David E. Dostal, J Clin Exp Cardiolog 2014, 5:3
http://dx.doi.org/10.4172/2155-9880.S1.019

OMICS Group Conferences
Accelerating Scientific Discovery

4th International Conference on
Clinical & Experimental Cardiology
April 14-16, 2014 Hilton San Antonio Airport, TX, USA

Mechanosensing and regulation of cardiac function

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Heart failure is the leading cause of morbidity and mortality in developed countries. Cardiac dysfunction in patients with hypertension-induced heart failure is characterized by reduced left systolic and diastolic ventricular function, which is associated with myocyte hypertrophy and ventricular remodeling. Although the pathophysiological mechanisms associated with pressure overload-induced cardiac hypertrophy have the focus of intense scientific investigation for over 3 decades, the cellular mechanisms remain poorly understood. There is abundant evidence that regulation of protein phosphorylation through intracellular kinases and phosphatases is a key mechanism by which cells respond to extracellular stimuli, Within this area of research, the stress activated protein kinases (SAPKs), which include c-jun N-terminal kinases (JNKs) and p38 MAP kinase, have been shown to be activated by a number of cellular stresses including mechanical stretch. Recent studies by our laboratory and others suggest that p38 is responsible for causing many of the pathological aspects of heart failure, whereas JNK may be cardioprotective. Using *in vivo* and *in vitro* models, we have demonstrated that both β1-integrin and the angiotensin type I receptor (AT1) serve as mechanosensors, which can temporally regulate contractile function in cardiac myocytes through regulation of p38 and JNK. These studies also revealed that JNK plays a major role in maintaining cardiac diastolic function by regulating intracellular calcium through phosphorylation of phospholamban. The demonstration that JNK has an important role in the regulation of contractile function and diastolic function may provide a new therapeutic approach for the treatment of diastolic heart disease.

Biography

David E. Dostal received his Ph.D. in 1986 at the University of Missouri-Columbia followed by postdoctoral training at the University of Virgina, Charlottesville. Currently, he is a Professor at Texas A&M Health Science Center and Research Biologist at the Central Texas Veterans Health Care System in which his laboratory, funded by the NIH and the Veterans Affairs, conducts research in the area of heart failure and cellular signaling mechanisms. He also serves as a member of the NIH Cardiac Contractility, Hypertrophy, and Failure Study Section, Chair of an American Heart Association Signaling Study Section, an International Expert Panel member for the Singapore Ministry of Health and several scientific journal editorial boards. He has recently received the prestigious Research Career Scientist Award from the Department of Veterans Affairs for his scientific achievements.

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