Peifeng Li, J Stem Cell Res Ther 2013, 3:5
http://dx.doi.org/10.4172/2157-7633.S1.010

OMICS Group Conferences
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2nd International Conference and Exhibition on
Cell & Gene Therapy

October 23-25, 2013 Holiday Inn Orlando International Airport, Orlando, FL, USA

MicroRNAs in the therapy of myocardial infarction

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Heart failure is a leading cause of mortality worldwide. Recently, a fundamental shift in the underlying etiology of heart failure has occurred, in which the most common cause of heart failure is no longer hypertension or valvular disease, but myocardial infarction. The heart is an organ composed of terminally differentiated post-mitotic cardiac myocytes. Since the loss of cardiomyocytes cannot be compensated by efficient cell proliferation, abnormal induction of apoptosis in cardiomyocytes may lead to pathophysiological disorders including myocardial infarction. However, patients with myocardial infarction can only receive symptomatic treatment, and an appropriate therapeutic strategy has not been well established. MicroRNAs (miRNAs) are a class of small noncoding RNAs about 22 nucleotides long, and they can bind to the 3' un-translated region (3'UTR) of mRNAs, thereby inhibiting mRNA translation or promoting mRNA degradation. Our work shows that microRNAs levels are altered in ischemia/reperfusion injury. Modulations of miR-499 levels can influence apoptosis, myocardial infarction and cardiac remodeling. We also found that miR-484 suppresses Fis1 translation and inhibits Fis1-mediated apoptosis and the consequent myocardial infarction. Intriguingly, cardiac fibroblasts can be reprogrammed to stem cells by microRNAs. Our data provide novel evidence suggesting that microRNAs are important regulators of myocardial infarction. The therapeutic approaches for myocardial infarction can be developed by modulating microRNAs.

Biography

Peifeng Li has completed his MD and Ph.D. at the age of 30 years from Chinese Academy of Medical Science and postdoctoral studies from Max-Delbruck Center for Molecular Medicine, Germany. He is a Research Assistant Professor at University of Illinois at Chicago. He has published more than 62 papers in reputed journals and has been serving as an editorial board member of Dataset Papers in Biology.

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