Sarnai Battsengel, J Clin Exp Cardiolog 2015, 6:4
http://dx.doi.org/10.4172/2155-9880.S1.024

OMICS International
conferenceseries.com

5th International Conference on
Clinical & Experimental Cardiology
April 27-29, 2015 Philadelphia, USA

ADSC enhance the miR-181b regulation in apoptosis signaling pathway in through ADSC/cardiac cells co-culture under Pg-LPS treatment

Sarnai Battsengel
China Medical University, Taiwan

Heart disease and other cardiovascular diseases are leading cause of death globally. Infections causing periodontal disease are still a major risk factor for cardiovascular disease. Porphyromonasgingivalis is a major pathogen of periodontal disease. Recently Stem cells represents a promising therapy for heart disease such as myocarditis, ischemia. miRNAs are small non coding RNAs of 22-30 nucleotidies in length. Role of miRNA is very important to control cellular development and cellular process in eukaryotes. Main function of miRNA is to down-regulate gene expression in a variety of manners, including translational repression, mRNA cleavage, and deadenylation. Aim of this study is to find out the regulatory role of miRNA 181b in cardiac apoptosis signaling pathway under Pg-LPS (LPS isolated from porphyromonas oral Periodontal bacteria) challenge.Further we checked whether co-culture with Adipose Derived Stem cell (ADSC) regulates miRNA-181b H9c2 cells under Pg-LPS treatment. We found that ADSC/cardiac cell co-culture under Pg-LPS treatment, reverse the apoptosis protein markers and enhance survival markers in H9c2 cells. Then we found that miR-181b significantly decreased in H9c2 under Pg-LPS treatment. However it has reversed in ADSC/coculture group under Pg-LPS treatment by QPCR assay. Further we checked whether miRNA 181b regulates apoptosis signaling pathway in H9c2 by targeting the caspase3 signaling pathway. While miR-181b mimic decreased caspase3, miR-181b inhibitor increased caspase. Furthermore we observed cell viability by MTT assay and miR 181b-mimic increased cell viability whereas, miR-181b inhibitor decreased cell viability. Meanwhile, survival protein markers in H9c2 cells co-cultured with ADSC increased with miR-181b mimic and decreased with miR-181b inhibitor. Therefore we conclude that ADSC enhances miR-181b regulation in H9c2.

Biography

Sarnai Battsengel is studying in China Medical University,Taichung Taiwan for Master Degree.

sarnai.tsengel@gmail.com

Notes: