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Lung-specific RNA interference of coupling factor 6, a novel peptide, attenuates pulmonary arterial hypertension in rats

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Background: Pulmonary Arterial Hypertension (PAH) is a progressive and life-threatening disease associated with high morbidity and mortality rates. However, the exact regulatory mechanism of PAH is unknown. Although mitochondrial Coupling Factor 6 (CF6) is known to function as a repressor, its role in PAH has not been explored.

Objective: We investigated the involvement of endogenous CF6 in the development of PAH.

Methods and Results: PAH was induced with Mono Cro Taline (MCT), as demonstrated by significant increases in pulmonary artery pressure and vessel wall thickness ($p<0.01$). CF6 upregulation was confirmed by quantitative RT-PCR and ELISA. Immunohistochemistry analysis revealed a large amount of CF6 localized to all three layers of the vascular wall and the surrounding tissues in the PAH rats; however, it was barely detectable in Endothelial Cells (ECs). A total of 2×1010 gp of Adeno-Associated Virus (AAV) was used for transduction of CF6 short hairpin RNA (shRNA) or a control vector, which was intratracheally transfected into the rats before or after MCT injection. The CF6 shRNA effectively reduced the CF6 mRNA and protein levels in the PAH rats and also reversing the 6-keto-PGF1a levels in circulation and in lung tissue ($p<0.05$). Finally, we found that CF6 shRNA reduced inflammatory infiltration, reversed endothelial dysfunction and vascular remodeling and ameliorated the severity of pulmonary hypertension and right ventricular dysfunction ($p<0.05$) at 4 weeks when it was used as both a pretreatment and rescue intervention.

Conclusions: CF6 contributes to the pathogenesis of PAH, and it may function in association with prostacyclin. The blockage of CF6 could reverse the progression of PAH; thus, it might be applied as a novel therapeutic approach.

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