normal control subjects. (mRNA level: DCM: 0.57±0.08, NC: 0.19±0.03, p<0.01; protein level: DCM: 208.94±50.31 µg/ml, NC: 175.69±44.56 µg/ml, p<0.01) (2) The IL-35 subtype-EBl3 or its protein level was significantly decreased in DCM patients compared with normal control subjects. (EBl3 mRNA level: DCM: 0.15±0.03, NC: 0.33±0.07, p<0.01; protein level: DCM: 128.63±24.08 µg/ml, NC: 179.73±45.89 pg/ml, p<0.01) (5) 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=-0.859, p<0.01).

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

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-immunosorbert 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. 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 (15.3% 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 (64.55±24.2963 vs 57.63±20.1472, p=0.426) and thromboxane A2 (0.90±0.2158 vs 0.76±0.2256, p=0.372) was of no difference between the RAS group and the control group. The concentration of endothelin-1 (276.57±58.1451 vs 78.32±23.6533, p<0.001) and norepinephrine (193.75±41.8599 vs 54.41±17.5051, p=0.006) was higher, prostacyclin (6.1947±5.2692 vs 14.5436±5.3967, 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.

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 CCl182, 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 CCl182, 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 CCl182, 780. Flow cytometry demonstrated that the S-phase progression of cell cycle in the VSMCs was promoted significantly by CCl182, 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...