by quantitative real-time polymerase chain reaction (PCR) and enzyme-linked immunosorbent assay (ELISA). Macrophage polarization and its mechanism were evaluated by flow cytometry and western blot. Additionally, toll-like receptor 4 (TLR4) small interfering RNA and mitogen-activated protein kinase (MAPK) inhibitors were used to further confirm the molecular mechanism of curcumin on macrophage polarization.

RESULTS Curcumin dose-dependently inhibited M1 macrophage polarization and the production of TNF-α, IL-6, and IL-12. It also decreased TLR4 expression, which regulates M1 macrophage polarization. Furthermore, curcumin significantly inhibited the phosphorylation of ERK, JNK, p38, and nuclear factor (NF)-κB. In contrast, siTLR4 in combination with p-JNK, p-ERK, and p-p38 inhibition reduced the effect of curcumin on polarization.

CONCLUSIONS Curcumin can modulate macrophage polarization through TLR4-MAPK/NF-κB pathway inhibition, indicating that its effect on macrophage polarization is related to its anti-inflammatory and atheroprotective effects. Our data suggest that curcumin could be used as a therapeutic agent in atherosclerosis.

GW26-e0056

Effects of Nicacin on ApoE−/− Mice’ Adipose Tissue, Serum Lipid and Atherosclerosis
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OBJECTIVES To investigate the effects of Nicacin on ApoE−/− mice’ adipose tissue, serum lipid and atherosclerosis, and furthermore to explore the potential relation among them.

METHODS Total 28 male ApoE−/− mice were randomly divided into 3 groups: control group (n=8), model group (n=10), Nicacin treatment group (n=10), respectively fed with normal diet, high cholesterol diet, high cholesterol diet + 1% w/w Nicacin. After 14 weeks, serum lipid level (total triglyceride, total cholesterol, HDL cholesterol, LDL cholesterol and apolipoprotein-A-I) was measured by enzymatic method and by immunoturbidimetry. Lesions of aortic arteries were stained with hematoxylin eosin. The content of cholesterol in arterial wall and subcutaneous adipose tissue in groin was quantitated by high performance liquid chromatography. The expression of LXRα, ABCA1 and ABCG1 mRNA in subcutaneous adipose tissue was determined by RT-PCR.

RESULTS The content of cholesterol in subcutaneous adipose tissue in control group, model group, Nicacin treatment group was (3.13 ± 0.19), (20.81 ± 1.97) and (4.00 ± 0.81) mg/g, respectively. Compared with model group, Nicacin treatment could increase the expression of LXRα, ABCA1 and ABCG1 mRNA 144%, 47.3% and 73.8%, respectively. And furthermore, it could also downregulate the level of serum total triglyceride, total cholesterol and LDL cholesterol, upregulate the level of serum HDL cholesterol, apolipoprotein-A-I. Pearson correlation analysis showed that there was a positive relation between the content of cholesterol in arterial wall and the ratio of intima/media thickness (r=0.58, P<0.05). In addition to those above, Nicacin treatment could thin the intima thickness, decrease the ratio of intima/media thickness and downregulate the sub-endothelium lipid-accumulation, especially cholesterol -accumulation.

CONCLUSIONS Nicacin treatment may promote the reverse cholesterol transport of peri-adipose tissue, bring the changes of serum lipid and its precursor, but the roles and mechanisms of Nicacin on the end-organ damage remains unclear so far. Recently, some findings support the (pro)renin-(P)RR interaction at exceptionally high (pro)renin levels in vitro. However, the conflicting results obtained with handle region peptide (HRP) in vivo and in vitro argue against the idea that this drug truly blocks the (pro)renin-(P)RR interaction in the intact animals. In this study, we investigated the roles of cardiac (P)RR and its downward signals on cardiac hypertrophy in hypertensive rats with abdominal aortic constriction, following treatment of HRP and PLC-b3 inhibitor, U73122.

RESULTS The levels of (P)RR and PLC-b3 significantly increased in the left ventricle in hypertensive rats (P<0.01, respectively). The surface area of cardiomyocytes and MAP rose markedly (P<0.01). HRP significantly decreased the level of (P)RR and U73122 suppressed PLC-b3 at the protein level. Treatment of both HRP and U73122 significantly decreased levels of PKCα, ERK1/2 and Raf-1 in the heart (P<0.001). Meanwhile, the surface area of cardiomyocytes and MAP were decreased after treatment (P<0.01).

CONCLUSIONS This study demonstrates that (P)RR inhibitor, HRP and PLC-b3 inhibitor, U73122 decreased levels of (P)RR, PLC-b3, PKC-α, ERK1/2 and Raf-1 in the heart, respectively. Meanwhile, administration of both reagents lowered the surface area of cardiomyocytes and MAP. These findings indicate that cardiac (P)RR may activate PKC-β3, PKC, ERK1/2 and Raf-1 signals and lead to hypertension and cardiac hypertrophy.

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GW26-e0106

Nestin-1β3 Integrin Promotes Cell Motility and Proliferation in Human Carotid Endarterectomy Specimens
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OBJECTIVES To invesigate the role of integrins in human carotid endarterectomy specimens.

METHODS NESTIN-1β3 integrin proteins were detected using immunohistochemistry and western blotting in human carotid endarterectomy specimens.

RESULTS NESTIN-1β3 integrin was highly expressed in human carotid endarterectomy specimens.

CONCLUSIONS NESTIN-1β3 integrin plays a key role in human carotid endarterectomy specimens.

GW26-e0058

Effect of Niacin on Cardiac Sarcoplasmic Reticulum Calcium Pump Activity in Rats
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OBJECTIVES Discovery of (pro)renin receptor (P)RR uncovered a novel function of (pro)renin as a receptor ligand in addition to enzyme and its precursor, but the roles and mechanisms of (P)RR on the end-organ damage remains unclear so far. Recently, some findings support the (pro)renin-(P)RR interaction at exceptionally high (pro)renin levels in vitro. However, the conflicting results obtained with handle region peptide (HRP) in vivo and in vitro argue against the idea that this drug truly blocks the (pro)renin-(P)RR interaction in the intact animals. In this study, we investigated the roles of cardiac (P)RR and its downward signals on cardiac hypertrophy in hypertensive rats with abdominal aortic constriction, following treatment of HRP and PLC-b3 inhibitor, U73122.

RESULTS The levels of (P)RR and PLC-b3 significantly increased in the left ventricle in hypertensive rats (P<0.01, respectively). The surface area of cardiomyocytes and MAP rose markedly (P<0.01). HRP significantly decreased the level of (P)RR and U73122 suppressed PLC-b3 at the protein level. Treatment of both HRP and U73122 significantly decreased levels of PKCα, ERK1/2 and Raf-1 in the heart (P<0.001). Meanwhile, the surface area of cardiomyocytes and MAP were decreased after treatment (P<0.01).

CONCLUSIONS This study demonstrates that (P)RR inhibitor, HRP and PLC-b3 inhibitor, U73122 decreased levels of (P)RR, PLC-b3, PKCα, ERK1/2 and Raf-1 in the heart, respectively. Meanwhile, administration of both reagents lowered the surface area of cardiomyocytes and MAP. These findings indicate that cardiac (P)RR may activate PKC-β3, PKC, ERK1/2 and Raf-1 signals and lead to hypertension and cardiac hypertrophy.

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GW26-e0107

Protective Effects and Mechanism of Penecyclidine Hydrochloride on Myocardial Ischemia Reperfusion Injury in Rats
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OBJECTIVES To investigate the effects and mechanisms of penecyclidine hydrochloride (PHC) on myocardial ischemia reperfusion injury(IRI).

METHODS A rat model of myocardial IRI was established by the ligation of left anterior descending coronary artery for 30 min followed by 3 h perfusion. The rats were randomly divided into 6 groups (n=18 in each group): a) Sham group: the rats underwent sham cavity injection. The MIRI animal model was established by blocking the left anterior descending artery for 45min followed by 4h of reperfusion. Real-time PCR and western blotting were used to detect renalase expression in the heart tissue. Double staining and TUNEL were used to detect the necrosis and apoptosis in the myocardial cells, respectively.

RESULTS All rats subjected to MIRI exhibited lower levels of renalase in the heart tissue than did the sham-operated group (P<0.05, n=6). The (RNAi) group rats exhibited lower renalase levels than did the controls and also exhibited more serious necrosis (7.12±0.56% vs. 3.32±0.93%, P<0.05, n=6) and apoptosis (15.2±8.2% vs. 6.6±6.8%, P<0.05, n=6); however, pretreatment with the recombinant renalase significantly reduced myocardial cell necrosis (1.51±0.12% vs. 4.13±0.02%, P<0.05, n=6) and apoptosis (21.3±5.0 vs. 52.6±10.4, P<0.05, n=6) relative to the control rats.

CONCLUSIONS Exogenous recombinant renalase protein reduced myocardial cell necrosis and apoptosis. Recombinant renalase protein might be a new cardiovascular drug for ischemia/IR injury.