accelerate the age-related RLA stiffness occurs earlier than to accelerate the age-related RAA stiffness. The mechanism is probably associated with the up-regulated level of RAGE in IRA media, while the AGES in serum or IRSA media may be involved in the late stage.

**GW26-e4656**

Polymorphism of RBP4 Locus Is Associated with 5-Year Survival in acute coronary syndrome after coronary revascularization

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**OBJECTIVES** The rs7094671 was single nucleotide polymorphism of RBP4 locus that was associated with prevalence of coronary artery disease. No data concerning their association with long term prognosis after myocardial infarction is available. The aim of our study was to investigate the association of the RBP4 locus with 5-year overall mortality in patients with acute coronary syndrome after coronary revascularization.

**RESULTS** The baseline characteristics were well-balanced between carriers (AA, n = 19; AG, n = 73) and GG (n = 200) of the RBP4 variant. During the follow-up period (2.45±11.98months), the primary endpoint occurred more frequently in carriers of A allele than in non-carriers of A allele (24.8% versus 9.3%; hazard ratio [HR] = 2.656; 95% confidence interval [CI] = 1.228-5.735; P = 0.018). Kaplan-Meier estimation of the primary outcome measure (death of any cause) during the follow-up period. (AA groups versus GG groups: adjusted HR = 6.321, 95% CI 2.081-20.20, P = 0.001). GA groups versus GG groups: adjusted HR = 1.930, 95% CI = 0.478-7.548, P = 0.605).

**CONCLUSIONS** The RBP4 locus is associated with 5-year mortality in high-risk patients with acute coronary syndrome.

**GW26-e4667**

Effect of Oxidatively Modified Low-Density Lipoprotein on Osteodifferentiation of Mesenchymal Stem Cells Co-cultured with Vascular Smooth Muscle Cells

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**OBJECTIVES** Increasing evidences revealed that bone marrow-derived mesenchymal stem cells (BM-MSCs) played important role in wound healing and vascular remodeling in vivo. However, the mechanism in the development of atherosclerosis and vessel calcify remains unclear. The aim of this study was to investigate the effect of oxidatively modified low-density lipoprotein (ox-LDL) on osteodifferentiation of BM-MSCs co-cultured with smooth muscle cells with or without osteogenic inducer, and further to explore the mechanism of BM-MSCs participating in atherosclerosis and vessel calcify.

**METHODS** BM-MSCs and vascular smooth muscle cells (VSMCs) were prepared from Sprague-Dawley rats and co-cultured in a transwell coculture system, which allowed the diffusion of secreted factors but prevented cell contact. Thecombined group (both osteogenic inducer and ox-LDL), ox-LDL group, osteogenic inducer group and control group were allocated according to factorial design method. The effect of ox-LDL on the osteogenic potential was determined by cell morphology, real-time PCR, immunofluorescent staining, alkaline phosphatase (AKP) activity and osteopontin (OPN) synthesis.

**RESULTS**

1. All groups expressed OPN mRNA and AKP and but the OPN mRNA and AKP expression levels in combined group were the highest after 14 days of cell culture.

2. The result of immunity histochemistry also showed that OPN and AKP expression levels in combined group were the highest after 14 days of cell culture.

**CONCLUSIONS** Ox-LDL can promote osteogenic inducer-mediated osteodifferentiation of BM-MSCs co-cultured with smooth muscle cells.

**GW26-e4724**

The role of mAKAPβ in the process of cardiomyocyte hypertrophy induced by angiotensin II

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**OBJECTIVES** Angiotensin II (AngII) is the central product of the reninangiotensin system (RAS) and this octapeptide contributes to the pathophysiology of cardiac hypertrophy and remodeling. mAKAPβ is an A kinase anchoring protein (AKAP) that has the function of binding to the regulatory subunit of protein kinase A (PKA) and confining the holoenzyme to discrete locations within the cell. In this study, we aim to investigate the role of mAKAPβ in AngII induced cardiomyocyte hypertrophy and the possible mechanisms involved.

**METHODS** Cardiomyocytes from neonatal rats were treated with AngII. Subsequently, the morphology of the cardiomyocytes was observed and the expression of mAKAPβ and cardiomyocyte hypertrophic markers was measured. mAKAPβ-shRNA was constructed for RNA interference; the expression of mAKAPβ and hypertrophic markers, the cell surface area and the [3H] Leucine incorporation rate in the AngII-treated rat cardiomyocytes were detected following RNA interference. Simultaneously, changes in the expression levels of phosphorylated extracellular signal-regulated kinase (p-ERK2) in the cardiomyocytes were assessed.

**RESULTS** The cell size of the AngII - treated cardiomyocytes was significantly larger than that of the untreated cardiomyocytes. The expression of hypertrophic markers and p-ERK2, the cell surface area and the [3H] Leucine incorporation rate in the AngII-treated rat cardiomyocytes were detected following RNA interference. Simultaneously, changes in the expression levels of phosphorylated extracellular signal-regulated kinase (p-ERK2) in the cardiomyocytes were assessed.

**CONCLUSIONS** AngII induces hypertrophy in cardiomyocytes and mAKAPβ is possibly involved in this process. The effects of mAKAPβ on AngII-induced cardiomyocyte hypertrophy may be associated with p-ERK2 expression.
Conclusions in addition to increased blood pressure, heart rate of SHR were also higher than WKY, indicating the presence of significant sympathetic activation of hypertension. Catestatin can lower heart rate in hypertension transiently, but long-term supplement does not affect heart rate, which may be other compensatory mechanisms. Catestatin does not also affect blood pressure in hypertension. These conclusions indicate catestatin may inhibit the sympathetic activation in hypertension to some extent, and involve in the pathogenesis of hypertension, but not be the deciding factor.

RESULTS

Objectives

Cardiac microvascular endothelial cells (CMECs) dysfunction is an important pathophysiological event in the cardiovascular complications induced by diabetes. However, the underlying mechanism is not fully clarified. Autophagy is involved in programmed cell death. Here we investigated the potential role of autophagy in the CMECs injury induced by high glucose.

Methods
CMECs were cultured in normal or high glucose medium for 6h, 12h and 24h respectively. The autophagy of CMECs was measured by green fluorescence protein (GFP)-LC3 plasmid transfection. Moreover, the apoptosis of CMECs was determined by flow cytometry. Furthermore, 3-Methyladenine (3MA), ATG7 siRNA and rapamycin were administrated to regulate the autophagy state. Moreover, Western blotting assay was performed to measure the expressions of Akt, mTOR, LC3 and p62.

Results
High glucose stress decreased the autophagy, whereas increased the apoptosis in CMECs time dependently. Meanwhile, high glucose stress activated the Akt/mTOR signal pathway. Furthermore, autophagy inhibitor, 3-MA and ATG7siRNA impaired the autophagy and decreased the apoptosis in CMECs treated by high glucose stress. Conversely, rapamycin up-regulated the autophagy and decreased the apoptosis in CMECs under high glucose condition.

Conclusions
Our data suggested that autophagy, as an adaptive response, is directly inhibited by high glucose in CMECs. Furthermore, the autophagy was mediated, at least in part, by mTOR signaling.

Activation of Cannabinoid Receptor 2 improves therapeutic efficacy of Adipose-Derived Mesenchymal Stem Cells to alleviate myocardial ischemia injury through AMPK/SIRT1-AMPK Pathway

Activation of Cannabinoid Receptor 2 (CB2R) agonist AM1241 will improve survival of adipose-derived mesenchymal stem cells (AD-MSCs) after transplantation into infarcted hearts and further discussed its underlying mechanisms.

Methods
We investigated the therapeutic effects of Cannabinoid Receptor 2 (CB2R) agonist AM1241 and co-transplantation of MSCs on cardiac repair in myocardial infarction by using bioluminescence imaging. AD-MSCs were isolated from Fluc-¢-eGFP transgenic mice (Tg (Fluc- eGfp)). Animals were divided into 7 groups: (1) Sham group, (2) MI group, (3) MI+CB2R group, (4) MI+AD-MSCs group, (5) MI+AD-MSCs+CB2R agonist AM1241 pretreatment before transplantation group, (6) MI+AD-MSCs transplanted with SIRT1 siRNA+ CB2R agonist AM1241, and (7) MI+AD-MSCs+CB2R agonist AM1241 group. Cardiac performance was then quantified by echocardiography as well as molecular and pathological analysis of heart samples at serial time points. The survival and engraftment of transplanted MSCs were also assessed by both bioluminescence imaging and histologic analysis. To reveal possible mechanisms, AD-MSCs were subjected to hypoxia/serum deprivation (H/SD) injury to simulate ischemic conditions in vivo. Western blot assay was used to detect the expression of related signal transduction proteins in inflammation and oxidative stress.

Results

Conclusions
CB2R agonist can enhance the functional survival of transplanted AD-MSCs in infarcted myocardium, at least partially, via