, Foundation Leducq), and contracts with industrial partners.

Specific lines for this call will also delve into the participation and role of neuroimmune interfaces and perivascular spaces, glymphatic system/meningeal lymphatics, blood-brain barrier and cerebral

vasculature, among others, by using dedicated animal models and multidisciplinary cutting-edge technologies. Preclinical findings will be validated in human samples from ongoing collaborations with clinical groups. In addition to the mechanistical insight provided, this project will serve to deliver a framework to improve prevention, diagnosis, and treatment of VCID. Bioinformatician expertise in multi-omics data analysis with a solid knowledge in neuroimmunology and in the neurovascular complex will be highly valuable.

CNIC Group 7: Cardiovascular Imaging and Population Studies

PI: Valentin Fuster

Research Line: Cardiovascular Imaging and Population Studies

Description: The Cardiovascular Imaging and Population Studies Group, led by Dr. Valentin Fuster, has developed research applications for non-invasive, high-resolution and high-sensitivity imaging technologies to support translational research and population studies in preclinical atherosclerosis.

Among their main studies, the group is performing a longitudinal study to evaluate their trajectories very early in their courses (i.e. in asymptomatic stages) in order to understand the interrelationship between brain and cardiovascular disease at the preclinical level. The Progression of Early Subclinical Atherosclerosis (PESA), led by Dr. Valentin Fuster, is a prospective ongoing study that included over 4,000 middle-aged asymptomatic participants back in 2010 with the aim of tracking the trajectories of atherosclerosis and associated disorders from early stages to symptomatic phases. The candidate will join the PESA-Brain sub-study, led by Dr. Marta Cortes-Canteli, in which 1,000 PESA participants are currently undergoing a thorough neurocognitive testing together with amyloid-PET and a highly comprehensive brain magnetic resonance imaging protocol to study brain morphology, connectivity and perfusion, vascular lesions and intracranial atherosclerosis.

CNIC Group 8: Multidisciplinary Translational Cardiovascular Research

PI: Héctor Bueno

Research Line: Multidisciplinary Translational Cardiovascular Research (MTCR)

Description: The Multidisciplinary Translational Research (MTCR) Group,led by Dr. Héctor Bueno, is a platform for innovative knowledge generation in cardiovascular health and disease. The mission of the MTCR Group is to promote the generation of disruptive knowledge and innovation with a humanitarian perspective to foster, prevent or improve the health or quality of life of citizens and patients, increasing the efficiency/safety of the healthcare system. The research lines offered by the MTCR group include

- Mental health and cardiovascular risk
- Social determinants of cardiovascular health and disease

- Cardiovascular epidemiology, quality of care and outcomes
- Impact of gender on cardiovascular health and disease
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CNIC Group 9: Regulatory Molecules of Inflammatory Processes

PI: Pilar Martin

Research Line: Regulatory Molecules of Inflammatory Processes

Description: The Regulatory Molecules of Inflammatory Processes group, led by Dr. Pilar Martín, seeks to study the therapeutic and diagnostic potential of T cells, their immunomodulatory receptors and microRNAs, in the management of cardiovascular disease (CVD) and in the development of precision medicine tools. T lymphocytes are pivotal in the development of CVD and, together with certain microRNAs have been shown to be altered in blood and cardiovascular tissues during their progression. In this context, the research of the group encompasses a holistic approach to understand the full spectrum of cognitive impairments, whether they stem from neurodegenerative diseases, immune-related factors, or the natural aging process. We also explore the intricate intersections between immune system disturbances induced by cardiovascular risk factors, such as diet or vascular remodeling, and cognitive dysfunction. The candidate will join studies aimed to assess cognitive impairments through behavioral tests, complemented by state-of-the-art molecular biology techniques, including single-cell RNA sequencing and advanced imaging modalities like magnetic resonance imaging (MRI) and PET-CT scans. These cutting-edge methods will enable us to track and analyze changes in both animal models and human subjects, providing valuable insights into the complex relationship between neuroinflammation and cognitive deficits.

CNIC Program: Novel mechanisms of atherosclerosis

Coordinator: José Javier Fuster – Clinical leaders: Valentín Fuster and Inés García Lunar

CNIC Group 10: Hematovascular Pathophysiology

PI: José Javier Fuster

Research Line: Pathophysiological effects and dynamics of age-related clonal hematopoiesis

Description: Clonal hematopoiesis has recently emerged as a novel risk factor for atherosclerotic cardiovascular disease (CVD), with significant implications for personalized medicine. This condition is typically driven by the acquisition of certain somatic mutations in the hematopoietic system, which lead to the clonal expansion of the mutant cell and the subsequent propagation of the mutation throughout the immune system. Our prior human and mouse studies support the direct contribution of some of these mutations to atherosclerotic CVD through the exacerbation of specific inflammatory responses (e.g. Science 2017, JACC 2021, Nature CVR 2023). In this context, one of our current objectives is to

understand the factors that modulate the dynamics of clonal hematopoiesis and its impact on atherosclerosis. To achieve this, we will employ both bulk and single-cell omics analysis on deeply characterized human cohorts and conduct innovative experiments using mice and cultured cells. This interdisciplinary research may be of interest to investigators across multiple fields, including genomics, bioinformatics, experimental hematology, immunology, and cardiovascular pathophysiology.

More information:

https://scholar.google.com/citations?user=9c-_E-YAAAAJ&hl=en ; https://atheroclonal.com/

CNIC Groups 11: Cardiovascular prevention through non-invasive imaging and Hematovascular Pathophysiology

PI: Inés García Lunar and José Javier Fuster

Research Line: New inflammatory drivers of human atherosclerosis

Description: Despite the efficacy of interventions targeting traditional cardiovascular risk factors like cholesterol-lowering drugs, atherosclerotic cardiovascular disease (CVD) remains the leading global cause of death. In this context, there is a gaping hole in our approach to CVD prevention. It is widely recognized that atherosclerosis, the primary cause of most CVD events, results from a maladaptive inflammatory response to the chronic exposure to various CVD risk factors. Yet, targeting inflammation for CVD prevention remains an unfulfilled promise due to the scarce understanding of the specific inflammatory factors that drive human atherosclerosis development across the disease continuum. In this context, this collaborative research is aimed at identifying the inflammatory drivers of the development of atherosclerosis during its preclinical stages. At the core of this endeavor are several human cohorts that are tracking subclinical atherosclerosis development in apparently healthy individuals through longitudinal non-invasive imaging. Using these unique resources and advanced omics analyses, we seek to identify the specific immunomodulatory mechanisms that, when dysregulated, drive human atherosclerosis development.

CNIC Group 12: Molecular and Genetic Cardiovascular Pathophysiology

PI: Vicente Andrés

Research Line: A-type lamin-dependent control of atherosclerosis during physiological and premature aging

Description: We are broadly interested in identifying mechanisms that control age-related atherosclerosis because: a) the world population is undergoing a profound demographic change due to progressive aging; b) aging is the main risk factor for atherosclerotic cardiovascular disease (aCVD), which is the number one killer in developed countries and is expected to become soon the leading cause

of morbi-mortality worldwide; c) most aCVD-related deaths are considered premature and preventable. Our laboratory has made seminal contributions into the role of nuclear A-type lamins in pathophysiological processes, and has led research into Hutchinson-Gilford progeria syndrome (HGPS), a rare premature aging syndrome caused by progerin, a mutant form of lamin A that provokes exaggerated aCVD and premature death. Moreover, progerin has been detected at low level in aged tissues of non-HGPS individuals, suggesting a role in normal aging. Understanding how progerin causes CVD and premature aging may therefore shed light on normal aging, and viceversa. In this research we will use animal models and analysis of human samples to unravel new cellular and molecular mechanisms through which nuclear A-type lamins and progerin regulate aCVD and aging.

CNIC Group 13: Mechanoadaptation and Caveolae Biology

PI: Miguel Ángel del Pozo

Research Line: Neurovascular coupling: blood flow mechanosensing and atherogenesis

Description: Our group studies disturbed blood flow mechanosensing by arterial Caveolae, omega-shaped invaginations promoting atherosclerosis (AS). Brain blood flow is exquisitely regulated to match regional energy demands by a complex interplay between neurons and blood vessels termed “neurovascular coupling” (NVC). Caveolae have been recently shown to determine NVC specifically at arterioles, where blood flow is high as compared to capillaries, devoid of caveolae but still expressing Cav1 (the main protein component of caveolae). On the other hand, we have recently discovered dolines, non-caveolar Cav1-invaginations capable of responding to subtle changes in plasma membrane tension, like those occurring at capillaries, where we hypothesize they localize. NVC dysregulation leads to Alzheimer’s disease (AD) and other cognitive impairment disorders, and AD and AS are intriguingly linked by ApoE4, the only known predictor of the first. Thus, this project bears potential transcriptional impact. The successful candidate will combine in vitro and in vivo models to uncover the role of Cav1 across brain vasculature, analyzing how caveolae vs dolines regulate blood flow sensing in different brain regions. This research falls within the context of the AtheroConvergence international consortium, coordinated by the PI. More information: EMBO Pubmed

CNIC Group 14: Nanomedicine & Molecular Imaging

PI: Carlos Pérez-Medina

Research Line: Human antibody-enabled cardiovascular personalized theragnosis

Description: The ultimate goal of our research is to develop fully human antibodies (HuAbs) into diagnostic tools and personalized therapeutic agents to treat atherosclerosis. Specifically, we are isolating promising candidates from a macrolibrary of HuAbs selected in vivo from atherosclerotic samples. These candidates subsequently are characterized and produced in different formats suitable

for therapeutic purposes or imaging with positron emission tomography (PET). In vivo testing of HuAb-based therapies and PET radiotracers is carried out in mouse and pig models of atherosclerosis.

CNIC Group 15: B Lymphocyte Biology

PI: Almudena Ramiro

Research Line: Antibody-mediated atheroprotection: novel perspectives to immunotherapeutic approaches in cardiovascular disease

Description: Atherosclerosis progression involves various arms of the adaptive immune response, whose role in in the disease is still poorly understood. For the last years we have been studying the antibody adaptive immune response in atherosclerosis and we have identified atherosclerosis-associated antibodies that can have a protective function in this disease. Our research focuses on understanding the mechanisms of atheroprotection mediated by antibodies, with the ultimate aim of designing novel immunomodulatory approaches of therapeutic interest.

CNIC Group 16: Immunobiology

PI: David Sancho

Research Line: Gut microbiota-derived metabolites in atherosclerosis diagnosis, prognosis and therapy

Description: The gut microbiota influences our metabolism and may affect atherosclerosis (AT) progression. However, how the specific products from microbiota (metabolites) can modulate AT is poorly known. Our group studies the association of gut microbiota and their metabolites with AT, focusing on mechanisms driving inflammation and on their connection with macrophages. We assess the potential of microbial metabolites to identify AT in groups of human volunteers with early AT, and their potential to predict future AT by studying patients with more advanced AT. We also investigate the mechanism of action of these promising metabolites in order to find new targets with potential to treat AT. Our research can result in new diagnostic/prognostic tools for AT and can unveil novel strategies for the prevention and/or therapy of AT.

CNIC Group 17: Cardiovascular Proteomics

PI: Jesús Vázquez

Research Line: Application of Advanced computational methods for the analysis of PostTranslational Modifications to Cardiovascular Biology

Description: We are developing “open search” algorithms that allow true hypothesis-free identification and quantification of any post-translational modification from high-throughput mass spectrometry-based proteomics. In this project, we aim to further extend the development of semi supervised approaches to

the interpretation of the data and to integrate the quantitative information in large-scale experiments. We will also apply these developments to study molecular mechanisms underlying cardiovascular diseases, including subclinical atherosclerosis and other diseases related to vascular-remodeling.

CNIC Program: Cardiovascular regeneration

Coordinator: Miguel Torres – Clinical leader: Hesham Sadek

CNIC Group 18: Genetic Control of Organ Development and Regeneration

PI: Miguel Torres

Research Line: Coronary Lymphatic Vasculature Development and roles in Cardiac Repair

Description: The contractile ability of the mammalian heart critically relies on blood coronary circulation, essential to provide oxygen and nutrients to myocardial cells. In addition, the lymphatic vasculature is important for the myocardial immune response, extracellular fluid homeostasis and response to injury. Recent studies identified different origins of coronary lymphatic endothelial cells, however, the cues that govern coronary lymphangiogenesis remain unknown. Furthermore, the roles of the coronary lymphatic vasculatures following injury of the adult heart remain controversial. We have developed new mouse models that allow to study the cellular origins of the coronary lymphatic vasculature and its essential interactions with the epicardium. In this project we will exploit these models to determine the mechanisms that allow coronary lymphatic vessel angiogenesis during development and after acute myocardial ischemia. Furthermore, we will study the roles of the postnatal cardiac lymphatic vessels following cardiac injury.

References:

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Liu et al., Nature, 588(7839):705-711. doi: 10.1038/s41586-020-2998-x

De la Cruz et al. bioRxiv 2023.02.10.528007; doi: https://doi.org/10.1101/2023.02.10.528007

CNIC Group 19: Myocardial regeneration via cardiomyocyte cell cycle regulation

PI: Hesham Sadek

Research Line: Myocardial regeneration via cardiomyocyte cell cycle regulation

Description: The long-term goal of Sadek Lab research program is to find a cure for heart failure through the discovery of key mechanisms that regulate endogenous heart regeneration in mammals. Specifically, my laboratory is interested in understanding how the endogenous regenerative properties of the

mammalian myocardium are regulated by intrinsic cardiomyocyte mechanisms as well as by signals in the intrauterine and postnatal environment. Ultimately, we aim to leverage these findings to identify therapeutic targets that can reactivate cardiomyocyte proliferation and induce myocardial regeneration in humans.

CNIC Group 20: Molecular Genetics of Angiogenesis

PI: Rui Benedito

Research Line: Mechanisms and therapies to promote endothelial protection against reperfusion injury

Description: Reperfusion of vessels with oxygen rich blood after an ischemic event exacerbates the endothelial damage caused by hypoxia, leading to the further loss of blood vessels and surrounding tissue damage. Our lab has identified that arterialized endothelial cells are protected against the transition from hypoxia to normoxia caused by the reperfusion. Our research aims at genetically and pharmacologically arterializing capillary ECs, in order to protect blood vessels and the surrounding tissue against the damaging effects of reperfusion.

CNIC Group 21: Intercellular Signaling in Cardiovascular Development and Disease

PI: José Luis de la Pompa

Exploring the molecular mechanisms regulating cardiomyocyte maturation. Description: Ventricles power the heart's beat. The signaling molecule Neuregulin-1 (Nrg1) is vital for trabecular growth and ventricular wall maturation, orchestrating cardiomyocyte processes like migration, adhesion, and cell cycle progression. We have identified a set of Nrg1-dependent genes which appear to be involved in cardiomyocyte differentiation and ventricular maturation (PMID: 37846569).

Phenotypic transitions in genetic cardiomyopathies. Description: Genetic cardiomyopathies are heart muscle disorders caused by genetic mutations. Mouse modelling of disease-causing mutations identified in families with mixed cardiomyopathy phenotypes (HCM+LVNC), indicates that there is a developmental transition involving the transcription factor Prdm16, in which an early fetal-neonate hypertrabeculation evolves postnatally into hypertrophic cardiomyopathy. We characterize the underlying molecular mechanisms with the ultimate goal to generate fundamental knowledge with translational implications for CHD (PMID: 36325906, 37405741).

CNIC Group 22: Functional Genetics of the Oxidative Phosphorilation System (GENOXPHOS)

PI: José Antonio Enríquez

In silico design of pharmacological targets for heart regeneration. Description: Identification of novel therapeutics in heart regeneration is demanded. This research aims to discover potent therapeutic drugs

based on the expression profile of heart-specific genes involved in regeneration by comprehensively analyzing sequencing data exclusively.

Deciphering of the mitochondrial complexes behind heart regeneration. Description: The supramolecular structures of complex IV have different metabolic and physiological functions, ranging from metabolic maturation necessary for correct tissue physiology, to plasticity and adaptation to different metabolic requirements. Using different genetic approaches, the research aims to understand the supramolecular organization behind the heart regeneration**.**

Preclinical model of heart regeneration therapy based on mitochondrial performance. Description: OMA1 is a mitochondrial protease that participates in various processes: the regulation of mitochondrial structure, the activation of the integrated stress response and, eventually, the cell's entry to apoptosis. Current efforts are focused in proving the therapeutic potential of this target in heart regeneration, exploring the possible adverse effects that the absence of this protein might have, due to the marked translational nature of this research.

CNIC Group 23: Development of the epicardium and its role during regeneration

PI: Nadia Mercader

Research Line: Medaka as a model to test genes and molecules promoting heart regeneration

Description: The natural capacity of zebrafish for heart regeneration is not shared by a second teleost fish, namely Medaka. **This research aims at using this latter model for a small molecules and genetic screening of pro-regenerative compounds and genes**.

CNIC Group 24: Cardiac tissue engineering and regenerative therapies

PI: Florian Weinberger

Research Line: Harnessing anti-fibrotic strategies to improve cardiomyocyte transplantation

Description: The research aims to combine cardiomyocyte differentiation from human IPSCs with different pharmacological and genetic anti-fibrotic strategies to improve in vivo cardiomyocyte transplantation in animal models. We hypothesize that this approach will allow cardiomyocytes to better invade the fibrotic scar tissue and replenish the injured area with more new muscle following injury. Combining different genetic (i.e. genetic modifications of iPSC), molecular, and imaging tools with tissue engineering and physiological analysis of cardiac function, we will study the functional consequences of anti-fibrotic strategies on cardiomyocyte transplantation success. The goal is to eventually develop strategies to improve cardiomyocyte transplantation.

CNIC Program: Novel arrhythmogenic mechanisms

Coordinator: Silvia Priori – Clinical leader: David Filgueiras

CNIC Group 25: Molecular Cardiology

PI: Silvia Priori

Research Line: DNA and RNA Therapies for the management of ventricular arrhythmias and prevention of sudden cardiac death

Description: The research is focused on two severe inherited arrhythmogenic diseases: catecholaminergic polymorphic ventricular tachycardia (CPVT) and Long QT syndrome type 8 (LQT8). The group is currently working on two existing mice models for the dominant and the recessive forms of CPVT with the objective of investigating the effects of gene therapy strategies developed by the Molecular Cardiology Laboratories at the ICS Maugeri Institute in Pavia (Italy) on intracellular calcium handling and cellular electrophysiology. A most ambitious effort of the group is the development of a knock-in pig to model LQT8.

CNIC Program: Cardiovascular health promotion

Coordinator: Rodrigo Fernández Jiménez – Clinical leader: Valentín Fuster

CNIC Group 26: Cardiovascular Health and Imaging

PI: Rodrigo Fernández Jiménez

Research Line: Cardiovascular health trajectories and outcomes

Description: This research aims to study the association of different lifestyle and cardiovascular risk factors (unhealthy diet, low levels of physical activity, obesity, etc.) with cardiovascular and brain health outcomes in humans using a longitudinal approach and advanced scientific methods.