75.7% patients had hypertension history and 33.0% patients had hyperalkemia history. For group: The 3 patients with adenial hyperplasia had hyperalkemia history than that without adenial hyperplasia (39.2% vs. 20.7%, P<0.001). The frequency of CC genotype of rs3740835 in KCNJ5 gene was higher in control group rather than that in adenial hyperplasia group (67.7% vs 58.2%, P=0.047), though the significance only reached the borderline. (3) Logistic regression model was constructed to explore the potential factors that related to CT scan-based adenial hyperplasia in whole study population. The results showed that the rs3740835 in KCNJ5 gene (0.642 (0.424-0.971), P=0.036) as well as plasma potassium [0.432 (0.261-0.715), P=0.01] played protective roles in adenial hyperplasia, adjusted for body mass index, gender, age. Conclusions: The rs3740835 variants in KCNJ5 gene as well as plasma potassium levels were contributing factors to adenial hyperplasia in patients with PA from Xinjiang.

**GW25-e5201**

The mechanism research of Compound Danshen dripping pills accuring myocardial infarction from the TLR4-NF-kB - PECA1-1 pathways

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**Objectives:** To observe the protection and its mechanism of Compound Danshen dripping pills on myocardial infarction.

**Methods:** 1. Male Wistar rats were randomly divided into normal group, model control group, experimental model group, isosorbide dinitrate (ISD) group, Compound Danshen (FFDS) group, 10 rats in each group. The myocardial infarction models and the model of myocardial infarction with the Qi and blood stasis were established respectively, and the models rats were treated with isosorbide dinitrate and Compound Danshen dripping pills intervention. Cardiac ejection fraction, myocardial infarction, GMP-140 of blood serum and TRL4, TRAF-6, IL-1β, PECAM-1, NF-kB, protein and NF-kB gene expression of myocardial tissue were observed.

**Results:** After NIBT staining, myocardial tissue in the normal group were stained purple; There were large grey infarcted region in myocardial tissue of each model group. There were smaller gray infarction area than ISD group and FFDS group. The ejection fraction (EF) in the model group, significantly lower than that in normal group; Compound Danshen dripping pills could improve cardiac function (P<0.01); GMP-140 of blood serum of FFDS group were lower than that of model group (P<0.05).

Immunohistochemical results showed TRL4, TRAF-6, IL-1β, PECAM-1, NF-kB protein expression in the normal group was low, the model group were higher than those in normal group (P<0.01); isosorbide dinitrate and Compound Danshen dripping pills could decrease TRL4, TRAF-6, IL-1β, PECAM-1, NF-kB protein expression (P<0.01). RT-PCR results show, NF-kB gene level was lowest in the normal group. NF-kB gene level of FFDS group was lower than that in model group (P<0.01).

Conclusions: The effect of Compound Danshen dripping pills on myocardial protection may be related to regulation of TRL4/TRA4-6/NF-kB pathway.

**GW25-e5348**

Effects of Simvastatin combined with ezetimibe on atherosclerosis in ApoE-/- mice

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**Objectives:** To investigate effects of Simvastatin combined with ezetimibe on atherosclerosis in ApoE-/- mice with high-fat diet.

**Methods:** 36 male apoE-/- mice (age, 8weeks) on a C57BL/6 background were randomly divided into three groups with 12 animals in each group. Animals were treated with intragastric administration as follows: Model group received PBS buffer per day for 8 weeks; Simvastatin group received simvastatin (20mg/kg intragastric administration) every day for 8 weeks; Combination therapy group were treated with 10mg/kg ezetimibe and 20 mg/kg simvastatin every day for 8 weeks. Then the whole aorta mice were collected for oil red O staining use.

**Results:** Serum TG, TC and LDL-C levels were significantly lower in simvastatin and combination therapy group (P<0.01). TG and LDL-C levels were decreased more significantly in combination therapy group (P<0.01, compared with simvastatin group). The percentage of aortic plaque area in simvastatin group and combined therapy group was significantly reduced (P<0.05, in which the combined therapy group decreased more significantly (P<0.01).

Conclusions: Simvastatin + ezetimibe therapy inhibits atherosclerosis more obviously.

**GW25-e5365**

Bone Marrow stem cells: immune property genes assay and effect of transplantation on the immune cells of heart failure patients

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**Objectives:** To investigate the effects of stem cells derived from bone marrow (BMSCs) are immunogenic and have immunosuppressive.

**Methods:** To evaluate the related mechanisms and the effect of transplantation on bone immunogenic cells, we examined immune property genes expression in BMSCs and levels of T-lymphocytes subgroups and immunoglobulins (Ig) in heart failure (HF) patients with and without BMSCs transplantation. BMSCs express immune tolerance genes HLA-E, HLA-A and HLA-F and immunomodulation genes VEGF, TFGB1, HGF, HMMOX1, IL1b, IL-6, LIF, LGALS-1/5/6, COX1/2 and PTGE, while they do not express immune response-related genes HLA-DR, HLA-DQ, HLA-DP, CD80, CD86, CD40 and CD40L.

**Results:** There were no significant differences in blood CD3+, CD4+, or CD8+ T-lymphocytes subgroups and the CD4+/CD8+ ratio between patients who received cell implantation and controls at the baseline and 3 days, 14 days and 1 month after transplantation. In addition, no significant differences among T-lymphocytes subgroups and CD4+/CD8+ ratio among different time points were found in the transplantation group or the control group. No obvious changes of plasma IgG/IgM were observed in HF patients with BMSCs transplantation.

**Conclusions:** The immune properties of BMSCs are due to the expression of immune avoidance and immunomodulation genes in the absence of immune response-related genes. BMSCs are secure in immunological aspects when used as seed cells for cardiac repair.

**GW25-e5383**

A RTK-based functional RNAi screen reveals determinants of PTX-3 expression

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**Objectives:** The aim of the present study was to explore the role of receptor tyrosine kinases (RTKs) in the regulation of expression of PTX-3, a protector in atherosclerosis.

**Methods:** Human monocytic U937 cells were infected with a shRNA lentiviral vector library targeting human RTKs upon LPS stimuli and PTX-3 expression was determined by ELISA analysis. The involvement of downstream signaling in the regulation of PTX-3 expression was analyzed by both Western blotting and ELISA assay.

**Results:** We found that knocking down of ErbB2/3, EphA7, and FGFR3 and RET impaired PTX-3 expression without effects on cell growth or viability. Moreover, inhibition of AKT, the downstream effector of ErbB2/3, also reduced PTX-3 expression. Furthermore, we showed that FGFR3 inhibition by anti-cancer drugs attenuated p38 activity, in turn induced a reduction of PTX-3 expression.

**Conclusions:** Altogether, our study demonstrates the role of RTKs in the regulation of PTX-3 expression and uncovers a potential cardio toxicity effect of RTK inhibitor that needs to be understood in cancer patients who have symptoms of atherosclerosis or are at the risk of atherosclerosis.

**GW25-e5399**

Effects of AngII and Ang-(1-7) on the Cholesterol Eflux in THP-1 Derived Foam Cells

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**Objectives:** To investigate the effects of angiotensinII (AngII) and angiotensin-(1-7) [Ang-(1-7)] on scavenger receptor class B type1 (SR-BI), ATP-binding cassette transporter A1 (ABCA1) and cholesterol efflux in THP-1 derived foam cells.

**Methods:** Human monocyctic cell line (THP-1) were induced into macrophages by liquid scintillator. Human monocytic cell line (THP-1) were induced into macrophages by liquid scintillator. Human monocytic cell line (THP-1) were induced into macrophages by liquid scintillator.

**Results:** Compared with the control group, AngII decreased SR-BI and ABCA1 in both human monocyctic cell line (THP-1) and increases the cholesterol efflux in THP-1 derived foam cells.

**Conclusions:** Foam Cells

**GW25-e3426**

Homocysteine impairs macrophage cholesterol efflux via LXR alpha hypermethylation

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**Objectives:** The mechanisms of homocysteine-mediated lipid disorder are poorly understood. Liver X receptors alpha, as a cholesterol-sensing nuclear receptors, are the key regulators of macrophage cholesterol efflux. This work aimed to explore the methylation modification mechanisms of LXR alpha in homocysteine impairing cholesterol efflux in THP-1 macrophage.