RESULTS associated with the metabolism of ox-LDL were chosen for quantified by immuno-ELISA. Intracellular reactive oxygen species (ROS) generation was effectively decreased. Treatment of TT improved endothelial integrity of thoracic aorta, decreased arterial pressure and heart rate, and showed against weight gain effects. TT demonstrated excellent slimming benefits, anti-hypertension and endothelial protective effects. It also suggested that cardiac myocyte-fibroblast interaction plays a key role in diabetic myocardial fibrosis. Specifically, our study indicates myocyte HMGB1-fibroblast TLR4/IL-33 axis contributes to the development of myocardial fibrosis and dysfunction in mice with diabetes.

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GW26-e3847 Postinfarction Gene Therapy With Hepatocyte Growth Factor Mitigates Cardiac Remodeling and Dysfunction
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OBJECTIVES To investigated beneficial effects and its mechanisms of naked plasmid expressing recombinant human hepatocyte growth factor on left ventricular remodeling and dysfunction.

METHODS Acute myocardial infarction was induced male SD rats by ligation anterior descending of left coronary artery. These rats were randomly assigned to HGF group (n=8); a single myocardial injection of naked plasmid expressing HGF (250 ug/injection) immediately after left coronary artery ligation. Control group (n=8); myocardial injection of same dose naked plasmid without HGF, normal group (n=10); the suture was passed but not tied treated. After four or eight weeks, cardiac function was evaluated by echocardiography respectively, the cardiac specimens at eight-week time point were subjected to Masson staining and immunohistochemical analysis.

RESULTS Four weeks later, left ventricular remodeling and dysfunction were apparent, and LV anterior wall thickness (LVAWT) were significantly reduced (P<0.001) in the control group. However, left ventricular remodeling and dysfunction were still significantly improved (P<0.05). Furthermore, significant mitigation of LVAWT was seen in HGF-treated rats (P<0.005). Eight weeks later, the infarct size significantly reduced and the infarct wall was thinner in the HGF-treated rats (P<0.005). Myocardial fibrosis was significantly reduced and the density of blood capillary was significantly increased in the myocardial infarcted area in HGF group (P<0.001)

CONCLUSIONS Recombinant human hepatocyte growth factor improves postinfarction cardiac remodeling and dysfunction by reducing infarct size and myocardial fibrosis and increasing by density of blood capillary.

GW26-e3875 Diabetes Blunt the Compensatory Enhancement of SUMOylation Intensity of Sarcoplasmic Reticulum Calcium-transporting ATPase After Myocardial Infarction
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OBJECTIVES Diabetes is an independent risk factor of heart failure and mortality after myocardial infarction(MI). The activity and
expression of Sarcomplasmic Reticulum Calcium-transporting ATPase (SERCA2a) decrease in diabetes, leading to diastolic and systolic dysfunction of myocardium. It was recently reported that SUMOylation could elevate the activity and stability of SERCA2a. We assume that diabetes might affect the intensity of SUMOylation of SERCA2a after MI.

METHODS Diet-induced type 2 diabetic rats and controls were divided into seven groups: sham or one of six experimental groups: I/R+Rapamycin (RAPA) group, I/R+DCPIB group, I/R+DCPIB+RAPA group, I/R+3MA (1mg/kg) group, I/R+DCPIB+3MA group, and I/R+RAPA group. Rats were anaesthetized and killed 24 hours after reperfusion. Primary cardiomyocytes were isolated from rat hearts. Myocardial ACE2, eNOS, and SUMO1 were determined by Western blotting. Na+-K+-ATPase activity was determined as the rate of inorganic phosphate released in the presence or absence of ouabain.

RESULTS The D4 receptor agonist, PD168077, decreased AT1 receptor expression in rat renal proximal tubule cells. The D4 receptor antagonist, PD168077, increased Na+-K+-ATPase activity in WKY cells. However, in SHR cells, the inhibitory effect of D4 receptor on AT1 receptor expression and function in renal proximal tubules (RPTs) cells from Wistar-Kyoto (WKY) rats, the regulation of D4 receptor on AT receptor expression in spontaneously hypertensive rats (SHRs).

METHODS Sprague-Dawley rats were randomly divided into sham operation group, I/R group, and I/R+Rapamycin (RAPA) group, I/R+DCPIB group, I/R+3-methyladenine (3MA, a autophagy inhibitor) group and I/R+RAPA+DCPIB group, with 6 rats in each group. Rats were perfused with heart perfusion solution 30 minutes and subsequent reperfusion 24 hours. DCPIB (10nM/kg), RAPA(4mg/kg) and 3MA (1mg/kg) were administered as intraperitoneal injection 10 min before the onset of reperfusion. The intensity of SUMOylation of SERCA2a was measured by Western blotting. Na+-K+-ATPase activity was determined as the rate of inorganic phosphate released in the presence or absence of ouabain.

RESULTS The D4 receptor agonist, PD168077, decreased AT1 receptor expression in rat renal proximal tubule cells. The D4 receptor antagonist, PD168077, increased Na+-K+-ATPase activity in WKY cells. However, in SHR cells, the inhibitory effect of D4 receptor on AT receptor expression and function in renal proximal tubules (RPTs) cells from Wistar-Kyoto (WKY) rats, the regulation of D4 receptor on AT receptor expression in spontaneously hypertensive rats (SHRs).