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Molecular Basis of Cardiac Fibrotic Remodeling: Prognosis of Fibrosis in the Heart

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Deadline for manuscript submissions:
closed (20 October 2024)

Message from the Guest Editors

Dear Colleagues,

Cardiac fibrosis is a pathological process with excessive secretions and deposits of extracellular matrix proteins such as collages, elastin, proteoglycans, and fibronectin. The differentiation of fibroblasts to myofibroblasts is recognized as a critical process in fibrosis. Transforming growth factor beat (TGF-β) promotes this process through Smad-2/3-dependent or -independent pathways. Cellular sources and molecular mechanisms that regulate TGF-β production and function remain incompletely understood.

Atrial fibrotic remodeling is associated with atrial fibrillation. Expansion and dilatation of ventricles result in cardiac remodeling and eventual heart failure. Fibrotic remodeling is one of the major features of cardiac remodeling. Molecules that prevent or ameliorate cardiac fibrosis may be novel targets for chronic heart failure, coronary artery disease, diabetic cardiomyopathy, or other heart disease.

The aim of this Special Issue is to highlight recent advances of molecules with definitive mechanisms of cardiac fibrosis and remodeling. Investigators of fibrotic remodeling of the heart are especially encouraged to submit.

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