CONCLUSIONS Isolation and culture of primary CSCs with 37°C coating cell culture bottles and modified CCM is a more superior method, which lays the foundation for the further experimental study.

GW26-e1432
Effect of Rosiglitazone on Insulin Resistance and ROS/IKK Signaling Pathway in Vascular Endothelial Cells
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OBJECTIVES To explore the protective effect of rosiglitazone on insulin resistance (IR) induced by high glucose in vascular endothelial cells and its possible mechanism.

METHODS Human umbilical vein endothelial cells (HUVECs) was divided into 3 groups: the normal control group cultivated in DEME medium with 5.5 mM/l glucose; the high glucose group (HG) cultivated in DEME medium with 33 mM/l glucose for 24 h after the IR model was set up; the rosiglitazone group cultivated in DEME medium with 33 mM/l glucose and 10 μM of rosiglitazone for 24 h after the IR model was set up. The cell viability, nitric oxide (NO), endothelin-1 (ET-1), mitochondrial membrane potential, reactive oxygen species (ROS), P- I KK and iκ Bα protein levels were detected.

RESULTS Compared with the normal control, the cell viability, the level of NO and the mitochondrial membrane potential were decreased, levels of ET-1 and ROS increased, P-I KK expression was upregulated, and iκ Bα expression was down-regulated in HG group (all P < 0.01). Rosiglitazone reversed these changes (P < 0.05).

CONCLUSIONS Rosiglitazone has the protective effect on insulin resistance induced by high glucose in vascular endothelial cells via inhibiting ROS/IKK signaling pathway.

GW26-e2914
A study of the mechanism of valsartan pre-protecting adriamycin-induced cardiotoxicity
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OBJECTIVES To investigate the effects of valsartan pre-protects adriamycin-induced cardiotoxicity and the mechanism.

METHODS 61 of 8w SD rats were divided into 4 groups as follows: Control group (n = 9) were feded normally; adriamycin-induced cardiotoxicity (AT1) group; group D: valsartan; group C: valsartan (high dose) intervened (VLD) group, Group D: valsartan (high dose) intervened (VLD) group, Group C (n = 6): valsartan (low dose) intervened (LVD) group, Group BC: valsartan (high dose) intervened (VLD) group, Group C (n = 6): valsartan (low dose) intervened (LVD) group. The first dose of adriamycin, totally 6 mg/kg was administrated (2 mg/kg, 3 times in 24h). Before 24 h after the IR model was set up, the cell viability, nitric oxide (NO), endothelin-1 (ET-1), mitochondrial membrane potential, reactive oxygen species (ROS), P- I KK and iκ Bα protein levels were detected.

RESULTS Compared with the normal control, the cell viability, the level of NO and the mitochondrial membrane potential were decreased, levels of ET-1 and ROS increased, P-I KK expression was upregulated, and iκ Bα expression was down-regulated in HG group (all P < 0.01). Rosiglitazone reversed these changes (P < 0.05).

CONCLUSIONS Valsartan might via activating RAS in rats' myocardial tissues to cause angiotensin overexpressed. Angiotensin-stimulated to generate more ROS. So the superabundant ROS caused the myocardial apoptosis and fibrosis.

GW26-e2924
Endothelial progenitor cells join in HHcy impaired angiogenesis
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OBJECTIVES During the last decade, we and others have demonstrated that HHcy can inhibit endothelial cell growth and postinjury reendothelialization, accelerate neointimal formation, also can impair endothelial relaxation, stimulate vascular smooth muscle cell proliferation, and inhibit high-density lipoprotein biosynthesis. However, the fundamental basis of HHcy-induced angiogenesis remains unknown, especially the role of endothelial progenitor cells in angiogenesis.

METHODS 1. Angiogenesis of HHcy mice under myocardial infarction. 1.1 Carcinogenesis was measured with echocardiography (VisualSonics Vevo 770); 1.2 Hearts were moved at 2 weeks/6 weeks after myocardial infarction and kept at -80°C until used. Frozen heart tissues were cut into 5μm thick slices. Adjacent sections (taken at the midpoint between LAD ligation site and apex) were stained with rabbit polyclonal antibodies against CD31.

2. Flow cytometry analysis. A volume of 200μl of peripheral blood was incubated for 30 minutes in the dark with monoclonal antibodies against mouse vascular endothelial growth factor receptor 2 (VEGFR2) followed by PE-conjugated secondary antibody.

3. MACS Separation-purity SCA-1+ cells. Purity of SCA-1+ cell is based on the use of MACS MicroBeads, MACS Columns and MACS Separators.

4. Intravenous transfections of SCA-1+ cells maybe play a role in HHcy MI mice angiogenesis.

RESULTS 1. HHcy impairs mouse cardiac function. Ejection fraction (EF) and fractional shortening (FS) were lower in HHcy mice group than control group, as well as heart capillary density. HHcy mice hearts have depressed function and less capillary density after myocardial infarction stress. Survival rate is also lower in HHcy mice.

2. Peripheral blood derived EPC percentage decreased in HHcy mice group and bone marrow derived EPC percentage is higher in HHcy mice group, but cell death rate is also higher in HHcy mice.

3. Intravenous transfection of SCA-1+ cells treatment induce PB derived EPC percentage increase in both control mice group and HHcy mice group. 6 weeks survival rate increased from 12.5% to 27.3% in HHcy mice group, but cell death rate is also higher in HHcy mice group.

CONCLUSIONS EPC joined angiogenesis after myocardial infarction which is so important to cardiac function. Cell treatment restores ischemia-induced angiogenesis in HHcy mice.
OBJECTIVES The relationship between the beneficial effects of pioglitazone in reducing clinical events and plaque inflammatory burden remains unknown. This study aims to determine whether pioglitazone can reduce the number of plaque thrombosis incidences and whether decreasing plaque inflammation plays a role in this mechanism by which pioglitazone reduces plaque thrombosis.

METHODS Atherosclerotic rabbits were divided into two groups: the atherosclerosis group (n=13) and pioglitazone group (n=10). The rabbits underwent pharmacological triggering to induce thrombosis. Serum inflammatory markers, FDG uptake, macrophage and neovessel staining detected arterial inflammation. PET/CT scans were performed twice (baseline and post-treatment scans). Plaque area, macrophage accumulation, and neovessels were measured and the histological sections were stained for p65, CD68, CD31 and Masson’s trichrome. Results Plaque area, macrophage density, and neovessels density was significantly lower in rabbits with pioglitazone without than without (15% vs. 38%, P < 0.001; 15.00 ± 2.30 vs. 27.00 ± 1.60; P < 0.001; 8.80 ± 3.94 vs. 28.26 ± 2.49; P < 0.001; 16.50 ± 3.09 vs. 29.00 ± 2.11; P < 0.001, respectively). Moreover, plaque area and macrophage density were positively correlated with SUV values.

CONCLUSIONS Our study suggests that pioglitazone can reduce the number of plaque thrombosis incidences by decreasing plaque inflammation. 18F-FDG-PET/CT can detect plaque inflammation and assess the effects of antiatherosclerotic drugs.

GW26-e2212 Effect of Atorvastatin on IKK/NF-κB Pathway of Insulin Resistance in Vascular Endothelial Cells Fanghui Chen The First Affiliated Hospital of Gannan Medical University

OBJECTIVES To investigate the influence of atorvastatin on IKK/NF-κB pathway of insulin resistance in vascular endothelial cells.

METHODS Human umbilical vein endothelial cells (HUVECs) were cultured in vitro with high glucose and insulin to establish a model of insulin resistance. Then the cells were treated with 10μmol/L of atorvastatin for 24h. Western blot method was used to assay the expression of pIκK, IκBα, NF-κB p65 protein. The levels of nitric oxide (NO), endothelin-1 (ET-1), TNF-α, IL-6, ICAM-1 and VCAM-1 were measured by enzyme linked immunosorbent assay (ELISA) kits.

RESULTS HUVECs were cultured with high glucose and insulin, the levels of ET-1, TNF-α, IL-6, ICAM-1 and VCAM-1 were increased, level of NO decreased, p-IκK and NF-κB p65 expression was upregulated, and IκBα expression was down-regulated (all P<0.05). Atorvastatin reversed these changes (all P<0.05).

CONCLUSIONS Atorvastatin has the protective effect on insulin resistance induced by high glucose in vascular endothelial cells. The underlying mechanism may be connected with inhibition of IKK/NF-κB Pathway.

GW26-e0013 The effect and mechanism of Glucagon-like peptide-1 protects against the diabetic cardiomyopathy Qinan Wu, Xiaotian Lei, Xiaguang Gan, Wuquan Deng, Bing Chen, Ziwen Liang, Ziwen Liang The first affiliated Hospital of the Third Military Medical University

OBJECTIVES To investigate the effect and mechanism of Glucagon-like peptide-1(GLP-1) on diabetic cardiomyopathy.

METHODS All rats were divided into normal group(N), diabetes group(D, STZ and high fat diet to construct diabetic animal model), diabetes treat with insulin group (DI) and diabetes treat with Exenatide (a glucagon-like peptide-1 receptor agonist) group (DA). After modeling for 12 weeks, we detected cardiac function by multimedia bio-signal recorder, detected apoptosis rate of cardiomyocyte by TUNEL, detected blood serum adiponectin by ELISA, detected protein expression of PPARα, NF-κB by western-blotting.

RESULTS Compare with N group, the cardiac myocyte apoptosis rate of D group was significantly increased, the concentration of adiponectin was significantly decreased(P<0.05), the expression of PPARα was decreased (P<0.05), the expression of NF-κB were significantly increased, LV -dP/dt was significantly decreased, LVEDP was significantly increased(P<0.05); Compare with D group, the apoptosis rate of DA group cardiomyocyte was significantly decreased(P<0.05), the concentration of adiponectin was significantly increased(P<0.05), the expression of PPARα and LV -dP/dt were significantly increased (P<0.05), LVEDP, the expression of NF-κB were decreased (P<0.05). Compare with DA group, although the glucose handling difference (P<0.05), the cardiac myocyte apoptosis rate and LV -dP/dt of DI group were significantly increased(P<0.05), the expression of PPARα and LV -dP/dt were decreased (P<0.05), the expression of NF-κB was significantly increased, the concentration of adiponectin was significantly increased(P<0.05).

CONCLUSIONS GLP-1 can reduce the number of plaque thrombosis incidences and whether pioglitazone in reducing clinical events and plaque inflammatory burden remains unknown. This study aims to determine whether pioglitazone can reduce the number of plaque thrombosis incidences and whether decreasing plaque inflammation plays a role in this mechanism by which pioglitazone reduces plaque thrombosis.

GW26-e0749 The effects of Local Cardiac Denervation on Cardiac Nerve Sprouting and Ventricular Electrophysiology after Chronic MI in dogs Rui Jiang, Jugang Chen, Yingying Jin, Jingjie Li The First Affiliated Hospital of Harbin Medical University

OBJECTIVES The aim of this study was to investigate the impact of the coronary sinus (CS) and great cardiac vein (GCV) peripheral nerves ablation on cardiac nerve sprouting and ventricular electrophysiology after experiment MI in dogs.

METHODS Twenty-one anesthetized dogs were randomly divided into the sham-operation (n = 2), MI (n = 8) and MI-denervation groups (n = 8), respectively. The dogs were ligated with left anterior descending branch for MI induction, the CS and GCV peripheral nerves were ablated by radiofrequency. 4 week after ablation or the generation of MI, the incidence and duration of ventricular arrhythmias (VA) were monitored for 1h during left stellate ganglion (LSG) stimulation, and programmed electrical stimulation was performed to measure the ventricular effective refractory period (ERP) and to induce ventricular fibrillation threshold (VFT) in all animals. At the end of experiment, the density of tyrosine hydroxylase (TH) and growth associated protein 43(GAP43)-positive sympathetic nerve fibers as well as infarct size were also detected. Systemic arterial pressure and heart rate were monitored during experiment.

RESULTS 4 week after ablation or the generation of MI, two dogs in only MI group died; the incidence and duration of VA in MI-ablation group were significantly lower than that MI-placebo group during LSG stimulation (P<0.05), and sustained ventricular tachycardia was induced in MI group. The infarcted border zone ERP and VFT in MI-denervation groups were significantly improved when compared with MI group. Furthermore, TH and GAP43-positive sympathetic nerve fibers in the cardiac base and infarcted border zone were also decreased (P<0.05). Systemic arterial pressure and heart rate as well as infarct size were displayed a marked difference when compared with MI group (P<0.05).

CONCLUSIONS Using the canine MI model, we showed that local ablation of CS and GCV peripheral nerves improves cardiac nerve sprouting and ventricular electrophysiology after chronic MI, and reduces the occurrence of VA with no obvious effects on heart rate, systemic arterial pressure and infarct size. Therefore, local cardiac denervation may protect from ventricular arrhythmias complicating chronic MI.

GW26-e0757 Inhibitory Effects of Luofengning-0 Formula on the Growth and Proliferation of Human Coronary Artery Smooth Muscle Cells and Endothelial Cells in Vitro: An Exploration of a New Type of Complex Monomer of Chinese Herbs Eluting Stents Hongmei Li,1,2 Mengqiong Sun,1,2 Ang Gao,1,2 Zhen Wang,1,2 Xueqing Yang,1,2 Tian Sun,1,2 Changbo Xuan,1,2 Xian Wang1,2 Af Pathway. Rui Jiang, Jugang Chen, Yingying Jin, Jingjie Li The First Affiliated Hospital of Harbin Medical University

OBJECTIVES To prove the effectiveness and feasibility of Luofengning-0 complexes and provide the experimental data for the prevention of restenosis, we investigated the inhibitory effects of different ratio of...