CONCLUSIONS Isolation and culture of primary CSCs with 37°C coating cell culture bottles and modified GCM 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 mmol/L glucose; the high glucose group (HG) cultivated in DEME medium with 33 mmol/L glucose for 24 h after the IR model was set up; the rosiglitazone group cultured in DEME medium with 33 mmol/L glucose and 10 μmol/L 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-1IκBα and IκBα 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, and IκBα expression was upregulated, and IκBα 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 fed normally; adriamycin-induced cardiotoxicity) group: intraperitoneal ADR, 2.5 mg/kg.w, totally 6 weeks, cumulative dose was 15 mg/kg. Group C (n=17): valsartan(low dose) intervened (VLD) group, Group D: valsartan(high dose) intervened group (VHD). In group C and D, valsartan was separately (10 mg/(kg.d) and 30 mg/(kg.d) given from the first dose of adriamycin, totally 6 weeks. After 6 weeks administration of valsartan and adriamycin, observed 4 weeks. Use echocardiography to detect myocardial infarction and apoptosis in myocardial tissues. Use qRT-PCR to detect collagen I, collagen III, caspase3 and caspase8’s expressions.

RESULTS
1. Compared with blank control group, renin, angiotensin II, aldosterone synthase, ROS in the myocardial tissues of the model group are evidently overexpressed (P<0.05). In group model, rats myocardial tissue, caspase3, caspase8 mRNA levels increased significantly (P<0.05), the myocardial collagen volume increased significantly (P<0.05).
2. Valsartan high dose group compared with blank control group, there was no significant statistical differences about indexes above (P>0.05). compared with model control group, the valsartan high dose group and the blank control group, there were significant differences about the above indexes (P<0.05).
3. There were no significant differences in LVEDD among the four groups (P>0.05); About LVESD, LVEF, LVFS, there were no significant difference between the valsartan low dose group and the model control group. The above index average were less than the model control group and the valsartan low dose group. The differences among the three groups were significant (P<0.05). Compared with the high dose group and the blank control group, the above indexes in model control group were higher. The differences were significant (P<0.05). There were no difference between the valsartan high dose group and blank control group (P>0.05).

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-impaired angiogenesis remains unknown, especially the role of endothelial progenitor cells in angiogenesis. Role of endothelial progenitor cells in HHcy impaired angiogenesis. Intravenous transfusions of Sca-1+ cells maybe play a role in HHcy MI mice angiogenesis.

METHODS
1. Angiogenesis of HHcy mice under myocardial infarction. 1.1 Cardiac function was measured with echocardiography (VisualSonics Vevo 770); 1.2 Hearts were moved at 2 weeks/6 weeks after myocardial infarction and kept at -80℃ until needed. Frozen heart tissues were cut into 5 μm 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 200 μl peripheral blood/bone marrow were 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 transfusions of Sca-1+ cells in HHcy MI mice angiogenesis. To evaluate the homing to infarcted heart of injected cells, 200 μl purified Sca-1+ cells were labeled with CellVue® Nho/Perinfluorescence (near-infrared) 780 (Excitation max: 745 nm/Emission max: 776 nm, Mol. Targeting Tech. Inc. West Chester, PA) and injected peri-ortobally into C57/B6 mice, 6 hours before MI procedure.

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 transfusion 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 cHcy -/+ cell treat group, and also 62.5% to 80% in cHcy +/+ cell treat group; The LVEF increased from 19.3% to 38.5% in cHcy +/+ cell treat group, and also 31.6% to 50.9% in cHcy +/+ cell treat group.

CONCLUSIONS EPC joined angiogenesis after myocardial infarction which is so important to cardiac function. Cell treatment restores ischemia-induced angiogenesis in HHcy mice.

GW26-e3551
Plaque Thrombosis Are Reduced by Attenuating Plaque Inflammation with Pioglitazone and Are Evaluated by Fluorodeoxyglucose Positron Emission Tomography
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