Yin Hua Zhang, J Pharmacogenomics Pharmacoproteomics 2015, 6:3
http://dx.doi.org/10.4172/2153-0645.C1.006

OMICS International
conferenceseries.com
3rd International Conference on
Predictive, Preventive and Personalized Medicine & Molecular Diagnostics
September 01-03, 2015 Valencia, Spain

Functional analysis using metabolic substrates-induced *in vitro* model of cardiac metabolic syndrome

Yin Hua Zhang
Seoul National University, Korea

Cardiac metabolism is essential in myocardial contraction. Here, we analyzed the effect of metabolic substrates (fatty acids, pyruvate and lactate in normal Tyrode's solution, termed NF) on myocyte contractility in rat left ventricular myocytes. Our results showed that NF significantly increased myocyte contraction and intracellular Ca^{2+} transients. L-type Ca^{2+} current or Na^{+}-Ca^{2+} exchanger activity was not increased and myofilament Ca^{2+} sensitivity was reduced by NF, suggesting key role of myofilament on cardiac Ca^{2+} homeostasis and contraction with NF. Furthermore, NF diminished insulin-dependent tyrosine phosphorylations of insulin receptor or receptor substrate and eNOS-Ser^{1177}. Beta-adrenergic stimulation with isoprenaline significantly increased spontaneous myocyte contraction during diastole in NF. Collectively, NF impairs insulin signaling and reduces bioavailability of eNOS-derived NO, which desensitizes myofilament Ca^{2+} sensitivity, increases Ca^{2+} level and contraction. In addition, it predisposes beta-adrenergic arrhythmogenesis in cardiac myocytes. The results reveal that it resembles an *in vitro* model of cardiac metabolic syndrome.

Biography

Yin Hua Zhang has completed her PhD in 1999 from Seoul National University, College of Medicine and Post-doctoral studies from Oxford University, Cardiovascular Medicine. She directs Cardiovascular Laboratory studying Physiology and Pathology of the heart. She has published more than 40 papers in high ranking journals and is holding a number of national grants.

yinzhang@snu.ac.kr

Notes: