**e0208** NERVE GROWTH FACTOR PROMOTE ANGIOGENESIS OF MESENCHYMAL STEM CELLS
doi:10.1136/hrt.2010.208967.208
Wang Wenxia, Hu Xinyang, Xie Xiaojie, Liu Xianbao, Wang Jian-an. Second Affiliated Hospital, Zhejiang University College of Medicine, Hangzhou, China

**Objective** To investigate whether nerve growth factor (NGF) can enhance angiogenesis of mesenchymal stem cells (MSCs), and the possible mechanism.

**Methods** MSCs were seeded into matrigel-coated 24-well plates, and cultured with NGF at different concentrations (0 ng/ml, 25 ng/ml, 50 ng/ml, 100 ng/ml, 200 ng/ml) for 24 h, the tube formation of MSCs was observed and photographed using an inverted microscope. K-252a, the specific inhibitor of NGF receptor TrkA, was used to inhibit the tube formation promoted by NGF. Western Blot was applied to compare the VEGF expression between different groups.

**Results** NGF can promote MSCs tube formation in vitro, which was peaked at the concentration of 50ng/ml with its tubular lengths 2.24-fold increased (p<0.05), and was attenuated by K-252a. There was no significant difference of the VEGF expression between NGF treated and control group.

**Conclusion** NGF enhanced the ability of MSCs angiogenesis in vitro, and TrkA pathway may be involved.

---

**e0209** PLATELETDERIVED MICROPARTICLES AFFECTS VASCULAR ENDOTHELIAL GROWTH FACTOR EXPRESSION AND THE MECHANISM IN ENDOTHELIAL CELLS
doi:10.1136/hrt.2010.208967.209
Jia Hongdan, Mao Yongmin, Li Ximing, Cui Rangzhuang, Yu Huining, Pei Chengmin, Cong Hongfang, Tianjin Chest Hospital, Tianjin, China

**Objective** Detect vascular endothelial growth factor induce by platelet-derived microparticles (PMPs) in human umbilical vein endothelial cells (HUVECs) and its downstream transduction pathway. The aim of our study was to discuss the possible mechanism of PMPs effecting on VEGF releasing and its clinical significance in endothelial cells.

**Methods** Applying the method of flow cytometry (FCM) to isolate platelet-derived microparticles (PMPs) from platelet poor plasma (PPP), and stained with fluorescein isothiocyanate mononal antibody against CD61, applying flow cytometry (FCM) method to detect the quantification of PMPs, and the total protein concentration of PMPs was determined using Semi-automatic Biochemical Analyser. HUVECs were commonly cultured. Different concentrations of PMPs intervene HUVECs and incubate for different time. We used semi-quantitative reverse transcription PCR techniques to measure VEGF, phosphatidylinositol-3 kinase (PI3K), extracellular signal-regulated kinase (ERK) and VEGF type II receptor (KDR) mRNA levels.

**Results** 1. High density of PMPs can be obtained from PPP after intervention of ADF, Thrombin and detected by FCM. 2. Different concentration of PMPs intervene HUVECs, VEGF mRNA level in control group (non intervention group) was significantly higher than the four different concentration of PMPs groups (p<0.05, respectively). In contrast, KDR mRNA level in control group (non intervention group) was significantly lower than the four different concentration of PMPs groups (p<0.05, respectively). ERK mRNA level in 50 μg/ml PMPs group was significantly higher than the other four groups (1.141 vs 0.749, p=0.004), PI3K mRNA level in 100 μg/ml PMPs group was significantly higher than the other groups (1.544 vs 0.999, p=0.004). 3. Just as the effect of different concentration PMPs on mRNA expression, VEGF mRNA level in control group (non intervention group) was significantly higher than the 24 h intervention time group (0.518 vs 0.746, p<0.001). KDR mRNA level in the 4 h and 24 h groups were significantly higher than control group (p<0.05, respectively). PI3K mRNA level in 24 h group was significantly higher than the control group (2.622 vs 0.999, p=0.004).

**Conclusions** Abundant PMPs can be obtained from PPP after activation. PMPs may induce the biological processes of blood vessel and angiogenesis via VEGF and its downstream signal transduction pathways.

---

**e0210** CILOSTAZOL REDUCES NEOINTIMIAL HYPERPLASIA BY INHIBITION SUPEROXIDE PRODUCTION AND EXPRESSION OF LECTIN-LIKE OXIDISED LDL RECEPTOR-1 AFTER BALLOON COMMON ARTERIAL INJURY IN A RAT MODEL
doi:10.1136/hrt.2010.208967.210
Hua-ke Su, Yao-ming Song, Shi-yong Yu, Wenyun-Guo, Qiliang-Liu, Wenjun-Li. Department of Cardiology, Xinqiao Hospital, Third Military Medical University, Chongqing, China

Lectin-like oxidised low-density lipoprotein receptor-1 (LOX-1) is a membrane protein that can support the binding, internalisation, and proteolytic degradation of oxidised low-density lipoprotein. The LOX-1 expression and superoxide generation increases in the neointima after balloon injury. Cilostazol, a well-know phosphodiesterase type 3(PDE3) inhibitor for the treatment of peripheral arterial disease, has vasodilator properties and an anti-proliferative action on the growth of vascular smooth muscle cells. To investigate whether cilostazol suppresses intimal hyperplasia and to elucidate its mechanism, we examined the effects of cilostazol to the expression of LOX-1 mRNA and protein, superoxide generation and neointimal hyperplasia of the rat carotid artery after balloon injury. The injury was performed inserting the balloon catheter through the rat common carotid artery and after 14 days a histopathological analysis revealed a significant restenosis with smooth muscle cell proliferation and neointima formation that was associated with an enhanced expression of LOX-1, superoxide generation, Pretreatment of rats with cilostazol (100 mg/kg/day) reduced neointima formation, superoxide generation, and LOX-1 expression (p<0.05). Here, we show that Cilostazol reduces neointimal hyperplasia by inhibition superoxide generation and expression of lectin-like oxidised LDL receptor-1 after balloon common arterial injury in a rat model.

---

**e0211** SI MIAO YONG AN DECOCTION PROMOTES Atherosclerotic Plaque Stability in Vulnerable Plaque Rabbits
doi:10.1136/hrt.2010.208967.211
1Li Peng, 2Jun-Ping Zhang, 1Ming Li, 1Liang-Jun Li, 1Ying-Zhi Xu, 1Guang-Yin Zhang, 1Cui Yang, 1Yu-Nan Zhou. 1Central Laboratory of Pharmacology, Tianjin University of Traditional Chinese Medicine, Tianjin, China; 2Department of Cardiovascular Medicine, The First Affiliated Hospital of Tianjin University of Traditional Chinese Medicine, Tianjin, China

**Background** Si Miao Yong An decoction and its medicine MAILoosing injection are proved-effect medicine treating ischaemic cardiovascular disease, but whether it can stabilise the atherosclerotic plaque is currently no clear conclusions. We want to explore the role of Si Miao Yong An decoction intervention on rabbit aortic atherosclerotic plaque instability.

**Methods** Japanese white rabbits were divided into control group, model group, Simvastatin group and Si Miao Yong An group. The
animals were killed at the end of the experiment. The pathological changes in aortic was observed by HE colouring, masson staining and oil red staining. CRP, MMP-9, ICAM-1 and NF-kB/p65 were tested with ELISA, immunochemistry or RT-PCR.

Results Pathological observation showed that thin fibrous cap and big lipid core area was the feature of plaque in the model group. The main components of plaque were macrophages and fat, collagen and actin content of plaque was little, which had showed the pathological characteristics of unstable plaque. We found atherosclerotic lesions of aorta in two drug groups were lighter than in model group (p<0.05 or p<0.01). Si Miao Yong An decoction was superior to Simvastatin in increasing fibrous cap thickness and actin content, reducing lipid core area and MMP-9 expression in plaque (p<0.01). However, there was no significant difference between the two drugs in reducing NF-kB/p65 and ICAM-1 mRNA expression (p>0.05).

Conclusions Based on these results, we believe that Si Miao Yong An decoction can promotes atherosclerotic plaque stability by fighting against inflammatory, inhibiting matrix degradation and lipid deposition.

---

**e0212** L-CARNITINE TREATMENT IMPROVES SURVIVAL AND EFFECTS OF TRANPLANTED BONE MARROW MESENCHYMAL STEM CELLS IN POST-INFARCT RATS HEARTS

doi:10.1136/hrt.2010.208967.212

Peilei Li, Ming Lin, Changsheng Xu. Department of Cardiology, The First Affiliated Hospital, Fujian Medical University, Fuzhou, Fujian, China

Objective To investigate if the L-carnitine can improve the survival rate of transplanted MSCs after myocardial infarction, and enhance therapeutic effect of MSCs.

Methods Five rats were separately obtained, MSCs were isolated and purified by density-gradient centrifugation and adherence method. 60 rats were randomly assigned into five groups: sham operation group (n=12), model group (n=12), LC group (n=12), MSCs group (n=12), and LC+MSCs group (n=12). Rats in the sham operation group received chest open, without ligation of the left coronary artery. In other four groups, the left coronary artery was ligated to establish myocardial infarction models. Following 20 min of coronary artery ligation, 250 μL MSCs (2×10⁶ cells per animal) were injected into the left ventricular wall of the infarcted hearts (50 μL into 1 injected foci) and peri-infarct zone (200 μL into 4 injected foci) in MSC group and LC+MSCs group, rats in the model group and sham operation group received intramyocardial injection of the same volume of cell-free DMEM. From 3 days prior to MSC transplantation to ended 4 days post-transplantation, rats in the LC group and LC+MSCs group were separately administrated with LC (100 mg/(kg·d)) intraperitoneally. Rats in the model group, sham operation group and MSCs group separately administrated with PBS (100 mg/ (kg·d)). The heart function was evaluated by left ventricular ejection fraction, shortening fraction, and the indexes of blood dynamics 4 weeks after transplantation, the survival of MSCs and myocardial fibrosis in myocardial infarction were detected using immunohistochemistry.

Results Compared with the model group, ejection fraction, fractional shortening, the left ventricular end-systolic pressure (LVESP), left ventricular end-diastolic pressure (LVEDP), the maximal rate of isovolumetric contraction (+dp/dtmax, −dp/dtmax) and myocardial fibrosis were improved in the group MSCs, group LC and group LC+MSCs (p<0.05); Compared with the MSCs group, the survival of MSCs, ejection fraction, fractional shortening, LVESP, LVEDP, +dp/dtmax, −dp/dtmax and myocardial fibrosis in group LC +MSCs have significantly improved (p<0.05).

Conclusion Cardiac function and myocardial fibrosis can be improved by LC and MSCs in acute myocardial infarction rat models, but the effect is limited. Pretreated with LC, MSCs transplantation will achieve better result for improving the survival of MSCs.