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Letter to Editor

Research Trends in Heart Failure and Fibrosis

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Abbreviations

LOX: Lysyl Oxidase; ECM: Extracellular Matric; HF: Heart Failure; AngII: Angiotensin II; CFs: Cardiac Fibroblasts

Heart failure is a complex syndrome affecting 64.3 million people only in 2017. Cases seem to soar globally and approximately 3.500 patients are included in the waiting list for heart transplant this year. HF is characterized by a pro-inflammatory profile and fibrotic tissue which in non- ischemic cases appears to be reversible [1]. Fibrosis represents a key role in the pathophysiology of HF via stimulation of Left ventricular remodeling and contributing to diastolic dysfunction [2,3].

In an ageing continent, all of the well-known cardiovascular risk factors (hypertension, diabetes, smoking etc.) have detrimental effects on the class of the interstitial fibrosis (Figure 1) [4]. Due to that knowledge, targeting personalized therapy in patients with interstitial fibrosis may lead to extension of the myocardiac viability [5].

Figure 1: Central illustration-Left Ventricular Macrostructural and Microstructural Remodeling in Healthy Aging, Stage A and Stage B Heart Failure [1].

According to Lim, experiments in mice models have shown that cardiac macrophages produce and respond to IL-10 (fibrosis-related cytokine) under certain stimulation [6]. As responders, they express osteopontin and indirectly activate cardiac myofibroblasts, promoting collagen deposition and myocardial stiffness. Also, several other transduction molecules contribute to the remodeling process (TGF-β, AngII, Aldosterone, lactoferine, A1 annexin). Hence, new drug strategies targeting the macrophage microenvironment are at the forefront [6,7].

Regarding reparative and interstitial fibrosis, Lysil oxidase (LOX) is an enzymic family known for its pivotal role in regulating collagen fibers cross-linking (Figure 2). However, according to Gonzalez A., *et al*. LOX may be involved in motility and migration of fibroblasts. From a clinical aspect, in experimental models, cardiac LOX mRNA found increased in patients with dilated cardiomyopathy [5]. In this framework, LOX emerges as an attractive novel therapeutic target [8].

Figure 2: Types of fibrosis [6].

The trans differentiation of cardiac fibroblasts into myofibroblasts is a "worthy of attention" process as the pro-inflammatory features of cardiac fibroblasts determining the level of myofibroblasts' maturation and ensure the proper clearance of the wound after the ischemic process. Recent murine studies have shown that depletion of specific receptors in isolated CFs results to less inflammation and subsequently to a sufficient ECM environment [9].

In the COVID era, the recent successful mRNA vaccine development, growned a special interest in mRNA therapeutics. Vascular Endothilian Growth Factor vaccines induces cardiovascular regeneration by increasing the capillary density in the injured muscle. Histological examination of humans after the injection have shown increased capillary and arteriole density and reduced fibrosis in the peri-infarct region [10].

Finally, the field of myocardial fibrosis is continually evolving and ground-breaking research is involved. However, connection of the preclinical research with patient care is needed. With preceded circulating biomarkers and the integration of cardiac imaging application, these findings may diminish the burden of cardiovascular disease.

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