RESEARCH TOPIC MEM3
Inflammatory and Epigenetic Drivers in Heart Failure

Curriculum MEM standard

Research Area
Cardio

Laboratory name
Molecular cardiology

Research Supervisor
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Abstract
Heart Failure with preserved Ejection Fraction (HFpEF) is gradually becoming the most prevalent form of heart failure globally. HFpEF management is primarily based on treating underlying conditions and symptoms, and there are limited evidence-based pharmacotherapies for this disorder, which creates an urgent clinical need. A key factor for our scarce therapeutic armamentarium to combat HFpEF is the pathophysiological and etiological heterogeneity of this disorder, which hinders the development of rationally-designed therapeutics. Tackling this heterogeneity requires either de design of personalized therapeutic strategies or the identification of pathophysiological mechanisms shared by the different subtypes of HFpEF, which could pave the way for more universal therapies. In the setting of shared mechanistic drivers of HFpEF, a hypothesis that has received some experimental support in recent years is that exacerbated inflammatory responses underlie many forms of HFpEF. However, the specific immunomodulatory mechanisms that, when dysregulated, drive the development and clinical progression of HFpEF in humans remain poorly understood. The candidate will work on the understanding of the molecular mechanisms of HFpEF.

Main technical approaches
Basic knowledge of molecular and cellular biology techniques

Scientific references
Panico C et al. Single-Cell RNA Sequencing Reveals Metabolic Stress-Dependent Activation of Cardiac Macrophages in a Model of Dyslipidemia-induced Diastolic Dysfunction, Circulation, (2023) 10.1161/CIRCULATIONAHA.122.062984

Serio S, et al: Cardiac aging is promoted by pseudohypoxia increasing p300-induced glycolysis. Circulation Research, 2023;133:687–703 doi: 10.1161/CIRCRESAHA.123.322676

Martini E et al. Circulation 2019 Dec 17;140(25):2089-2107. doi: 10.1161/CIRCULATIONAHA.119.041694.

Anselmo A, et al. , Myocardial hypoxic stress mediates functional cardiacextracellular vesicle release. European Heart J, 2021 Jul 21;42(28):2780-2792

Kallikourdis M, et al. T cellcostimulation blockade blunts pressure overload-induced heart failure: Nature Comm, 8:14680.

Type of contract

Scholarship of € 25.000 gross per year awarded by Istituto Clinico Humanitas. This sum is subject to IRPEF income tax and exempt from social security contributions.

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