Universidad de Navarra

Máster en Investigación Biomédica
Facultad de Ciencias

**Research Project Proposal**
Academic year 2016-2017

**Project Nº 28**

**Title:** Study of the mechanisms involved in fibroblasts activation in Myocardial Infarction and development of new nanotechnological therapeutic strategies

**Department/ Laboratory:** Stem Cell Laboratory (1.01) / Center for Applied Medical research, Center for Applied Medical research, CIMA

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**Summary**

The cardiovascular diseases constitute the greatest health risk in the occidental countries. According with the last inform of the World Health Organization (http://www.who.int/cardiovascular_diseases/en) these pathologies provoke at the global level, around 30% of the deaths, equivalent to more than 17 million annual deaths, from which the ischemia is the principal cause (1). In the case of myocardial infarction, the main problem is the lack of an effective regeneration of the myocardium after ischemia, which ends up in an irreversible loss of the cardiac tissue and its substitution by a non-functional scar. This remodeling process occurs as consequence of fibroblasts activation which are the principal mediators of collagen deposit and scar formation (2). Interestingly, different epigenetic mechanisms are involved in the activation of these cells that are not deeply understood yet (3).Therefore, as cardiac fibroblasts play a prominent role in heart scarring, it is essential to understand and control their activity in order to develop efficient treatments for heart failure. In this project, we will study such mechanisms in a transgenic Cg1-GFP mouse model (healthy and infarcted). Cg1-GFP+ (myo)fibroblasts will be selected from hearts and gene/microRNA expression analyzed by RNAseq. Histological characterization will be performed also in order to determine the mechanisms involved. The results obtained from these studies will be of great relevance not only for better understanding the mechanisms of fibrosis in the heart but also to develop new nanotechnological therapeutic strategies that will be next tested in our murine model of myocardial infarction.

**References**

**1.** Mozaffarian D, Benjamin EJ, Go AS et al. Heart Disease and Stroke Statistics-2016 Update: A Report From the American Heart Association. Circulation. (2016) 133(4):e38-e360.

**2.** Travers JG, Kamal FA, Robbins J et al. Circ Res. 2016;118(6):1021-40. Cardiac Fibrosis: The Fibroblast Awakens.

**3.** Boon RA, Dimmeler S. MicroRNAs in myocardial infarction. Nature Reviews Cardiology. 2015;12(3):135-42.

**POSSIBILITY OF PhD**

YES*

* (PhD grant required)