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A Brief Note on Cardiac Fibroblast

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Editorial Note

The cardiac fibroblast is the predominant cell type responsible for the secretion of a majority of ECM components in the heart, including collagens I, III, and IV, as well as laminin and fibronectin. It accounts for about 90% of nonmyocyte cells in the heart. A fraction of fibroblasts undergoes phenotypic conversion to myofibroblasts in response to mechanical stress and neurohormonal stimulation, which are characterised by increased expression of -smooth muscle actin and increased secretory activity. Myofibroblasts, which are important for collagen production and the contraction/realignment of nascent collagen fibres, are derived from tissue-resident fibroblasts that become activated after tissue injury, according to recent research. Myofibroblasts move into the tissue's immediate vicinity and play a key part in scar formation. Multiple paracrine signalling pathways may be used by cardiac myofibroblasts to alter the phenotype of cardiac myocytes.

According to several lines of evidence, cardiac fibroblasts and myocytes secrete proteins that regulate surrounding cells. Transforming Growth Factor-1 (TGF-1), Fibroblast Growth Factor-2 (FGF2), members of the IL-6 family, and the recently identified cytokine IL-33 are among the proteins that have been involved thus far. Mast cells, which are bone marrow-derived cells that "home" to and reside in the myocardium, may also play a role in ECM remodelling, according to growing data.

Mast cells in the myocardium are found mostly around blood arteries and between myocytes, where they can release profibrotic cytokines and growth factors that impact ECM remodelling. Mast cells attracted to the heart during inflammation were found to be responsible for TGF-1–mediated fibroblast activation, myocardial fibrosis, and LV diastolic dysfunction in animal experiments.

The increasing rise in collagen content of the heart, as previously mentioned, is one of the histologic hallmarks of progressing HF (myocardial fibrosis). In individuals with ischemic cardiomyopathy, studies in failing human myocardium have revealed a quantitative rise in collagen types I, III, VI, and IV, as well as fibronectin, laminin, and vimentin, and a drop in the type I/III collagen ratio. Clinical investigations also demonstrate a progressive loss of collagen cross-linking in failing hearts, as well as a loss of collagen network connectivity with individual myocytes, which would be predicted to result in significant changes in LV form and function.

Furthermore, loss of fibrillar collagen cross-linking has been linked to progressive LV dilatation following myocardial damage. Collagen buildup can happen "on the fly" around intramural coronary arteries and arterioles (perivascular fibrosis) or in the interstitial space (interstitial fibrosis), and it doesn't require myocyte cell death. Collagen can also accumulate as a result of microscopic scarring that develops as a result of cardiac myocyte cell death.

This scarring, also known as "replacement fibrosis," is a response to the loss of parenchyma and is essential for the heart's structural integrity. Increased fibrous tissue would be expected to increase myocardial stiffness, which would likely lead to less cardiac shortening for a given degree of afterload. Furthermore, myocardial fibrosis may operate as a structural basis for atrial and ventricular arrhythmias, resulting in homogeneous activation, bundle branch block, and dyssynchrony, as well as sudden death.

Although the complete complement of molecules responsible for fibroblast activation is unknown, several of the typical neurohormones and cytokines produced in HF (ET, TGF-, cardiotrophin-1) are adequate to activate fibroblasts. In experimental HF models, the administration of ACE inhibitors, beta blockers, and aldosterone receptor antagonists has been linked to a reduction in myocardial fibrosis.

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