visualized by enhanced chemiluminescence detection kit. β-actin was used as an internal control.

RESULTS Results showed that tanshinone IIA could protect H9c2 cells from H2O2-induced cell death concentration-dependently. MIR-133 was involved in tanshinone IIA action. Decreases of miR-133 expression induced by increasing concentration of H2O2 could be completely reversed by tanshinone IIA treatment. Inhibition of MIR-133 function by transfection of specific inhibitor abolished the cardioprotective effects of tanshinone IIA against H2O2-induced cell death. Furthermore, our results also showed that tanshinone IIA could activate Akt kinase by phosphorylation at serine 473. We furtherly determined the Bcl-2 protein levels by western blot and found that tanshinone IIA could completely reverse the decreases of Bcl-2 protein induced by H2O2; but the effect of tanshinone IIA on Bcl-2 protein in oxidative environment was suppressed by PI3K inhibitor, wortmannin, which indicated that tanshinone IIA exerted cardioprotective effects against H2O2-induced cell death through the activation of PI3K/Akt signal transduction pathway and consequent upregulation of Bcl-2 expression.

CONCLUSIONS The present study indicates that tanshinone IIA is a promising natural cardio-protective agent.

GW26-e4380 Screening and Differences for Glycolytic Metabolism Related Genes Between Two Ventricles in Monocrotaline Induced Pulmonary Arterial Hypertension Rats
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OBJECTIVES To investigate the differences of cardiac energy metabolism between left and right ventricle in the monocrotaline (MCT) induced pulmonary arterial hypertension (PAH) rats.

METHODS PAH was induced by a single subcutaneous injection of MCT (50 mg/kg) in rats, who developed right heart failure eventually. PAH rats were randomly divided into three groups according to the treatment of MCT: MCT-2week, 3week, 4week group (MCT-2w, 3w, 4w). At the end of study, the hemodynamics and right ventricular hypertrophy were compared in each group. The expression levels of proliferating cell nuclear antigen (PCNA) and TdT-mediated dUTP nick end labeling (TUNEL) were detected between left and right ventricular myocardial cells. The differences of genes expression from the glycolytic key candidate genes were screened between two ventricles.

RESULTS After MCT injection for three weeks, mean pulmonary arterial pressure, right ventricular systolic pressure and right ventricular hypertrophy index were significantly increased. The morphology and structure were changed obviously in left and right ventricular myocardial cells. Proliferation and apoptotic resistance of myocardial cells were markedly increased compared with control group. The expression of HK1 mRNA began to rise in right ventricle of MCT-3w group, which was obviously earlier than left ventricle (MCT-4w group). In the MCT-4w group, the expression of HK2 (hexokinase 2), PDHc (pyruvate dehydrogenase complex α), LDHA (lactate dehydrogenase A) mRNA was significantly increased in left ventricle, while the expression of LDHA only were up-regulated in right ventricle. The HK1 mRNA expression was further confirmed by the expression of HK1 protein and immunohistochemistry analysis.

CONCLUSIONS Energy metabolic shift occurred in the left and right ventricles in PAH. Up-regulated expression of HK1 appeared earlier in right ventricle than left ventricle. Interference of glycolysis of right ventricle may be a novel target of PAH in the future.

GW26-e4397 Effect of Rosuvastatin on Vascular Remodeling and Vascular Reaction of Spontaneously Hypertension Rats
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OBJECTIVES To observe the effect of rosuvastatin on remodel and vascular reaction of aorta from spontaneously hypertension rats (SHR).

METHODS SHR and Wister-Kyoto (WKY) rats were randomly divided into 1. WKY control group; SHR treated with rosuvastatin 6.25 mg/(kg/day); SHR treated with rosuvastatin 12.5 mg/(kg/day); rosuvastatin 25 mg/(kg/day); SHR control group. Therapy continued for 8 weeks. Tail systolic blood pressure was weekly measured. At the end of this study, aorta was moved rapidly. A part of aorta was gavaged with TMZ (20mg/kg) or Placebo for 8 weeks. Then, echocardiography and Pressure-Volume detection were used in the assessment of cardiac function.

RESULTS Compared with WKY, the systolic blood pressure was higher, and vessel lumen diameter (L) decreased, while media thickness (M) and M/L increased in SHR. The contractions to Phe, KCl in aorta were increased in SHR than in WKY. The relaxation to Ach decreased in SHR, but there was no difference of relaxation to SNP. Treatment with rosuvastatin induced a reduction in systolic blood pressure; increasing in L and decrease in M and M/L. Rosuvastatin reduced the contraction of aorta to Phe and KCl; increased the relaxation of aorta to Ach, but had no effect on the relaxation to SNP.

CONCLUSIONS The antihypertensive effect of rosuvastatin in SHR was accompanied by protect on endothelial function: retarding the remodeling of aorta; improving the vascular reaction to different vasomoters, resuming the balance of relaxation and contraction.

GW26-e4479 Nrf2 Is Crucially Required for Sulforaphane to Prevent High Fat Diet/Low Dose STZ Induced Diabetic Cardiomyopathy
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OBJECTIVES The present study aimed to define whether sulforaphane (SFN) protects from type 2 diabetic cardiomyopathy (DCM) entirely through Nrf2.

METHODS 8 weeks of Nrf2-knockout (KO) and wild-types (WT, C57BL/6J) mice were used to induce a type 2 diabetes (T2DM), by feeding high-fat diet (HFD) for 3 months to induce insulin resistance and then injecting one dose of streptozotocin (STZ, 100 mg/kg body-weight) to induce hyperglycemia. Age-matched control mice were fed a normal diet (ND) for the same period. Both T2DM and control mice were treated with or without SFN at 0.5 mg/kg five days a week for 4 months along with continual feeding with either HFD or ND diet, respectively. Cardiac oxidative damage, hypertrophy and fibrosis were assessed by western blot, real-time qPCR and histopathological examination.

RESULTS SFN prevents diabetes-induced cardiac oxidative damage (increased accumulation of 3-nitrotyrosine and 4-hydroxynonenal), hypertrophy (increased the ratio of heart weight toibia length, β-MHC and atrial natriuretic peptide mRNA expression and mean arterial pressure of systole) and fibrosis (increased the accumulation of collagen) along with increase in Nrf2 expressions in the WT mice. However, Nrf2-KO diabetic mice suffered from greater cardiac damage and had more severe pathological alterations in the heart. SFN cardiac protection was completely lost in Nrf2-KO diabetic mice.

CONCLUSIONS The present study demonstrated for the first time that Nrf2 entirely mediates SFN’s prevention of HFD / STZ-induced type 2 diabetic cardiomyopathy.

GW26-e4609 Trimezatidine Attenuates Cardiac Remodeling Via Suppression AKT Signaling Pathway
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OBJECTIVES Trimezatidine (TMZ) is widely used in the therapy of myocardial ischemia. Recent studies suggest that additional TMZ treatment may be able to improve the systolic-diastolic function in heart failure patients. But the effects and underlying mechanisms of TMZ in cardiac remodeling are still unclear. The purpose of this study is to estimate the effects of TMZ in cardiac remodeling and try to explore the underlying mechanisms.

METHODS In our study, C57BL/6 mice (male, 8 to 10 weeks, 23.5 - 27.5 g weight) were subjected to aortic banding (AB) operation or Sham as control. After operation the mice were gavaged with TMZ (20mg/kg) or Placebo for 8 weeks. Then, echocardiography and Pressure-Volume detection were used in the assessment of cardiac function.