normal control subjects. (mRNA level: DCM: 0.37±0.08; NC: 0.19±0.03, p<0.01; protein level: DCM: 208.9±50.31 pg/ml; NC: 175.6±44.56 pg/ml; p=0.01) (2) The IL-35 subtype-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±45.89 pg/ml, p<0.01) (5) The secretion of IL-34 was markedly correlated 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 PBMCs 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-immunosorbent assay. The concentration of each vaso-active substance was compared and multi logistic regression was made to find 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=0.01) 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 57.6385±20.1472, p=0.426) and thromboxane A2 (0.904±0.2158 vs 0.7564±0.2256, p=0.372) was of no difference between the RAS group and the control group. The concentration of endothelin-1 (276.5759±85.481 vs 72.5275±25.6323, p<0.001) and norepinephrine (193.7581±41.8509 vs 54.4105±17.5051, p=0.006) was higher, prostacyclin (6.1947±3.2692 vs 14.5436±5.5367, 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 8.107, 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.
proliferation of VSMCs and play a functional role in atheroprotection. Efforts aiming at enhancing oestrogen receptor expression and/or activity may prove to be an attractive alternative therapy against atherosclerosis.

### e0106 THE EFFECT OF ADENOSINE AND ISCHAEMIA POSTCONDITIONING ON MMP-2 AND MMP-9 IN RABBIT ISCHAEMIA REPERFUSION MYOCARDIAL

**Objectives**
To observe the effect of adenosine and ischaemia postconditioning on MMP-2 and MMP-9 in rabbit ischaemia reperfusion myocardial.

**Methods**
The rabbits were divided into four groups in basic experiment: control group, antagonist group, postconditioning group and adenosine group. The activity of MMP-2 and MMP-9 was observed by gelatin zymography and the expression of MMP-2 and MMP-9 was observed by RT-PCR and Western Blot.

**Results**
The results of RT-PCR showed that the light density of MMP-2/GAPDH (0.76±0.22) in adenosine group and postconditioning group was slightly lower than that of control group and antagonist group.0.80±0.20 (p>0.05). The light density of MMP-2/GAPDH (0.77±0.12) in adenosine group and postconditioning group was significantly lower than that of control group and antagonist group 1.30±0.10 (p<0.01). There was no dramatic difference between adenosine group and postconditioning group. The results of Western blot showed that MMP-9 and MMP-2 in adenosine group and postconditioning group was lower than control group and antagonist group. The results of Zymography revealed that the light density of MMP-9 in adenosine group and postconditioning group much lower than those in control group and antagonist group (p<0.05). There was no difference between adenosine group and postconditioning (p>0.05).

**Conclusions**
Adenosine and postconditioning can decrease the expression and the activity of MMP-9 and inhibit the inflammation, relieving the ischaemia reperfusion injury.

### e0107 CARDIAC PROTECTIVE EFFECTS OF DIFFERENT DOSAGE ATORVASTATIN IN PATIENTS WITH STABLE ANGINA AFTER PERCUTANEOUS CORONARY INTERVENTION

**Background**
The incidence of myocardial injury limits the clinical outcomes of percutaneous coronary intervention (PCI). This randomised controlled study was designed to evaluate the protective effects of pretreatment atorvastatin on myocardial injury and inflammatory reaction after PCI.

**Methods**
82 patients with chronic stable angina without previous statins treatment in 2 months before PCI were randomised to receive atorvastatin 10 mg/qn (group A, n=27), 20 mg/qn (group B, n=28) or 40mg/qn (group C, n=27) treatment for 3 days before PCI. CK-MB, cTnI, hsCRP, IL-6, sICAM-1 were measured at baseline, 0 and 24 h after the procedure. 1-month clinical follow-up was obtained by office visit in all patients.

**Results**
The peak levels of CK-MB and cTnI were increased significantly in all three groups 24 h after PCI (all p<0.05). Either elevation above the upper normal limit (UNL) or >3×UNL of cTnI, there were significant differences between group A and B (p<0.05), and between group A and C (p<0.05), but no difference between group B and C (p>0.05). Similarly changes were also found in CK-MB. The level of IL-6, sICAM-1 and hsCRP 8 h after PCI were higher than those before PCI (all p<0.05). There were significant differences in IL-6 and hsCRP among the three groups (all p<0.05), but no significant difference in sICAM-1 (p>0.05). The level of hsCRP and sICAM-1 24 h after PCI were higher than those 8 h after PCI in all three groups (all p<0.05), but IL-6 significantly decreased (p<0.05). There were significant differences among the three groups (all p<0.05). No serious cardiovascular events occurred during follow-up.

**Conclusion**
Even short term pretreatment with atorvastatin before PCI may reduce procedural myocardial injury by reducing inflammatory factors in chronic stable angina patients.

### e0108 ESTROGEN INDUCES RECOVERY OF INJURED ARTERY ENDOTHELYL BY MOBILISING ENDOTHELIAL PROGENITOR CELL

**Introduction**
Mobilization of endothelial progenitor cells (EPC) restores endothelial function, representing a novel therapeutic direction for injured blood vessel recovery. The present study was designed to determine the effect of oestrogen on EPC mobilisation and regeneration of endothelium in mice.

**Methods**
Varioctomy is performed before treatment of 17β-oestradiol, 17β-oestradiol combination with oestrogen receptor agonist-ICI182780 and 17β-oestradiol with Pi3K blockers-LY294002. Then, carotid artery injury was preformed and neointima was evaluated by HE staining. 1 and 3 days later, mobilisation of EPCs was evaluated by FACS as double positive of Sca-1/VEGF-2. Evans blue was injected and area of reendothelialization was calculated after 7 days. To trace EPCs in vivo, 1×106 autologous spleen-derived EPCs were labelled with DAPI and transplanted through tail vein.

**Results**
1 or 3 days after carotid artery injury, EPCs of peripheral blood were 0.42±0.13% (n=6), 1.47±0.32% (n=6) (ovariectomy+E2); 0.15±0.24% (n=6), 0.25±0.04% (n=6) (ovariectomy); 0.45±0.16% (n=6), 0.65±0.21% (n=4) (ovariectomy+E2); 0.12±0.019% (n=6), 0.25±0.062% (n=6) (ovariectomy+E2+LY) and 0.12±0.019% (n=6), 0.24±0.067% (n=6) (ovariectomy+E2+ICD). Area of re-endothelialization were (ovariectomy, 28.33±13.49%, n=5) vs (ovariectomy+E2, 69.53±14.14%, n=5) vs (ovariectomy+E2+ICD, 83.11±7.94%, n=4) (p<0.01). In vivo tracing experiment detected blue fluorescence cells in injured sites that were also positive of CD31, indicating EPCs homing to target sites.

**Conclusion**
Oestrogen could induce EPCs mobilisation through ERs/Pi3K pathway which is helpful to promote endothelium recovery of injured carotid artery.

### e0109 THE EFFECTS OF C-REACTIVE PROTEIN ON TOLL-LIKE RECEPTOR 4 SIGNAL TRANSDUCTION ON CD14+ MONOCYTE

**Objectives**
To observe the effects of C-reactive protein (CRP) on Toll-like receptor 4 (TLR4) expression in CD14+ monocyte in human, and the role of CRP in the inflammatory mechanism.

**Methods**
CD14+ monocytes were isolated from blood in healthy volunteers by the Ficol density gradient and stimulated by CRP