CONCLUSIONS IFNγ may amplify the cascade of PKC signaling pathways and upregulate the expression of ACAT-1, and then promote the uptake, synthesis and esterification of cholesterol in RAW264.7 macrophages loaded or unloaded oxLDL.

GW26-e0225 Knock Down of PRKCI by siRNA Promoted Low Density Lipoprotein Production in Rat Myocardial Cells In Vitro Lin Fan1, Weiying Liu1, Yushuang Yang1, Dongna Liu1, Fanbo Meng1
1Department of Cardiology, Jilin University Affiliated Hospital of Sun Yat-sen University, Changchun, China
2Laboratory Animal Center of Jilin University, Changchun, China

OBJECTIVES Our previous gene chip study in patients with coronary heart disease suggested that protein kinase C iota (PRKCI) gene had low expression in the peripheral blood cells, which was contrary to previous notion that PRKCI promotes lipogenesis. We thought that PRKCI gene may affect the process of the occurrence of coronary heart disease. The aim of this study is to examine the function of PRKCI in lipid metabolism in rat myocardial cells (H9C2).

METHODS RNA interference (RNAi) vector targeting PRKCI and GFP empty vector were transfected into H9C2 to specifically knockdown the expression of PRKCI, GFP empty vector was used as the negative control. Culture medium was collected before transfection, 12h, 24h, 48h, 72h after transfection. The expression of green fluorescence protein was monitored under an inverted fluorescence microscope after transfected of 24h. The mRNA and protein levels, the low density lipoprotein (LDL) content in the supernatant of H9C2 cells were analyzed by spectrophotometry, RT-PCR, Western blot, and ELISA after 24h, 48h, respectively before and after siRNA transfection.

RESULTS The result of green fluorescence showed that the vectors were transfected into H9C2 successfully, and had no difference in transfection efficiency. The results of real-time RT-PCR using 

GW26-e2165 Gender-Related Differences in Patients with Acute Aortic Dissection From Xin Jiang Buamina Maitusong, Maisumu Mahemuti, Yitong Ma
Department of Cardiology, First Affiliated Hospital of Xinjiang Medical University, Urumqi, P.R. China

OBJECTIVES To investigate gender-related differences in incidence, clinical manifestation, and outcomes in patients with aortic dissection (AD).

METHODS Retrospective analysis of 400 patients with acute aortic dissection, receiving conservative medical treatment or aortic surgery at the First Affiliated Hospital of Xinjiang Medical University of, from January 2008 to May 2012, which 304 cases of male and 96 cases of female. Divided into two groups of men and women, and clinical characteristics and treatment outcomes of the two groups of patients was respectively analyzed.

RESULTS 304 cases of male and 96 cases of female respectively accounted for 76% and 24%. Male to female ratio of 3.18:1. Although less frequently affected by AAD, women were significantly older and had more often (female 54.21±12.38, male 49.62±12.63, P=0.0019). Male group of smoking is higher than the female group, (55.9%:44.17%, P=0.04), and especially in female. Male with Diabetes, cardio-vascular disease, Marfan syndrome, bicuspid aortic valve, aortic aneurysm, heart failure, surgery history included coronary PCI surgery, valve replacement surgery, coronary bypass surgery, and other factors etiology was not statistical difference between two groups. Clinical symptoms of chest pain, back pain, radiating pain, abdominal pain, bloating, vomiting, hemoptysis, blood in the stool was not statistical difference between two groups of patients. Sudden pain in the proportion of female common than male patient (96.88%:87.83%, P=0.02). The symptoms of irritability more common (17.43%:6.25%, P=0.01). The symptoms of bilateral chest more common (12.38, male 49.62). The pleural effusion Men more common than female group, (55.9%:44.17%, P=0.04). The symptoms of Drowsiness, coma and other psychiatric symptoms There was no statistical difference between two groups. The new Q waves or ST-segment elevation of ECG showed male is more common. (6.91:3.13, P=0.03). The unusual effusion Men more common than women (11.8%:3.3%, P=0.009). The aortic shadow widened, the widened mediastinum atelectasis, pneumonia and other chest X-ray findings of the two groups was no significant difference. Celiac trunk dissection and superior mesenteric artery dissection are more common than women (26.32%:11.46%, P=0.003; 17.11%:7.29%, P=0.028). Aortic intramural hematoma common in female (10.86%:21.88%, P=0.009). The cumulative coronary artery, aortic arch, the left and right renal artery, the internal and external iliac artery that affected by Aortic dissection in no significant difference between two groups. Type A dissection in women was associated with a higher surgical mortality of 37.08% versus 19% in men.
CONCLUSIONS The age of onset of aortic dissection of female patients older than men, sudden chest pain symptoms women is obvious than men, while symptoms of irritable male more common. Aortic intramural hematoma is more common in women. Suffering from acute type AAD women patients have higher operative mortality.

GW26-e4000 Kv4.3 Expression Improves Cardiac Contraction Without Inhibition of Relaxation in Heart Failure
Yangang Wang
Zhongnan Hospital of Wuhan University

OBJECTIVES To test whether expression of Kv4.3 in HF ventricular myocytes would improve cardiac contraction without affecting relaxation in HF by the addition of the function of Kv4.3+-transient with a reduction in diastolic Ca2+ leak, and a recovery of frequency-dependent acceleration of relaxation (FADAR), an intrinsic mechanism allowing faster ventricular relaxation and diastolic filling at fast heart rates. In contrast to KN93, a pharmacological CaMKII inhibitor, Kv4.3 expression did not affect myofilament sensitivity to Ca2+, assessed by measuring the gradient of cell length-lfur2 trajectory during contraction and late relaxation.

RESULTS HF ventricular myocytes with Kv4.3 expression presented a significant increase in fractional shortening and Ca2+ transient with a reduction in diastolic SR Ca2+ leak, and a recovery of frequency-dependent acceleration of relaxation (FADAR), an intrinsic mechanism allowing faster ventricular relaxation and diastolic filling at fast heart rates. In contrast to KN93, a pharmacological CaMKII inhibitor, Kv4.3 expression did not affect myofilament sensitivity to Ca2+, assessed by measuring the gradient of cell length-lfur2 trajectory during contraction and late relaxation. In vivo study showed that Kv4.3 expression increased EF from 45±1% in HF mice transfected with Ad-Kv4.3 (n=11) to 73±2% in mice transfected with Ad-Kv4.3 (n=10, p<0.05), while the E/E' ratio was unchanged (39±2 vs. 35±2, p<0.05).

CONCLUSIONS Our results suggest that Kv4.3 expression improves myocardial contraction without detrimental effect on cardiac relaxation. Instead, it recovers FADAR.

GW26-e1814 Role of Monocyte/Macrophage in TRPV1 Ablation-Induced Renal Injury in Salt-Sensitive Hypertension
Yingying Wang, Mingjun Zhu, Lei Lei Fan, Miaomiao Fan, Lin Cui, Xiaojing Wang, Shiyang Xie, Si Shen
Central Laboratory and Division of Cardiology, First Affiliated Hospital, Henan University of Traditional Chinese Medicine, Zhengzhou, 450000, China

OBJECTIVES Our studies show that deletion of the transient receptor potential vanilloid type 1 (TRPV1) gene aggravates deoxycorticosterone- one acetate (DOCA)-salt hypertension-induced renal injury, which is associated with increased renal monocyte chemoattractant protein-1 (MCP-1) production and monocyte/macrophage infiltration. The results suggest that TRPV1 ablation-induced aggravation of renal injury in DOCA-salt hypertension may be the result of enhanced renal monocyte/macrophage infiltration that is dependent of the MCP-1/C-C chemokine receptor 2 (CCR2) signaling pathway. Therefore, we hypothesized that MCP-1/CCR2-mediated monocyte/macrophage infiltration is a critical determinant of TRPV1 ablation-induced renal injury in salt-sensitive hypertension.

METHODS We induced salt-sensitive hypertension for 4 weeks by uninephrectomy and DOCA-salt in wild type (WT) and TRPV1-null (TRPV1-/-) mice with or without RS504393, a selective CCR2 antagonist.

RESULTS DOCA-salt treatment increased systolic blood pressure (SBP) to the same degree in both strains, but increased urinary excretion of albumin and 8-isoprostane and decreased creatinine clearance with greater magnitude in TRPV1-/- mice compared to WT mice (p<0.05). DOCA-salt treatment also caused renal glomerulosclerosis, tubulointerstitial injury, collagen deposition, monocyte/macrophage infiltration, proinflammatory cytokine and chemokine production, and NF-kB activation in greater degree in TRPV1-/- mice compared to WT mice (glomerulosclerosis index: 0.78±0.15 vs. 0.35±0.14; tubulointerstitial injury score: 3.37±1.0 vs. 2.01±0.49; collagen content: 21.8±2.3 vs. 13.8±2.4 μg/mg dry tissue; monocyte/macrophage infiltration: 74±4 vs. 42±5 cells/mm2; TNF-α: 1.0±0.22 vs. 0.76±0.21 pg/mg protein; MCP-1: 1.0±0.22 vs. 0.76±0.21 pg/mg protein; p65-NF-κB protein; 54.15 ± 36.3 ng/mg protein, P<0.05). Blockade of the CCR2 with RS504393 (4 mg/kg) had no effect on SBP in DOCA-salt-treated WT or TRPV1-/- mice compared to their respective controls. However, treatment with RS504393 ameliorated renal dysfunction and morphological damage, and prevented the increase in monocyte/macrophage infiltration, cytokine/chemokine production, and NF-kB activity in both DOCA-salt hypertensive strains with a greater effect in DOCA-salt-treated TRPV1-/- compared to DOCA-salt-treated WT mice.

CONCLUSIONS Our data showed that blockade of CCR2 with RS504393 attenuated DOCA-salt hypertension-induced renal injury in WT and TRPV1-/- mice independently of their effects on blood pressure. The protective effect was greater in TRPV1-/- mice compared to WT mice. The results suggest that deletion of TRPV1 aggravated salt-sensitive hypertension-induced renal damage possibly via enhancement of the MCP-1/CCR2-mediated monocyte/macrophage infiltration. [This work was supported by a grant from the National Natural Science Foundation of China (No. 81170243)].

GW26-e0197 Exploring the Active Ingredients in Chinese Yellow Wine Which Could Inhibit the Progress of Atherosclerosis in LDLR Knockout Mice
Liping Meng,1,2 Xiaoya Zhai,1 Chenjian Jiang,1 Zheng Ji,1 Yan Guo,1 Jufang Chi,2 Hangyuan Guo1
1Datong People’s Hospital; The First Clinical Medical College of Wenzhou Medical University

OBJECTIVES To explore the active ingredients in Chinese yellow wine which could inhibit the progress of atherosclerosis in LDLR knockout mice.