β-platelet globulin (β-TG), fibrinogen(FIB) and cyclic adenosine monophosphate (cAMP) contents in the plasma were detected by Enzyme-Linked Immunosorbent Assay.

RESULTS Echocardiography showed left ventricular ejection fraction (EF) and fractional shortening (FS) decreased significantly in saline group (EF: 41.46±3.26, FS: 20.72±1.79), QSYQ can significantly increase the EF and FS of the I/R injured rats (EF: 59.73±3.91, FS: 29.66±1.20). In addition, compared with saline group, QSYQ significantly increase the LVSP (saline group vs QSYQ group: 89.40±4.44 vs 95.75 ±4.62), and ±dp/dt max (saline group vs QSYQ group: 4234.84±392.60 vs 5374.28±419.84; -3911.16±165.95 vs -4284.72±299.40) of the I/R injure rats; HE staining results also showed that QSYQ can reduce myocardial inflammatory infiltration of I/R injure rats and protect myocardial tissue. Moreover, the level of plasma TXB2 (15.14±2.21), PF4 (1928.12±166.72), E-selectin (35.22±3.57), β-TG (42.32±6.64) and FIB (2120.27±312.89) increased in saline group, compared to Sham group. Pretreatment with QSYQ attenuated I/R-induced increase in TXB2, PF4, E-selectin, β-TG and FIB (10.58±0.54, 606.23±126.20, 23.64±4.69, 27.39±6.68 and 1660.98±208.15 respectively). The plasma 6-Keto-PGF1α (0.75±0.03), cAMP (33.05±8.13) and the ratio of 6-Keto-PGF1α/TXB2 (60.85±19.23) in saline group was lower than that in the sham group, all of which were significantly ameliorated by pre-treatment with QSYQ (0.91±0.10, 47.62±1.14 and 85.65±15.25 respectively $P<0.05$). These results suggest that Qishenyiqi pills can suppress ischemia-reperfusion injury induced platelet aggregation in rats with I/R injury, inhibit the inflammatory response and improve cardiac function.

CONCLUSIONS The results of the present study suggest that QSYQ protects against myocardial I/R injury via antiplatelet aggregation and activation action. Further, we provided evidence to speculate that the regulation of cAMP level, prostaglandins synthesis, 6KFG/TXB2 and TXB2 synthesis may be involved in the therapeutic action of QSYQ.

GW27-e0722

Autophagy Modulates High glucose induced Cardiac Microvascular Endothelial Cells apoptosis by mTOR signal pathway

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OBJECTIVES Cardiac microvascular endothelial cells (CMECs) dysfunction is an important pathophysiological event in the cardiovascular complications induced by diabetes. However, the underlying mechanism is not fully clarified. Autophagy is involved in programmed cell death. Here we investigated the potential role of autophagy on the CMECs injury induced by high glucose.

METHODS CMECs were cultured in normal or high glucose medium for 6h, 12h and 24h respectively. The autophagy of CMECs was measured by green fluorescence protein (GFP)-LC3 plasmid transfection. Moreover, the apoptosis of CMEC was determined by flow cytometry. Furthermore, 3-Methyladenine (3MA) and rapamycin were administrated to regulate the autophagy state. Moreover, Western blotting assay was performed to measure the expressions of Akt, mTOR, LC3 and p62. n important pathophysiological event in the cardiovascular complications induced by diabetes. However, the underlying mechanism is not fully clarified. Autophagy is involved in programmed cell death. Here we investigated the potential role of autophagy on the CMECs injury induced by high glucose.

RESULTS High glucose stress decreased the autophagy, whereas increased the apoptosis in CMECs time dependently. Meanwhile, high glucose stress activated the Akt/mTOR signal pathway. Furthermore, autophagy inhibitor, 3-MA, impaired the autoghagy and increased the apoptosis in CMECs induced by high glucose stress. Conversely, rapamycin up-regulated the autophagy and decreased the apoptosis in CMECs under high glucose condition. nd p62. n important pathophysiological event in the cardiovascular complications induced by diabetes. However, the underlying mechanism is not fully clarified. Autophagy is involved in programmed cell death. Here we investigated the potential role of autophagy on the CMECs injury induced by high glucose.

CONCLUSIONS Our data provide evidence that high glucose directly inhibites autophagy, as a beneficial adaptive response to protect CMECs against apoptosis. Furthermore, the autophagy was mediated, at least in part, by mTOR signaling.

GW27-e0747

Overexpression of Mcoln3 in the kidney aggratated salt sensitive hypertension in Dahl S rats

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OBJECTIVES Renal dysfunctional metabolism of sodium play a key role in the formation of salt sensitivity. The renal Mcoln3 genes of Dahl salt sensitive rat were screened using Genechip method in our previous study, which was over-expressed in kidney. We hypothesized that Mcoln3 may be the trigger point of renal dysfunctional sodium metabolism. The present study detemined whether the impairment of the renal Mcoln3 pathway was responsible for salt sensitive hypertension in Dahl S rats.

METHODS Male Dahl sensitive rats and 13 BN -SS rats were randomly divided into nornal salt group, high salt group and high salt plus Mcoln3 inhibition Gadolinium group respectively; After 6-week diet intervention, Mcoln3, SGK1 - ENaC, renin, angiotensinogen mRNA and protein expression level were determined.

RESULTS The renal expression of Mcoln3 was higher in Dahl salt sensitive rat. Dietary high salt could increase the expression of SGK1 and alpha ENaC in Dahl salt sensitive rats. However, when given Mcoln3 inhibition Gadolinium, SGK1 - ENaC mRNA and protein expression level also significantly lowered at the same time, but the RAAS system has no obvious change.

CONCLUSIONS Mcoln3 maybe play a role of regulating blood pressure by regulating kidney SGK1 - ENaC pathways. We believe that it would been of great significance to uncover the etiology of salt sensitive hypertension as well as to seek another drug targets for treatment of essential hypertension.

GW27-e0778

Combined Retrograde Coronary Venous Infusion of Fibroblast Growth Factor 2 and Bone Marrow Mesenchymal Stem Cells Restores Cardiac Function After Myocardial Infarction in Dogs

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OBJECTIVES Retrograde coronary venous infusion provides both increased regional fibroblast growth factor 2 (FGF-2) concentrations and homogeneous dissemination of bone marrow mesenchymal stem cells (MSCs) across the myocardium. We determined the effects of retrograde delivery of combined FGF-2 and MSCs on cardiac function in a canine infarct model.

METHODS Adult male mongrel dogs were subjected to myocardial infarction (MI) via ligation of left anterior descending coronary artery. After one week, all surviving animals underwent retrograde infusion of 10 mL of one of the following: combination FGF-2 (200 ng/mL) and MSCs (1×108 cells), MSCs alone (1×108 cells), FGF-2 alone (200ng/mL), or placebo (phosphate-buffered saline). Transthoracic echocardiography was performed at baseline, 1 week after MI (before infusion), and 4 weeks after infusion (just prior to euthanasia). After echocardiographic study, animals were euthanized, and the hearts were excised for histologic analysis.

RESULTS 18 animals (saline [n=3], FGF-2 [n=5], MSCs [n=5], and FGF-2+MSCs [n=5]) were included in the analysis. Four weeks after infusion, left ventricular ejection fraction (LVEF) was improved in the FGF-2+MSCs group by 11% ($P<0.01$). The treatment effect (delta LVEF) was significantly greater in both the FGF-2+MSCs group (7.43±1.51% versus -10.07±2.94%; $P<0.001$) and MSCs group (4.79±2.47% versus -10.07±2.94%; $P<0.01$) compared to saline, to greater degree in the combination group. Morphologic analysis revealed an increased infarct wall thickness in the FGF-2+MSCs group compared to all others ($P<0.05$), accompanied by increased vascular density and reduced apoptosis.

CONCLUSIONS Retrograde coronary venous infusion of combination of FGF-2 and MSCs restored cardiac function after MI. This novel combined treatment and delivery method is a promising strategy for cardiac repair after ischemic injury.