derenervation, n=10), RD-3d+MI group (RD performed three days before MI, n=15), Metropolit-3d+MI group (Metropolit treated three days before MI, n=15), ACEI-3d+MI group (Perindopril treated three days before MI, n=15), and ARB-3d+MI group (Losartan treated three days before MI, n=15). Cardiac function, autonomic nervous system parameters (HRV), and neuroendocrine activities (plasma renin, angiotension II, aldosterone and noradrenaline Levels) were evaluated 8 weeks post MI.

**Results:** Ten of 20 animals in the MI group, 5 of 15 in the RD-3d+MI group, 5 of 15 in the metropolit-3d+MI group, 7 of 15 in the ACEI-3d+MI group and 8 of 15 in the ARB-3d+MI group died within the eight week period after coronary artery ligation. The death rate in the metropolit group and the metropolit group were the same and much less than the MI, ACEI, or ARB groups (P<0.05). The death rate did not differ between the latter three groups. None in the control and RD group died during the experiment. There were no significant differences in body weight or the infarct size among all experimental groups eight weeks post-MI. The results showed that the physiologic benefits of RD on improving cardiac remodeling and function, water and sodium excretion, autonomic modulation and suppression of RAAS activation were significantly better than any of the three drugs alone and had no effect on normal control.

**Conclusions:** In this post-MI HF animal model, surgical RD provides effective interventions in reducing infarct size, cardiac remodeling, and improving cardiac function. Surgical RD provides effective treatments for post-MI HF and increased body weight and reduced infarct size. Surgical RD may be superior to medical therapy in these experimental conditions.

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**GW25-e0746**

**Technique of synchronous culture of endothelial progenitor cells and Smooth muscle cell derived rabbit bone marrow**

**Zhang Yongzhen, Xu Fuchi**

**Sports Department of Tainhan University, Taiwan**

**Objectives:** To isolate rabbit bone marrow-derived mononuclear cell then synchronous culture rabbit endothelial progenitor cells (EPCs) and smooth muscle progenitor cells (SMCs); study their biological properties and assess the possibility of the seed cells for tissue-engineered venous valves.

**Methods:** Density gradient centrifugation was used to obtain bone marrow blood mononuclear cells, which were separately cultured with EGM-2 complete medium containing 10% FBS to be induced to EPC and with EBM-2 medium without VEGF containing 5% FBS, 20ng/ml PDGF-BB for SPC induction. EPCs were cultured for 10 days and the cells fused as monolayer, showing a “stepping stone” appearance and expressing VEGFR-2, VWF and weakly expressing CD133. Under the transmission electron microscope, W-P bodies could be seen within the EPCs cytoplasm. Biological functions showed visible EPC grew on the matrigel in a blood vessel-like form. SPCs was cultured for 14 days and showed specific features of the vascular smooth muscle growth, namely, “peak-valley” growth way, SPCs expressed CD34 and SMC marker, the longitudinal axis, could be seen under the electron microscope. SPCs could not form vessel-likes structures on the Matrigel.

**Conclusions:** Mononuclear cells could be obtained through density gradient centrifugation of the bone marrow blood, which could be synchronous cultured to EPCs and SPCs in high purity, provided seed cells for Venous valve tissue engineering economical and simply.

**GW25-e0838**

**eNOS modified endothelial progenitor cells inhibit efficiently neointima formation and enhancement of vascular function**

**Cai Bin, Huanglan**

**Cardiovascular Department of Xiangia Hospital, Third Military Medical University**

**Objectives:** Loss of endothelial NO production after arterial injury may contribute to restenosis, characterized by neointima formation and elastic recoil. Previous studies have established that bone marrow-derived endothelial progenitor cells (EPCs) play an important role in vascular repair. In this study, we investigated that overexpression of eNOS gene in EPCs (eNOS-EPCs) may restore NO production and inhibit neointimal hyperplasia.

**Methods:** EPCs obtained from rat bone marrow were isolated using a Ficoll density gradient centrifugation, and expanded in endothelial basal medium. The endothelial progenitor cells (EPCs) were identified by immunologic cell chemical staining and with fluorescent labeling. EPCs were transduced with pseudotyped retroviral vectors expressing human eNOS (eNOS-EPCs) or green fluorescent protein (GFP-EPCs). eNOS or GFP modified EPCs were injected directly by intravenous tail vein after arterial injury and again 24 hours later. Two weeks after transplantation, eNOS proteins in the rat vessels were assayed by western blot. The morphology of arterial intima and media was studied by optical microscopy and image analysis system.

**Results:** The adherent cells were considered EPCs which had spindle shape and form blood-island-like structure during development. The adherent cells had many endothelial characteristics. Transduction efficiency of EPCs ex vivo was above 90%. eNOS gene transfer augmented EPCs proliferative activity, eNOS proteins were detected in the rat vessels. Transfused EPCs may home to the injury site and enhanced reendothelialization associated with decreased neointima formation. The antiproliferative effect of EPCs is further enhanced by overexpression of eNOS. Furthermore, eNOS overexpressed EPCs could induce significantly endothelium-dependent vasodilation function (EDVR).

**Conclusions:** In vitro, eNOS gene transfer enhanced EPCs proliferative activity. In vivo, eNOS overexpressed EPCs could accelerated reendothelialization and inhibit neointimal hyperplasia. The results show that gene modified EPCs facilitate the strategy of cell transplantation for vascular dysfunction and prevention of restenosis after angioplasty.

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**GW25-e0843**

**c-Met overexpression promote reendothelialization and inhibit neointimal formation after balloon injury**

**Songmingxiao, Huanglan**

**Cardiovascular Department of Xiangia Hospital, Third Military Medical University**

**Objectives:** To explore the effect of c-met overexpression in EPCs on reendothelialization after balloon injury. EPCs were derived from mouse bone marrow were isolated and cultured. 3-, (4, 6-diamidino-2-phenylindole) -2, 7-diaminofluorescein and 0.25% acetic acid were used to label the endothelial cells as EPCs. EPCs obtained from rat bone marrow were isolated using a Ficoll density gradient centrifugation, and expanded in endothelial basal medium. The endothelial progenitor cells (EPCs) were identified by immunologic cell chemical staining and with fluorescent labeling. EPCs were cultured for 10 days and the cells fused as monolayer, showing a “stepping stone” appearance and expressing VEGFR-2, VWF and weakly expressing CD133. Under the transmission electron microscope, W-P bodies could be seen within the EPCs cytoplasm. Biological functions showed visible EPC grew on the matrigel in a blood vessel-like form. SPCs was cultured for 14 days and showed specific features of the vascular smooth muscle growth, namely, “peak-valley” growth way, SPCs expressed CD34 and SMC marker, the longitudinal axis, could be seen under the electron microscope. SPCs could not form vessel-likes structures on the Matrigel.

**Conclusions:** Mononuclear cells could be obtained through density gradient centrifugation of the bone marrow blood, which could be synchronous cultured to EPCs and SPCs in high purity, provided seed cells for Venous valve tissue engineering economical and simply.

**GW25-e0845**

**Baseline serum uric acid level as a predictor of cardiovascular disease related mortality and all-cause mortality: A meta-analysis of prospective studies.**

**Zhaogang, Huanglan**

**Cardiovascular Department of Xiangia Hospital, Third Military Medical University**

**Objectives:** Serum uric acid (SUA) levels have been used to predict cardiovascular and all-cause mortality event, but the data have yielded conflicting results. We investigated whether SUA was an independent predictor for cardiovascular or all-cause mortality with prospective studies by meta-analysis. Serum uric acid (SUA) levels have been used to predict cardiovascular and all-cause mortality event, but the data have yielded conflicting results. We investigated whether SUA was an independent predictor for cardiovascular or all-cause mortality with prospective studies by meta-analysis.

**Methods:** PubMed and Embase were searched without language restrictions for publications available till April 2013. Only prospective studies on cardiovascular or all-cause mortality related to SUA levels were included. Pooled adjusted relative risk (RR) and corresponding 95% CI were calculated separately for the highest vs. lowest category or the lowest vs. middle category.

**Results:** For the highest SUA, eleven studies with 172, 123 participants were identified and analyzed. Elevated SUA increased risk of all-cause mortality (RR 1.24; 95% CI 1.09-1.42) and cardiovascular mortality (RR 1.37; 95% CI 1.25-1.5). Subgroup analyses showed that elevated SUA significantly increase the risk of all-cause mortality among men (RR 1.23; 95% CI 1.08-1.42), but not in women (RR 1.05; 95% CI 0.9-1.24). A marked decrease in the neointimal area and 1/M ratio was found in c-met-EPCs compared with EPCs group at day 21 (0.29±0.06 vs. 0.63±0.13, p<0.05, P<0.01). Red) overexpression improve EPCs proliferation, promote reendothelialization and inhibit neointimal formation after balloon injury.

**GW25-e0877**

**Relaxin-2 and relaxin-3 inhibit high glucose-induced apoptosis in neonatal rat ventricular myocytes**

**Zhang Xiaohui, Yin Xinhua**

**The Department of Cardiology, The First Affiliated Hospital of Harbin Medical University, Harbin, China**

**Objectives:** High concentrations of glucose induce apoptosis in cardiomyocytes, and contribute to diabetic cardiomyopathy. Relaxin-2 and relaxin-3 are two members of the relaxin peptide family that are cardioprotective. In the present study, we
investigated the effect of either relaxin-2 or relaxin-3 on high glucose-induced apoptosis and fibrotic effects in atrial specimens of 13 patients with AF and 10 subjects with sinus rhythm. Subsequently, the role of caveolin-1 in human atrial fibroblasts was studied.

Results: The results showed that the expression of Cav-1 was significantly downregulated, whereas TGF-β1 level, collagen I/III contents and atrial fibrosis were markedly increased, in AF. Western blot analysis demonstrated that treatment of human atrial fibroblasts (HAFs) with TGF-β1 resulted in a concentration- and time-dependent repression of Cav-1. Downregulation of Cav-1 with siRNA increased the TGF-β1-induced activation of Smad signal pathway and collagen deposition in HAFs. Furthermore, incubation of HAFs with the peptides derived from Cav-1 allowed Cav-1 gain-of function abolished the TGF-β1-induced production of collagens and fibrosis in atrium.

Conclusions: Therefore it was concluded that Cav-1 is an important anti-AF signaling mediator by conferring its anti-fibrotic effects in atrium.

GW25-e0554

Changes of blood pressure and neural damage factors in hypertensive dogs after renal sympathetic denervation

Jiang Fenglin, Zhang Zhihua
Department of Cardiology, the Third Xiangya Hospital of Central South University

Objectives: To observe the changes of blood pressure and S-100B, Neuron Enolase Protein in hypertensive dogs used high fat diet after catheter-based renal sympathetic denervation.

Methods: We evaluated the expression of Cav-1, transforming growth factor-β1 (TGF-β1), and fibrosis in atrial specimens of 13 patients with AF and 10 subjects with sinus rhythm. Subsequently, the role of caveolin-1 in human atrial fibroblasts was studied.

Results: The results showed that the expression of Cav-1 was significantly downregulated, whereas TGF-β1 level, collagen I/III contents and atrial fibrosis were markedly increased, in AF. Western blot analysis demonstrated that treatment of human atrial fibroblasts (HAFs) with TGF-β1 resulted in a concentration- and time-dependent repression of Cav-1. Downregulation of Cav-1 with siRNA increased the TGF-β1-induced activation of Smad signal pathway and collagen deposition in HAFs. Furthermore, incubation of HAFs with the peptides derived from Cav-1 allowed Cav-1 gain-of function abolished the TGF-β1-induced production of collagens and fibrosis in atrium.

Conclusions: Therefore it was concluded that Cav-1 is an important anti-AF signaling mediator by conferring its anti-fibrotic effects in atrium.