Peifeng Li et al., 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

miR-140 regulates cardiomyocyte apoptosis by targeting mitofusin-1

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MicroRNAs (miRNAs) are a class of small non-coding RNAs that mediate post-transcriptional gene silencing. Mitochondrial fission participates in the induction of apoptosis. It is poorly understood as to how mitochondrial fission program is regulated in cardiomyocytes. In particular, it remains largely unknown whether miRNAs can regulate mitochondrial fission. Reactive oxygen species and doxorubicin could induce mitochondrial fission and apoptosis in cardiomyocytes. Concomitantly, mitofusin 1 (Mfn 1) was downregulated, whereas miR-140 was upregulated upon apoptotic stimulation. We investigated whether Mfn1 and miR-140 play a function role in mitochondrial fission and apoptosis. Ectopic expression of Mfn1 attenuated mitochondrial fission and apoptosis. Knockdown of miR-140 inhibited mitochondrial fission. Our result further revealed that knockdown of miR-140 was able to reduce myocardial infarct sizes in the animal model. We explored the relationship between Mfn1 and miR-140, and observed that miR-140 could suppress the expression of Mfn1, and it exerted its effect on mitochondrial fission and apoptosis through targeting Mfn1. Our data revealed that mitochondrial fission occurs in the cardiomyocytes and can be counteracted by Mfn1. However, the function of Mfn1 is negatively regulated by miR-140. Our present work suggests that Mfn1 and miR-140 are integrated into the program of cardiomyocyte apoptosis.

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 an Associate Professor at Nebraska Medical Center. He has published more than 62 papers in reputed journals and has been serving as an editorial board member for many journals such as Dataset Papers in Biology, Journal of Clinical and Experimental Cardiology.

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