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Title: Mir-17 modulates smooth muscle cell markers, apoptosis and BMPR-II levels in human pulmonary artery smooth muscle cells

Dr. Gurukumar 31058 Kollongod Ramanathan gkollong@health.usf.edu [1], Ms. Prasanna 31059 Tamarapu ptamarap@health.usf.edu [1], Ms. Asfiya 31060 Younis ayunus2l@mail.usf.edu [1], Mr. Samuel 31061 Jalali samuel@mail.usf.edu [1], Ms. Sara 31062 Garcia sgarcia@mail.usf.edu [1], Dr. Richard F. 31073 Lockey rlockey@health.usf.edu [1] and Dr. Narasaiah 31074 Kolliputi nkollipu@health.usf.edu [1]. [1] Division of Allergy and Immunology, Joy McCann Culverhouse Airway Disease Center Department of Internal Medicine, Morsani College of Medicine, University of South Florida, Tampa, FL, United States, 33612 .

Body: Pulmonary Arterial Hypertension (PAH) is a progressively devastating disease characterized by excessive proliferation of the Pulmonary Arterial Smooth Muscle Cells (PASMCs). Recently micro RNA (miR) have been shown to play an important role in the pathogenesis of PAH. We describe in the present study the effects of over expression of miR17/92 as a cluster or miR17 alone on human pulmonary artery smooth muscle cells (HPASMC). HPASMC were commercially obtained and were transfected with miR17/92 or miR17 encoding plasmid or control vector by electroporation. Proliferation and apoptosis resistant state of PASMC transfected with miR was assessed by MTS and caspase3/7 Glo assays respectively. RNA and protein levels of important target genes in PAH were measured using Real-time RT-PCR and western blots. HPASMC transfected with miR17/92 or miR17 show decreased cell numbers and showed an increased apoptosis as assessed by increased caspase activity. Real-time RT-PCR analysis reveal that proliferation markers such as PCNA and Cyclin D1 are not significantly altered. SMC marker calponin and bone morphogenetic protein receptor-II levels were down regulated in both miR17/92 or mir17 transfected cells. Voltage gated potassium channel (Kv1.5) was up regulated in PASMCS treated with mir17/92 cluster but not miR17 alone indicating that different miRs in the 17/92 cluster differentially may regulate key molecules in the development of PAH.