

# 17<sup>th</sup> European Heart Disease and Heart Failure Congress &

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### Mechanisms underlying the development of pulmonary arterial hypertension in secretin gene deficiency

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Idiopathic (primary) pulmonary arterial hypertension (IPAH) is a disease with fatal prognosis, as seen by progressive right heart failure within three years after diagnosis. Extensive research in recent past has led to substantial advancement in our understanding of the genetic basis and pathophysiology of PAH resulting in better and more effective treatment strategies. However, very little is known about the pathological mechanisms underlying PAH and being a complex multi-factorial disease. Secretin (SCT), a brain-gut peptide hormone, has been found in the past to have pleiotropic functions including its important role in water homeostasis. We have found pathological features of PAH after targeted deletion of SCT gene and have explored the mechanisms underlying. Histopathological examinations and immunohistochemical staining of CD31 (endothelial cell specific marker protein) revealed development of pathological features of PAH in lungs of SCT knockout (SCT<sup>-/-</sup>) when compared to C57 mice such as thickened arterial wall, enlarged medial area and narrowed lumen. TUNEL staining and immuno fluorescent co-localization studies with CD31 revealed increased endothelial cell (EC) apoptosis in pulmonary arteries of SCT<sup>-/-</sup> mice. This is likely to be triggered by low levels of lung-vascular endothelial growth factor (VEGF) -transcripts measured by real-time PCR and -protein measured in bronchio alveolar fluid. Deletion of SCT gene leads to spontaneous development of moderate PAH in mice. It might be initiated by EC apoptosis in pulmonary arteries, possibly due to reduced VEGF levels, resulting in increased pulmonary pressure and pulmonary vascular remodeling. Although not an exact reflection of human idiopathic PAH, SCT<sup>-/-</sup> mice would be useful for obtaining mechanistic and therapeutic insights underlying PAH.

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### Antihypertensive and anti-hypertrophic effects of acupuncture at PC6 acupoints in spontaneously hypertensive rats and the underlying mechanisms

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Aim of this study is to investigate the effect of electro acupuncture (EA) at PC6 on the hypertension and myocardial hypertrophy in spontaneously hypertensive rats (SHRs). 30 SHRs were randomized into model, SHR+EA and SHR+sham EA group with WKY rats as normal control. EA was applied once a day in eight consecutive weeks. The blood pressure (BP), cardiac function and hypertrophy as well as the underlying mechanisms were investigated. After EA treatment, the enhanced BP in SHR+EA group was significantly lower than both before EA and model group. Echocardiographic, morphological studies showed that the enhanced anterior and posterior wall thickness (LVAWd and LVPWd), diameters and cross-sectional area (CSA) of cardiac myocyte, as well as the ratio of heart weight to body weight (HW/BW) were markedly diminished in SHR+EA group; while the reduced left ventricular ejection fraction, left ventricular short axis fraction shortening and E/A ratio were significantly ameliorated. The level of angiotensin-converting enzyme (ACE), angiotensin II type 1 and 2 receptors (AT1R, AT2R) in SHRs were also significantly attenuated by EA. The results suggested that EA at bilateral PC6 could arrest the hypertension development and ameliorate the cardiac hypertrophy and malfunction in SHRs, which might be mediated by the regulation of ACE, AT1R and AT2R.

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