RESULTS NVAF patients with thrombosis showed significantly higher sP-selectin (p < 0.001) than did NVAF patients without thrombosis. Allele frequencies of (-2123C/G, -1817T/C) were significantly higher in NVAF patients with thrombosis than in NVAF patients without thrombosis, but Thr715Pro did not show any polymorphism.

CONCLUSIONS PS gene polymorphism may contribute to thrombotic risk in AF. sP-selectin was a potential risk factor for thrombosis in AF.

GW26-e4504
Sympathetic Nerve Remodeling in Hypertensive Rabbits with Left Ventricular Hypertrophy and Heart Failure
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OBJECTIVES Sympathetic nerve (SN) remodeling plays an important role in the development of sudden cardiac death in patients with myocardial infarction. However, little has been known about the pattern of SN remodeling in patients with hypertensive left ventricular hypertrophy (LVH) or heart failure (HF). We aimed to evaluate the role and pattern of SN remodeling in hypertensive rabbit models.

METHODS Hypertensive LVH (70% diameter stenosis, n = 16) and HF (90% diameter stenosis, n = 12) rabbit models were prepared through abdominal aorta constriction, and confirmed with echocardiographic and hemodynamic measurements. The rabbits in Sham group (n = 14) received the same procedure without abdominal aorta constriction. Eight weeks after the index procedure, ex vivo electrophysiological parameters were determined. The distribution patterns and densities of myocardial SN were determined with immunohistochemistry. The myocardial levels of CAMP and norepinephrine were measured using radioimmunoassay and high-performance liquid chromatography, respectively. Finally, the protein expression level of beta-ARK1 was determined with western-blots.

RESULTS Compared to Sham group, LVH group had a significantly shorter left ventricular effective refractory period (122.75 ± 10.31 ms vs 95.71 ± 16.31 ms, P < 0.001). Six isolated hearts (6/8) in LVH group occurred inducible ventricular tachycardia or ventricular fibrillation under program stimulation, while none (0/8) in Sham group occurred (P < 0.007). All hearts (6/6) in HF group occurred spontaneous ventricular fibrillation, while the other two groups didn’t have. Compared to Sham group, LVH and HF groups had significantly lower average SN density (7972.08 ± 3238.78 μm²/mm² vs 3226.44 ± 2835.58 μm²/mm² vs 1498.85 ± 1752.33 μm²/mm², P < 0.001), but higher heterogeneity of SN distribution (4144.17 ± 2017.69 μm²/mm² vs 5768.22 ± 4582.05μm²/mm² vs 7858.17 ± 2965.67μm²/mm², P < 0.001). The myocardial levels of cAMP (7.94 ± 6.37 ng/g vs 20.60 ± 7.53 ng/g, P < 0.001) and norepinephrine were significantly lower in the failing hearts than did those in Sham group. Meanwhile, the LVH had comparable levels of CAMP and norepinephrine as compared with Sham group. In addition, HF group rather than LVH group showed significantly lower levels of beta-ARK1 expression compared to Sham group (P < 0.001).

CONCLUSIONS The present study suggests that both hypertensive LVH and HF would increase the heterogeneity of myocardial SN distributions with decreased overall SN activities, which could be translated into more vulnerable electrophysiological characteristics.

GW26-e4558
Microarray expression profile of long non-coding RNAs in human endothelial cells exposed to atheroprotective shear stress
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OBJECTIVES To identify differentially expressed long non-coding RNAs (lncRNAs) involved in flow-dependent regulation of vascular function.

GW26-e4592
Implication of C1q/TNF-related protein-12(CTRP-12) in patients with coronary artery disease
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OBJECTIVES C1q/TNF-related protein-12 (CTRPI-12), an adiponectin paralog, was recently identified as novel adipokines with metabolic regulatory properties. We analyzed the relationship between CTRP-12 and coronary artery disease (CAD).

METHODS Subjects (n = 188, 67:10 years, 79% male) suspected of having CAD were enrolled in the study and were divided into two groups, CAD and non-CAD subjects, according to the results of their coronary angiographies. Serum CTRP-12 levels of the subjects were measured by an enzyme-linked immunosorbent assay.

RESULTS Subjects with CAD had significantly lower circulating CTRP-12 concentrations compared to the non-CAD subjects (median [interquartile range]: 46.5 [26.6] vs. 53.5 [29.6] ng/mL, respectively; P = 0.01). The serum CTRP-12 levels significantly correlated with body mass index, the homeostasis model of assessment of insulin resistance, adiponectin levels, and CAD severity. Multivariate logistic regression analysis revealed that a decreased serum CTRP-12 level (log transformed) was independently associated with CAD for all subjects.

CONCLUSIONS Serum CTRP-12 levels are significantly associated with CAD in humans, suggesting that low CTRP-12 levels may contribute to CAD.

GW26-e4726
Sestrin isoform expression in the normal and failing hearts
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OBJECTIVES Oxidative stress and AMP-dependent protein kinase (AMPK)/mammalian target of rapamycin (mTOR) signaling pathway were closely involved in the development of cardiac hypertrophy and heart failure (HF). Previous study indicated that the Sestrin family suppressed oxidative stress and regulated AMPK/mTOR signaling. However, the expression and functions of Sestrin in hypertrophic and failing hearts are still unclear.

METHODS We performed quantitative RT-PCR to detect the mRNA expression of Sestrin family in the normal, hypertrophic (2 week after surgery; n = 4), 4 week after surgery; n = 4) and failing (more than 8 week after surgery; n = 4) mouse hearts induced by aortic banding.
(AB). We also detected the expression profile of Sestrin family in the cardiac tissue from patients with HF (HF group: n = 3) or with normal left ventricular function (control group: n = 3).

RESULTS We found that all three members of Sestrin family had elevated expression in the early stage of cardiac hypertrophy and decreased expression in failing hearts. Sestrin3 had a higher expression level compared to sestrin1 and sestrin2 in mouse heart. As shown in the manuscript, sestrin1 had the highest expression level and dropped far below the normal level at the failing stage, however, the expression of sestrin2 had no significantly difference between normal and failing human heart, while the sestrin3 in HF group was more than 2 fold increased compare to the normal group.

CONCLUSIONS Sestrin isoform had high expression in hearts especially sestrin1, and presented in a process of dynamic change during the development and transition of cardiac hypertrophy to heart failure. The expression profile in human hearts was different from that in the mouse heart. Further investigation of Sestrin function and regulation may provide new insights in hypertrophic and failing heart.

GW26-e4763 Berberine Prevents Atherosclerosis In Apolipoprotein E Knock Out Mice
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OBJECTIVES Berberine, the main alkaloid of Coptis Chinensis, has been widely used in traditional Chinese medicine for a long time. Berberine, the main alkaloid of Coptis Chinensis, has been recently shown to possess extensive cardiovascular pharmacological activities. In present study, we examined the effects of Berberine on aortic atherosclerosis in Apolipoprotein E gene knockout mice (ApoE-/−) and explored the potential underlying mechanisms.

METHODS 30 ApoE−/− mice, fed a high fat diet from 6 weeks of age, were randomized into three groups (n = 10): model group (ApoE−/−), Berberine group (ApoE−/−/Berberine group) and Simvastatin group (ApoE−/−/Simvastatin group). 10 6-week-old C57BL/6 were treated as the control group, fed a basic diet. After 36 weeks, we sacrificed the mice for various measurements with ELISA, Western blot and Real-time PCR.

RESULTS The results showed that treatment with Berberine significantly reduced blood lipid. Berberine has the effect of anti-proliferation of Smooth Muscle Cells. It could reduce the level of HS-CRP, IL-6 and TNF-α in plasma. And it could reduce protein and mRNA expression of NF-κB and MMP-9 in aorta. There is no significant difference between the control group and Simvastatin group.

CONCLUSIONS Berberine has the effect of anti-atherosclerosis and anti-inflammation in ApoE−/− mice. Our data have provided some experimental evidences to use Berberine in prevention and cure of atherosclerosis.

GW26-e5324 Study on Cross-immunization Protection of Coxsackievirus B3 gene vaccine
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OBJECTIVES Explore the (resulting in myocarditis) cross-protective immunity coxsackievirus B3 gene vaccine for other types of Coxsackie virus infection.

METHODS Using molecular biological method, Competence bacterium were produced and transformed by pcDNA3CVB3VP1 recombinant plasmid. the recombinant plasmids were extracted and identified by sect-enzyme, PCR and sequence; the accredited gene vaccine were Proliferated abundantly and BALB/c mice were immunized then. After x 6 weeks and x 6 weeks, immunization serum was acquired. CVB1, CVB3, CVB5m and CVB5 were Proliferated and titerated by virological experimental method; cross-immunization protection were observed by Neutralization test.

RESULTS After pcDNA3CVB3VP1 gene vaccine were identified, it was showed that the aimed CVB3VP1 fragment were conjugated with plasmid pcDNA3; results of neutralization tests indicate that pathological changes of Hela cells infected by CVB1, CVB3, CVB5m and CVB5 were attenuated due to adding serum from mice bodies inoculated with coxsackievirus B3. Moreover attenuating degree of pathological changes of Hela cells was different which were infected by different types of viruses.

CONCLUSIONS Coxsackievirus B3 gene vaccine plays a protective role in infection of CVB1, CVB3, CVB5m and CVB5; furthermore the protection is different in infection of CVB1, CVB3, CVB5m and CVB5.

GW26-e0207 Monocyte activation in atherosclerosis
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OBJECTIVES Monocyte recruitment in arterial wall is an early event in atherogenesis. The classically activated macrophages (M1 subpopulation) and alternative-activated macrophage (M2 subpopulation) can arise from monocytes in response to atherosclerosis or infection. To reveal the reason of relationship between intracellular cholesterol and cytokine expression, we aimed at exploring the potential underlying mechanisms. We attempted to evaluate the susceptibility to M1 and M2 activation of monocytes circulating in the blood of healthy individuals and patients with asymptomatic carotid atherosclerosis.

METHODS Cross-sectional clinical study was performed, which involved healthy donors, apparently healthy subjects with a predisposition to atherosclerosis, and patients with subclinical atherosclerosis. Study participants did not have clinical manifestations of atherosclerotic disease (ischemic heart disease, myocardial infarction, stroke history), did not take cardiotropic and lipid-lowering drugs, and did not have a history of microvascular diseases. Quantitative diagnostics of pro-atherosclerotic and atherosclerotic states were performed by high-resolution ultrasonography of carotid arteries followed by intima-media thickness (IMT) of common carotid arteries. To identify individual profiles of cell activation, monocytes were isolated from whole blood using magnetic CD14-positive separation. Functional analysis of monocyte activity included the measurement of concentrations of cytokines produced by cells under standardized conditions in response to pro-inflammatory stimulation with interferon-γ or anti-inflammatory stimulation with interleukin-4. Concentrations of TNF-α, IL-6, IL-8, IL-10, and CCL18 chemokine as a marker of anti-inflammatory activity were measured.

RESULTS Surprisingly, we found a dramatic individual difference in susceptibility to activation between monocytes isolated from the blood of different subjects, regardless of the presence or absence of antibiotics. Monocytes isolated from patients with documented atherosclerosis may migrate back into the circulation, possibly serving as a lipid clearance system. We attempted to find the relationship between cholesterol in monocytes and their susceptibility to activation. There was an obvious trend towards a reverse association between intracellular cholesterol level and the ability of monocytes to become activated; however, correlation coefficients did not reach statistical significance. To reveal the reason of relationship between intracellular cholesterol level and monocyte susceptibility to activation, we used atherogenic medium to induce cholesterol accumulation in cultured cells. Although modified LDL induced cholesterol accumulation in cultured monocyte-derived cells neither cytokine secretion nor cytokine genes expression were affected.

CONCLUSIONS We believe these observations are very important because the identified differences may explain the individual features of the immune response in different subjects. Supported by Russian Scientific Foundation (Grant # 14-15-00112).

GW26-e0466 Endothelial cell dysfunction induced by CD137-CD137L/CyclophilinnA activation through oxidative stress via NF-κappaB pathways
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OBJECTIVES Endothelial cell (EC) dysfunction is a key event in the onset and progression of atherosclerosis. Our previous studies showed...