immunoreactivity were increased in the DM group compared to the control group. The DM group showed marked myocardial disarray and increased fibrosis in spontaneous diabetic mice.

Results: P-smad2/3, CTGF were detected by western blot.

This study provided novel evidences that UII is involved in EndMT and cardiac fibrosis.

Conclusions: Whether or not the increased expression of Omi/HtrA2 causes cardiomyocyte apoptosis via release of cytochrome c. Relaxin is a peptide hormone with potent cardiovascular effects, which has been demonstrated to be safe and effective in acute heart failure in clinic trials. However, effect of relaxin on heart failure with preserved ejection fraction (HFpEF) is unknown. The aims of the study were to determine whether relaxin could improve the diastolic function of HFpEF and to investigate the underlying mechanisms.

Methods: In the present study, adenoviral vector expression relaxin-2 (Ad-RLN-2) and GFP (Ad-GFP) were constructed. Pressure overloadced rat model was established by abdominal aorta constriction (TAC) on SD rats, and 4 weeks later, echocardiography and cardiac hemodynamics were performed. These TAC rats were randomly assigned into 3 groups (TAC control group, without intramyocardial injection; TAC+Ad-RLN-2, with intramyocardial injection of Ad-RLN-2; TAC+Ad-GFP, with intramyocardial injection of Ad-GFP). And 12 days after intramyocardial injection, diastolic and systolic function were determined by echocardiography and cardiac hemodynamics. Then the rats were sacrificed, proteins and RNA were extracted from left ventricles; Western Blotting and quantitative real-time PCR were performed to prove that Neutural cardiomyocytes were isolated and were cultured with Ad-RLN-2 and Ad-GFP. And then laser confocal was used to detect the intracellular localization of phosphorylation Akt and PLB in neonatal cardiomyocytes.

Results: RLN-2 gene therapy was demonstrated to improve diastolic function by reducing diastolic and hystonic parameters. E/A ratio significantly increased in aging myocardial issue. Moreover, the myocardial collagen remodeling effectively. However, the mechanism of how relaxin prevent fibrosis in spontaneous diabetic mice is need to be further investigated. Whether the ion channel defect involved in the myopathy and arrhythmia. To date, the molecular pathogenesis underly this phenomenon is poorly understood. Whether the ion channel defect involved in the myopathy and arrhythmia. To date, the molecular pathogenesis underly this phenomenon is poorly understood. Whether the ion channel defect involved in the myopathy and arrhythmia. To date, the molecular pathogenesis underly this phenomenon is poorly understood.

Conclusions: These results demonstrate that increased expression of Omi/HtrA2 in aging cardiomyocytes promotes apoptosis via release of cytochrome c. Intramyocardial relaxin-2 gene delivery improves diastolic function of pressure-overloaded rats via increasing phospholamban phosphorylation by activating nuclear-targeted Akt.

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