OMICS Group Conferences
Accelerating Scientific Discovery

3rd International Conference on
Clinical & Experimental Cardiology

April 15-17, 2013 Hilton Chicago/Northbrook, USA

MIR-206 regulates pulmonary artery smooth muscle cell proliferation and differentiation

Narasaiah Kolliputi

University of South Florida, USA

Abstract

Pulmonary Arterial Hypertension (PAH) is a progressive devastating disease characterized by excessive proliferation of the Pulmonary Arterial Smooth Muscle Cells (PASMCs). Studies suggest that PAH and cancers share an apoptosis-resistant state featuring excessive cell proliferation. MicroRNA-206 (miR-206) is known to regulate proliferation and is implicated in various types of cancers. However, the role of miR-206 in PAH has not been studied. In this study, it is hypothesized that miR-206 could play a role in the proliferation of PASMCs. In the present study, the expression patterns of miR-206 were investigated in normal and hypertensive mouse PASMCs. The effects of miR-206 in modulating cell proliferation, apoptosis and smooth muscle cell markers in human pulmonary artery smooth muscle cells (hPASMCs) were investigated in *vitro.* miR-206 expression in mouse PASMCs was correlated with an increase in right ventricular systolic pressure. Reduction of miR-206 levels in hPASMCs causes increased proliferation and reduced apoptosis and these effects were reversed by the overexpression of miR-206. miR-206 over expression also increased the levels of smooth muscle cell differentiation markers α-smooth muscle actin and calponin implicating its importance in the differentiation of SMCs. miR-206 overexpression down regulated Notch-3 expression, which is key a factor in PAH development. These results suggest that miR-206 is a potential regulator of proliferation, apoptosis and differentiation of PASMCs, and that it could be used as a novel treatment strategy in PAH.

Biography

Narasaiah Kolliputi is an Assistant Professor at the University of South Florida. He graduated from Osmania University, India, where he received a doctoral degree in biochemistry. He received his postdoctoral training in MGH at Harvard Medical School. His present work at USF involves elucidating translational strategies to attenuate pulmonary arterial hypertension (PAH) and acute lung injury (ALI). Dr. Kolliputi's research is funded by NIH RO1 and American Heart Association Scientist Developmental grants.