the liposome control group RNA interference group proliferation is weak ($p<0.05$).
Conclusion 1. HIF-1a, SDF-1a and VEGF gene expression can be affected by HIF-1a siRNA in MSCs. 2. Hypoxia can made HIF-1 a, SDF-1 a and VEGF gene expression increased. 3. SDF-1 a and VEGF gene expression may be controlled by HIF-1 a in MSC. 4. Cell culture medium stimulate SMC profliction can be reduced by RNA interfence.

e0099 BAICALIN PROTECTION RAT CARDIOMYOCYTES FROM ISCHAEMIA-REPERFUSION INJURY AND ANTIARRHYTHMIA VIA INHIBITING L-TYPE CALCIUM CURRENT

doi:10.1136/hrt.2010.208967.99

Teng Wang, Jingjing Wang, Wenyun Gan, Congxin Huang. *Department of Cardiology, Renmin Hospital of Wuhan University, Cardiovascular Research Institute of Wuhan University, Wuhan, China*

Objective To investigate baicalin protection rat cardiomyocytes from inschemia-reperfusion injury and antiarrhythmia via blocking $I_{Ca\text{-}L}$.
Methods The degree of ischaemia-reperfusion injury was assessed by the recovery of LVDP and the magnitude of the reperfusion contracture with using approach of the Langendorff-perfused isolated rat hearts. The effects of baicalin on APs and ouabain-induced DAD and AT were performed on rat papillary muscles by conventional microelectrode technique. $I_{Ca\text{-}L}$ was recorded via using whole-cell patch-clamp technique in enzymatically dissociated single rat ventricular myocytes.
Results Compared with the pre-ischaemic control, baicalin could concentration-dependently improved recovery of LVDP, and reduced the lever of reperfusion contracture, and occurrence of arrhythmias. Baicalin significantly shortened ADP_{20}, ADP_{50} and APD_{90} in rat papillary muscles. Ouabain could apparently induced the DAD and TA in rat papillary muscles. With administration of baicalin, the electrophysiological parameters of ouabain-induced DAD and TA were markedly inclined to difficult occurrence. It illustrated that baicalin might inhibit influx of $I_{Ca\text{-}L}$. Baicalin significantly inhibited $I_{Ca\text{-}L}$ in a voltage-dependent and concentration-dependent procedure, with an IC_{50} value of 27.7 ± 1.9 μmol/l (E_{max} and nH were $115.2\pm3.3\%$ and 1.07 ± 0.05, respectivly). Moreover, baicalin shifted the I-V curve of $I_{Ca\text{-}L}$ upwards. According to statistic kinetic data, it was suggested that baicalin especially inhibit the $I_{Ca\text{-}L}$ by eliciting a negative shift of the steady-state inactivation without affecting the slop factor. To the effect of baicalin on the speed of $I_{Ca\text{-}L}$ recovery from inactivation, our data indicated that the time courses of recovery were prolonged markedly ($p<0.01$ compare with control group, respectively).
Conclusions Baicalin improved cardioprotection effects on ischaemia-reperfusion injury and decreased the occurrence of ouabain-induced DAD and TA, thus inhibited $I_{Ca\text{-}L}$. The effects of baicalin on inhibiting $I_{Ca\text{-}L}$ might contribute to baicalin antagonising ischaemia-reperfusion injury and arrhythmia.

e0100 ASSOCIATION BETWEEN CREG GENE POLYMORPHISMS AND CORONARY ARTERY DISEASE IN THE HAN POPULATION OF NORTH CHINA

doi:10.1136/hrt.2010.208967.100

Tao Wang, Xiaolin Zhang, Yaling Han, Chenhui Yan, Mingfang Huang. *Shenyang Northern Hospital*

Introduction The purpose of the present study was to assess the possible association between CREG and CAD in the Han population of North China.
Methods ALL five selected SNPs were genotyped in 1161 patients with angiographically documented CAD and 960 control subjects free from CAD who had normal coronary angiograms. Patients and controls were unrelated individuals of Han Chinese from the northeast region of China, genotype analyses were performed additive, dominant and recessive models. Binary logistic regression was used to control for the presence of vascular risk factors both in genotype and haplotype analyses.
Results Genotype frequencies of the five examined polymorphisms were similarly distributed between CAD group and controls ($p>0.05$). Further haplotype analysis also found no significant differences in the distributions between CAD group and controls ($p>0.05$).
Conclusion This study did not show a statistically significant association between common variants of CREG and CAD in northern Chinese Han population.

e0101 INTERLEUKIN-17A GENE VARIANTS AND RISK OF CORONARY ARTERY DISEASE:A LARGE ANGIOGRAPHY-BASED STUDY

doi:10.1136/hrt.2010.208967.101

Xiaolin Zhang, Fang Pei, Yaling Han, Chenhui Yan, Mingfang Huang, Tao Wang. *Shenyang Northern Hospital*

Objective Recent studies have also revealed that interleukin (IL)-17A plays a key role in atherosclerosis and its complication, but the relationship of its common variants with coronary artery disease (CAD) has not been extensively studied.
Methods We systematically screened sequence variations in the IL17A gene and designed an angiography-based case-controlled study consisting of 1031 CAD patients and 935 control subjects to investigate the association between the selected polymorphisms of IL-17A gene and CAD risk in Chinese Han population.
Results Frequencies of IL17A rs8193037 GG homozygote and G allele were significantly higher in the patient group than those in the control group ($p<0.001$; OR=0.68; 95% CI 0.54 to 0.85). Stratification analysis showed that the IL17A rs8193037 G allele significantly increased the risk of CAD only among male subjects ($p=0.001$; OR=0.63; 95% CI 0.47 to 0.83). After adjustment for conventional risk factors, binary logistic regression analysis showed that the the G allele carriers (GG +AG) had significantly increased CAD risk compared with the AA homozygotes (adjusted $p<0.001$; OR 0.43; 95% CI 0.33 to 0.58). ELISA showed augmented IL17A production in serum of the AMI patients.
Conclusions Based on our data, we speculated that rs8193037 of IL17A is associated with CAD risk in Chinese Han population and G allele of rs8193037 may be an independent predictive factor for CAD.

e0102 EXPRESSION OR SECRETION OF IL-34 AND IL-35 IN THE PERIPHERAL BLOOD MONONUCLEAR CELLS FROM PATIENTS WITH DILATED CARDIOMYOPATHY

doi:10.1136/hrt.2010.208967.102

Huang Sisi, Wu Weifeng, Lin Song, Huang Yanlan. *The First Affiliated Hospital of Guangxi Medical University*

Objective The aim of this study was to observe the level of interleukin (IL)-34 and IL-35 in peripheral blood mononuclear cells (PBMCs) with dilated cardiomyopathy (DCM), and explore the role of IL-34 and IL-35 in human DCM.
Methods 30 patients with DCM and 30 normal adults as control were studied. IL-34 and the subunit Epstein-Barr virus-induced gene 3(EBI3) of IL-35 mRNA expression in PBMCs were detected by reverse transcription—PCR (RT-PCR). IL-34 and IL-35 protein level in plasma were measured with ELISA.
Result (1) Results showed that the IL-34 mRNA level or its protein level was significantly elevated in DCM patients compared with

normal control subjects. (mRNA level: DCM :0.37±0.08, NC: 0.19±0.03, p<0.01; protein level: DCM: 208.94±50.31 pg/ml; NC: 175.69±44.56 pg/ml; p<0.01) (2) The IL-35 subuint-EBI3 or its protein level was significantly decreased in DCM patients compared with normal control subjects. (EBI3 mRNA level: DCM: 0.15±0.03, NC: 0.33±0.07, p <0.01; protein level: DCM: 128.68±24.08 pg/ml, NC: 179.73±43.89 pg/ml, p<0.01) (3) The secretion of IL-34 was markedly correlation with the secretion of IL-35 (r=−0.490, p <0.01). (4) The protein level of IL-34 in DCM patients had a positive correlation with heart function (r=0.598, P <0.01). (5) The protein level of IL-35 in DCM patients had a negative correlation with heart function (r=−0.839, p<0.01).

Conclusion The ability to express IL-34 and IL-35 protein or mRNA in PBMCs is abnormal and the change strongly correlates with ejection fraction and heart function of DCM patients.

e0103 THE HINDIII POLYMORPHISM IN THE LIPOPROTEIN LIPASE GENE PREDICTS TYPE 2 DIABETES RISK AMONG CHINESE ADULTS

doi:10.1136/hrt.2010.208967.103

[1]Qi Yue, [2]Liu Jing, [2]Wang Wei, [2]Wang Miao, [2]Sun Jiayi, [2]Liu Jun, [2]Li Yan, [2]Qin Lanping, [2]Wu Zhaosu, [2]Zhao Dong. *[1]Capital Medical University Beijing Anzhen Hospital Beijing Institute of Heart Lung And Blood Vessel Diseases Beijing China; [2]Capital Medical University Beijing Anzhen Hospital Beijing Institute of Heart Lung And Blood Vessel Diseases Beijing China*

Objective To explore whether the HindIII polymorphism in the lipoprotein lipase (LPL) gene has a potential role in susceptibility to type 2 diabetes, and whether this relation is influenced by regulating LPL or other risk factors.

Research design and methods Overall, 654 Han Chinese adults were recruited from a community-based cross-sectional study. Genotyping was performed using the PCR-RFLP technique. Pre-heparin LPL (PrLPL) and other metabolic variables were measured using standard methods.

Results Individuals with the HindIII H−/H− genotype tended to have higher PrLPL and lower triglyceride (TG) levels but an unexpected higher prevalence of type 2 diabetes compared with carriers with the H+H+ genotype. The association between the H−/H− genotype and diabetes risk remained unchanged across all subgroups of diabetes-related risk factors and PrLPL. In an additive model, the H−/H− genotype conferred 178% increased risk [OR:2.78; 95% CI 1.04 to 7.47] for diabetes after controlling for age and sex. The strength of this association increased further after adjusting for other traditional risk factors, and for PrLPL (OR=4.06; 95% CI= 1.35 to 12.23). Furthermore, the H−/H− genotype was also associated with an increased risk of dysglycemia defined as insulin resistance plus diabetes.

Conclusions This study revealed that Chinese adults with the LPL gene HindIII H−/H− genotype had a significantly increased risk of type 2 diabetes compared with individuals with other genotypes, even if they had favourable lipid profiles.

e0104 MATCHED CASE-CONTROL STUDY ON MECHANISM OF RADIAL ARTERY SPASM

doi:10.1136/hrt.2010.208967.104

Jia Dean, Zhou Yujie, Shi Dongmei, Wang Zhijian, Yang Shiwei, Liu Xiaoli. *Beijing Anzhen Hospital Capital Medical University*

Objects Radial artery (RAS) spasm is the most common complication in transradial coronary angiography and intervention. The aim of this study is to preliminary discuss the relationship between vaso-active substances and RAS, find out the mechanism of RAS, and provide theoretic evidence for the solution of RAS prevention.

Methods This is a prospective, matched case-control study. The patients who received transradial coronary angiography were enrolled. The patients who suffered from RAS during the procedure were enrolled, and the patients without RAS were matched 1:2 according to same gender, similar age within 2 years. The diagnostic criteria are clinical definition of RAS based on a questionnaire which was documented by angiography. Blood samples were obtained before the procedure, and were tested for nitric oxide, endothelin-1, prostacyclin, thromboxane A2 and norepinephrine using enzyme-linked-immunosorbent assay. The concentration of each vaso-active substance was compared and multi logistic regression was made to find out the risk factors of RAS.

Results 30 patients suffered form RAS and 60 patients without RAS were enrolled. of all the clinical and procedural characteristics, successful access at first attempt (46.7% vs 75.0%, p=0.010) and ratio of severe pain at cannulation (13.3% vs 1.7%, p=0.041) were different between the RAS group and the control group, the others were of no difference. The concentration of nitric oxide (64.5512±24.2963 vs 57.6385±20.1472, p=0.426) and thromboxane A2 (0.9040±0.2158 vs 0.7364±0.2256, p=0.372) was of no difference between the RAS group and the control group. The concentration of endothelin-1 (276.3739±85.1481 vs 78.5275±23.6323, p<0.001) and norepinephrine (193.7551±41.8509 vs 54.4108±17.8031, p=0.006) was higher, prostacyclin (8.1947±3.2692 vs 14.5436±5.5867, p=0.041) was lower in RAS group. Multiple regression showed that endothelin-1 (OR 2.714, 95% CI 1.329 to 4.984, p=0.005) and norepinephrine (OR 4.285, 95% CI 2.219 to 10.372, p=0.014) were the risk factors of RAS during the procedure.

Conclusions Among the vaso-active substances, the concentration of nitric oxide and thromboxane A2 was of no difference, prostacyclin was lower and endothelin-1, norepinephrine was higher in RAS patients than in patients without RAS. Multiple regression showed that endothelin-1 and norepinephrine were the risk factors of RAS during the procedure.

e0105 IN VITRO BLOCKADE OF OESTROGEN RECEPTOR PROMOTES THE PROLIFERATION OF VASCULAR SMOOTH MUSCLE CELLS

doi:10.1136/hrt.2010.208967.105

[1]Hong He, [2]Fa-Lin Yang, [3]Xin Wang, [4]Hui Zhu, [4]Jinghuan Huang, [4]Zimo Liu. *[1]Department of Cardiology, Qilu Department of Cardiology, Qilu Hospital, University of Shandong, Jinan, China; [2]Clinical Laboratory, Qilu Hospital, University of Shandong, Jinan, China; [3]Department of Clinical Medicine, the Second Affiliated Hospital, University of Shandong, Jinan, China; [4]Department of Cardiology, Qilu Hospital, University of Shandong, Jinan, China*

Background The proliferation of vascular smooth muscle cells (VSMCs) is a key event in the development of atherosclerosis. Oestrogen receptor is expressed in VSMCs. In vivo studies have shown that reduced levels of oestrogen receptor associate with atherosclerosis in females. Accordingly, we performed a series of experiments to test the hypothesis that blocking oestrogen receptor could enhance the proliferation of VSMCs in vitro.

Methods and results ICI182, 780, a pure oestrogen receptor antagonist, has been shown to block oestrogen receptor completely. When VSMCs isolated from rat aorta were cultured in the presence of ICI182, 780, the cellular growth augmented significantly in a dose-dependent manner. An increase in proliferating cell nuclear antigen (PCNA)-positive cells was also observed in VSMCs treated with ICI182, 780. Flow cytometry demonstrated that the S-phase progression of cell cycle in the VSMCs was promoted significantly by ICI182, 780, this effect was associated with an increase in cyclin D1 expression.

Conclusions These findings demonstrate that in vitro blockade of oestrogen receptor promotes the growth of VSMCs, suggesting that oestrogen receptor expressed in arteries acts to inhibit the