PCR (RT-qPCR). The level of membrane CD37 was measured by Flow Cytometry (FCM). The levels of NF-κB p65, phospo-NF-κB p65(p-p65) and NFATc1 of the VSMCs were determined by Western blot. TRAF6 was blocked by small interfering RNA(siRNA). MT was used to observe the cell proliferation. The cell supernatant of IL-2 and IL-6 were tested by ELISA.

RESULTS The level of CD37 was induced by TNF-α in smooth muscle cells after stimulating 24h(40.00±2.83 vs 1.00±0.00, p<0.05). The cells proliferated when treated with the agonist-CD37 mAb. The mRNA level of NFATc1 was increased after stimulated by agonist-CD37 mAb(2.07±0.09 vs 1.00±0.00, p<0.05), the protein of NFATc1 was also increased. Simultaneously, the mRNA level of TRAF6 was also increased(1.39±0.16 vs 1.00±0.00, p<0.05) and p-p65 protein was also increased(p<0.05). When TRAF6 was blocked by TRAF6 siRNA, the expression of p-p65 and NFATc1 was decreased after stimulated by agonist-CD37 mAb compared with the stimulated group(1.15±0.07 vs 2.07±0.09, p<0.05). Pretreated the VSMCs with PDTC(30μmol/L) after 30min, the expression of p-p65 was inhibited in cytoplasm and nucleus, we stimulated the cells by agonist-CD37 mAb at this time, the mRNA level of NFATc1 was also suppressed(1.15±0.07 vs 2.07±0.09, p<0.05) and the NFATc1 protein was inhibited(p<0.05). The cell supernatant of IL-2 and IL-6 went up by agonist-CD37 mAb(0.91±0.1 vs 1.29±0.17, p<0.05;0.51±0.04 vs 0.61±0.07, p<0.05), and decreased when NFATc1 was silenced(1.29±0.23 vs 1.00±0.00, p<0.05;0.61±0.07 vs 0.50±0.03, p<0.05).

CONCLUSIONS These results demonstrates that CD37 can be induced by TNF-α in VSMCs, and the CD37 may signalizing affect the expression of NFATc1 in mice VSMCs through TRAF6/NF-κB pathway.

GW26-e0806 Intracoronary Cardiomyocyte-Derived Cells for Heart Regeneration After Myocardial Infarction
Jie Qin, Yuefei Guo, Xiuzhen Chen, Xuelian Liu
Department of Radiology, The Third Affiliated Hospital of Sun Yat-sen University

OBJECTIVES We aimed to assess safety of therapy with cardiomyocyte-derived cells (CDCs) in patients with left ventricular dysfunction after myocardial infarction.

METHODS An independent data coordinating center randomly allocated patients in a 2:1 ratio to receive CDCs or standard care. For patients assigned to receive CDCs, autologous cells grown from endomyocardial biopsy specimen were infused into the infarct-related artery 1-5 months after myocardial infarction. The primary endpoint was proportion of patients at 6 months who died due to ventricular tachycardia, ventricular fibrillation, or sudden unexpected death, or new/progressed left ventricular infarction after cell infusion, new cardiac tumor formation on MRI, or a major adverse cardiac event. We also assessed 5-year protocol analysis. Mean baseline left ventricular ejection fraction (LVEF) was 40% (SD 22), and mean age was 60 years and older that had died of coronary heart disease, heart failure or renal failure, observing calcified lesions. Explant culture of arterial adventitia was used to observe calcification.

RESULTS Between May 1, 2009, and Dec 30, 2014, we randomly allocated 31 eligible participants of whom 25 were included in a per-protocol analysis. Mean baseline left ventricular ejection fraction (LVEF) was 39% (SD 12) and scar occupied 24% (10) of left ventricular mass. Biopsy samples yielded prescribed cell doses within 36 days (SD 6). No complications were reported within 24 h of CDC infusion. By 6 months, no patients had died, developed cardiac tumors, or MACE in either group. Four patients (24%) in the CDC group had serious adverse events compared with one control (13%; p=1.00). Compared with controls at 6 months, MRI analysis of patients treated with CDCs showed reductions in scar mass (p<0.001), increases in viable heart mass ratio (p<0.001) and regional contractility (p<0.001), and regional systolic wall thickening (p<0.001). However, changes in end-diastolic volume, end-systolic volume, and LVEF did not differ between groups by 6 months.

CONCLUSIONS Intracoronary infusion of autologous CDCs after myocardial infarction is safe and effective, warranting the expansion of such therapy to phase 2 study.

GW26-e10455 ITRAQ-Based Quantitative Proteomic Analysis of Heart in a Rat Model of Exhaustive Training
Haiyan Liu, Xuebin Cao
No. 252 Hospital of PLA

OBJECTIVES To explore the presence of informative protein biomarkers in the rat cardiac between the health group and exhausting group, The purpose of this study is to understand the difference in protein expression patterns between health and after exhaustive swimming and to evaluate the protein contributions to exhaustive training.

METHODS 20 male SD rats (male, the weight is 200±20g, SPF) were randomly divided two groups, health group and exhausting group. The rats of exhausting group were swimming in the artificial pond, get the swimming speed result in a model of Single Bout of Exhaustive swimming in Rats. After exhaustive swimming the hearts were collected immediately. We mainly adopted advanced 8-plex ITRAQ coupled with 2D LC-MS/MS technology for proteomics.

RESULTS Analysis of proteomic data found that 122 proteins were identified with quantitative information between the two groups, with the expression level of 69 proteins had significant differences at least, compared with the health group(ratio >1.2 or <0.7, and P<0.05), of which 50 proteins were up-regulated, 19 proteins down-regulated.

CONCLUSIONS This study provided a global view of potential mechanisms and potential biomarkers of heart, and demonstrated that ITRAQ combined with 2D LC-MS/MS quantitative proteomics is a powerful tool for biomarker discovery.

GW26-e1069 Exendin-4, An Guelcagon-like Peptide 1 Analogue, Attenuates Cardiomyocyte Hypertrophy Via AMPK/mTOR Pathway
Yue Zhou, Xin He, Yiyi Huang, Yili Chen, Jiangui He
Department of Cardiology, The First Affiliated Hospital of Sun Yat-sen University

OBJECTIVES Cardiac hypertrophy is the pathological basis of the development of various cardiovascular diseases and is a major independent risk factor of cardiovascular morbidity and mortality. Glucagon-like peptide 1 (GLP-1), an incretin peptide released from the intestine, exerts various cardioprotective actions and is proved to contribute in the regulation of cardiac functions. However, the role of Exendin-4, a stable GLP-1 analogue, in the cardiac hypertrophy remains unclear.

METHODS 1. Primary neonatal ventricular cardiomyocytes were cultured to establish the model of cardiomyocyte hypertrophy induced by phenylephrine (PE).
2. RNA isolation and quantitative real-time PCR (q-PCR) were performed to evaluate the transcriptional level of hypertrophic markers such as ANP, BNP and beta-MHC.
3. Western Blotting was carried out to observe the change of the signaling pathway proteins levels.
4. The cardiomyocyte morphological change was manifested by Immunofluorescence staining and measurement of the cell surface area.

RESULTS Our study demonstrated that exendin-4 attenuated cardiac hypertrophy induced by phenylephrine (PE), manifested by decreased hypertrophic markers such as ANP, BNP, beta-MHC and cell surface. Phosphorylated extracellular signal regulated protein kinase (phospho-ERK1/2) and phosphor-p38 mitogen-activated protein kinase (MAPK) and protein levels didn't change in the treatment of ANP and PE. In addition, we discovered that exendin-9 (39), a GLP-1 receptor antagonist, can remove the anti-hypertrophic effect of exen- din-4, evidently. Moreover, we showed that the anti-hypertrophic effect of exendin-4 was also significantly reversed by compound C, an AMPK inhibitor, and rapamycin, a selective blocker of mTOR.

CONCLUSIONS These results demonstrate that exendin-4 inhibit cardiac hypertrophy induced by PE via AMPK/mTOR pathway.

GW26-e1380 Vascular Adventitia Calcification and Its Underlying Mechanism
Na Li, Wenli Cheng
China-Japan Friendship Hospital

OBJECTIVES Previous research on vascular calcification has mainly focused on the vascular intima and media. However, we show here that vascular calcifications also occur in the adventitia as well. The purpose of this work is to help elucidate the pathogenic mechanisms underlying vascular calcification.

METHODS Mice were fed high fat diets (HFD) for 48 weeks, observing calcified lesions. Also included in this study were human subjects aged 60 years and older that had died of coronary heart disease, heart failure or renal failure, observing calcified lesions. Extract culture of fibroblasts, the primary cell type comprising the adventitia, was
induced for calcification. Culture of smooth muscle cells (SMCs), which comprise only a small percentage of all cells in the adventitia, in calcifying medium resulted in calcification.

RESULTS Following 48 weeks, calcified lesions were observed in the aorta adventitia and coronary artery adventitia of ApoE-/mice. Von Kossa staining showed calcification in the human aorta adventitia. Explant culture of fibroblasts, was successfully induced for calcification after vascular injury. Sphingosine-1-phosphate (SIP) via the sphingosine-1-phosphate receptor 3 (SIP3) induce proliferation, migration and angiogenesis of SMCs. This study aims to investigate effects of transplantation of SMCs overexpressing SIP3 on reendothelialization and neointimal formation in response to vascular injury in mice.

G2W6-e1588 Transplantation of EPCs Overexpressing SIP3 Promotes Vascular Repair in the Early Phase After Vascular Injury
Hang Wang, Keyin Cai, Li Wang, Kehong Zhao, Ying Tu, Xiao Wang, Jiahuan Li, Hao Huang
Cadre Ward Two, Wuhan General Hospital of Guangzhou Military Command, Wuhan 430070, China; 1Clinic center, Shenzhen Horntcorn Biotechnology Crop. Shenzhen 518400, China

OBJECTIVES Endothelial progenitor cells (EPCs) play important roles in the process of reendothelialization and prevent neointimal formation after vascular injury. Sphingosine-1-phosphate (SIP) via the sphingosine-1-phosphate receptor 3 (SIP3) induce proliferation, migration and angiogenesis of EPCs. This study aims to investigate effects of transplantation of EPCs overexpressing SIP3 on reendothelialization and neointimal formation in response to vascular injury in mice.

METHODS Spleen-derived EPCs were cultured and expanded in endothelial basal medium. EPCs were infected with lentivirus vectors expressing murine SIP3 (SIP3-EPCs) or green fluorescent protein (GFP-EPCs). Three days after gene transfection, the mRNA level and protein expression of SIP3 in the GFP-EPCs transplanted group and in the GFP-EPCs transplanted group were assessed. At day 7, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 14, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 21, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 28, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 35, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 42, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 49, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 56, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 63, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05). At day 70, the reendothelialized area in the SIP3-EPCs group was significantly larger than that in the GFP-EPCs transplanted group (P < 0.05).

CONCLUSIONS Our data suggest that transplantation of EPCs overexpressing SIP3 can have a combined effect of both amplifying the reendothelialization capacity of EPCs and inhibiting neointima formation so as to facilitate better inhibition of adverse remodeling after vascular injury. Therefore, gene modified EPCs may be applied in clinical progenitor cell therapy to improve vascular repair after vascular injury.

G2W6-e1598 Aberrant Expression of Circular RNAs in Endothelial Dysfunction
Yuqiang Ji, Manli Cheng
First hospital of Xi’an Jiaotong University, China

OBJECTIVES The aim of this study was to determine the expression profile of circular RNAs (circRNAs) in oxidized low-density lipoprotein (ox-LDL) treated human umbilical vein endothelial cells (HUVECs) compared with normal HUVECs through human circRNA microarray and predict their miRNA binding sites.

METHODS The global circRNAs expression profiles in ox-LDL treated HUVECs compared with normal HUVECs were measured by the Agilent Feature Extraction software (version 11.0.1.1) was used to analyze acquired microarray images. The circRNA/miRNA interaction was predicted with the Arraystar’s home-made miRNA target prediction software.

RESULTS Compared with normal HUVECs, 24 circRNAs were different expression (fold change >1.5, p-value cut-off is 0.05) in ox-LDL treated HUVECs. 15 circRNAs were up-regulated and 9 circRNAs were down-regulated. The most up-regulated circRNA was circRNA-104137 and the most down-regulated circRNA was circRNA-100188. The miRNA binding sites of circRNA-104137 were mir-455-3p, mir-218-1-3p, mir-423-5p, mir-503-5p and mir-223-3p. The miRNA binding sites of circRNA-100188 were miR-637, miR-608, miR-328-3p, mir-877-3p and mir-189-3p.

CONCLUSIONS The aberrantly expression profile of circRNAs in ox-LDL treated HUVECs compared with normal HUVECs indicates the potential roles of circRNAs in endothelial dysfunction. This study may provide new insights into the mechanism and potential targets for the endothelial dysfunction.

G2W6-e1596 Effects of atorvastatin on mRNA and protein expression of adropin in cultured human umbilical endothelial cells and rat artery smooth muscle cells
Liangping Zhao, Chengjia Zhang, Li Wang, Tao You, Weiting Xu, Jianchang Chen
Department of Cardiology, The Second Affiliated Hospital of Sooknow University China

OBJECTIVES Adropin is a newly-identified secretory protein that participates in the regulation of energy homeostasis and insulin response. Growing published evidence presented the beneficial association of adropin with coronary artery disease. A laboratory test showed the beneficial effects of adropin on endothelial cells proliferation and migration. The endothelial cell dysfunction and proliferation of vascular smooth muscle cell form the core mechanism of atherosclerosis. Therefore, to increase the expression of adropin by some therapy may play a role in the prevention and treatment of atherosclerosis. In the present study, the effects of atorvastatin on mRNA and protein expression of adropin in cultured human umbilical vein endothelial cells (HUVEC) and rat artery smooth muscle cell (RASMC) were investigated.

METHODS HUVEC and RASMC were cultured in vitro with atorvastatin of 0.002, 0.02, 0.2, 2 and 20 umol/L for 6, 12 and 24 hours. The proliferation of HUVEC and RASMC were detected by MTT chro-