EuroSciCon

May 24-25, 2018
London, UK

EuroSciCon Conference on
Clinical Cardiology and Cardiovascular Disease

Carlin S Long et al., Interv Cardiol J 2018, Volume: 4
DOI: 10.21767/2471-8157-C1-001

USING A DUAL-REPORTER MOUSE TO TRACK FIBROBLAST CELL TRANSITIONS

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Cardiac fibroblasts serve important roles in cardiac structure and intercellular communication in both normal and injured myocardium. While infiltrating, immune-inflammatory cells are the primary initiators of the early phase of the response to injury, cardiac fibroblasts are the principal resident tissue cell involved throughout the process of wound healing. Two critical aspects of cardiac fibroblast phenotype in response to injury are well recognized. First, the transition into the myofibroblast phenotype, so named due to their expression of contractile proteins, like smooth muscle alpha-actin which contribute to wound contracture. The myofibroblast is the primary source of collagen deposition which may persist for long periods of time following resolution of injury and scar maturation. Second, the physiologic resolution of the wound healing response requires the myofibroblast to inactivate these functions and return to the quiescent basal state. It is presumed that termination occurs by apoptosis, although the regulatory mechanisms remain undefined. Thus physiologically appropriate functions of cardiac fibroblasts require profound phenotypic transitions, and termination of the activated phenotype. Studying hepatic fibrosis, David Brenner used a unique transgenic reagent which simultaneously express the red fluorescent protein (RFP) under control of the alpha smooth muscle actin (aSMA) promoter and the green fluorescence protein (EGFP) under the control of collagen a1 (I) promoter. We took advantage of these animals to study these phenotypic transitions both *in vivo* and *in vitro*. For the *in vitro* studies we have used an automated program of "counting" red, green and yellow (red + green) cells and have subjected these cells to high-throughput screening in the presence of chemical libraries and candidates found to have the most promising with this approach *in vivo*. We believe this represents a unique approach for defining therapeutic approaches to study pathologic fibrosis with great potential.

Biography

Carlin S Long is a UCSF Professor of Medicine and Director of the Center for Prevention of Heart and Vascular Disease. He has earned his MD at the University of Texas Southwestern Medical School and received his Internal Medicine and Cardiology training at the University of California San Francisco where he stayed as faculty member until 1998 when he joined the faculty of the University of Colorado. His research is focused on understanding the role of pro-inflammatory molecules in the transition from compensated to decompensated myocardial failure. He is particularly interested in how the cells in the heart "speak" to one another in normal and abnormal growth with a particular interest on the cardiac fibroblast which initiates the process of repair within the heart muscle in response to injuries such as heart attack, but also that seen in long-standing high blood pressure and certain valvular diseases.

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