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THE ST3GAL-IV GENE MEDIATES STRESS-DEPENDENT CARDIAC REMODELLING IN MICE

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Jianyong Qi, Minzhou Zhang. *Intensive Care Unit, Guangdong Province Hospital of Chinese Medicine*

Objectives To explore the role and mechanisms of ST3Gal-IV gene mediates stress-dependent cardiac remodelling in mice.

Methods By using transverse aortic constriction (TAC) model, we observed the phenotype of the ST3Gal-IV gene knock-out mice and wild type mice after TAC, dynamic analysed the changes of cardiac structure and function by ultracardiography (UCG), then mice were sacrificed and heart weight(HW), left ventricular weight (LVW) were evaluated. Consequently, we analysed action potential and calcium current on the cardiac surface by high-resolution optical mapping, furthermore, we analysed the protein expression and signalling transduction by western-blot and separating cytoplasm and nucleus protein methods.

Results ST3Gal-IV knockout mice were more susceptible to heart failure compared with wild-type mice. Voltage-dependent calcium

channels were dysfunction in hearts of ST3Gal-IV knockout mice, which resulted in calcium overload, enhanced the amplitude of action potential, increased protein expression of calcineurin-A (CnA), and translocated nuclear factor of activated T cells (NFATc3) into nucleus, so as to activate the hypertrophic genes expression, and led to heart failure.

Conclusions With the deficiency of ST3Gal-IV gene, heart failure was more susceptible to forming through calcium-calc signalling pathway. Therefore, ST3Gal-IV gene plays a vital role in cardiac ion channel function, which keeps normal cardiac systolic function.