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

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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

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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

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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