### **UCLA**

# **UCLA Previously Published Works**

#### **Title**

Cardiac fibroblasts: The good, the bad, the ugly, the beautiful

### **Permalink**

https://escholarship.org/uc/item/5bs6w5h7

#### **Authors**

Sadoshima, Junichi Weiss, James N

### **Publication Date**

2014-05-01

#### DOI

10.1016/j.yjmcc.2014.03.009

Peer reviewed



Contents lists available at ScienceDirect

## Journal of Molecular and Cellular Cardiology

journal homepage: www.elsevier.com/locate/yjmcc



Editorial

### Cardiac fibroblasts: The good, the bad, the ugly, the beautiful



This special issue is focused on the role of fibroblasts in cardiac biology. Fibroblasts are the most numerous cells in the heart, and although their role in promoting fibrosis in the diseased heart has been recognized for more than 50 years, it is only more recently that their active involvement in both normal cardiovascular biology and many other aspects of cardiac pathophysiology besides fibrosis have been recognized. Not only do fibroblasts play a key role in cardiac development, but once the heart has achieved its mature adult form, fibroblasts are the sentinel cells that are continuously reshaping the extracellular matrix to optimize the contractile function of the myocytes embedded within that matrix. Fibroblasts are also the key player in scar formation when the heart is injured. This becomes the double-edged sword in heart disease - when the heart is injured, scar formation is essential to maintain the structural integrity of the heart walls and prevent rupture - however, scar formation also impairs contractile performance and makes the heart susceptible to arrhythmias. The therapeutic dream is to be able to direct cardiac repair in a way that maintains the heart's structural stability without impairing its electromechanical function. Fibroblast biology will play a critical role in attempts to regenerate cardiac tissue using stem cell approaches, since fibroblasts will manufacture the extracellular matrix in which the regenerated myocytes reside. These are challenging issues, as the biology of cardiac fibroblasts is complex because they are comprised of heterogeneous populations of cells with distinct developmental origins which may dictate their basal functions as well as responses to stress.

Accordingly, the goal of this special issue is to provide a state-of-theart update on fibroblast biology as it relates to the heart. We have invited a number of leading researchers in this field to share their views on diverse aspects of fibroblast biology, ranging from its roles in cardiac development, to maintaining the adult heart in a healthy state, to heart diseases, and finally, to the therapeutic potential for repairing diseased hearts. The articles are arranged roughly in this sequence, first focusing on the cell biology of fibroblasts and myofibroblasts in the developing and adult heart, next reviewing their physiological roles as mechanosensors and modulators of cardiac electrophysiology via gap junction coupling, followed by their role in wound healing, aging, and cardiac pathologies including hypertrophy, ischemia, and arrhythmias. Finally, the last two articles focus on the therapeutic potential of modulating fibroblast biology.

We are indebted to our scientific colleagues for their willingness to take the time and effort to share their thoughts about this exciting field with the scientific community, and especially for doing so in a timely manner. We are also very grateful to the editorial staff of the Journal who made our role as the editors of this special issue a pleasure to undertake.

#### **Conflict of interest**

None.

Junichi Sadoshima

Department of Cell Biology and Molecular Medicine, Rutgers New Jersey Medical School, 185 South Orange Avenue, MSB G609, Newark NJ 07103, USA

Tel.: +1973728619

E-mail address: sadoshju@njms.rutgers.edu.

James N. Weiss

Division of Cardiology, David Geffen School of Medicine at UCLA 3645 MRL Bldg 675 C, Young Drive So., Los Angeles, CA 90095, USA Corresponding author. Tel.: +13108259029.

E-mail address: jweiss@mednet.ucla.edu.

13 March 2014