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

doi:10.1136/hrt.2010.208967.106

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**Objective** 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. There was no dramitic 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 that of 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** CARDIAL PROTECTIVE EFFECTS OF DIFFERENT DOSAGE ATORVASTATIN IN PATIENTS WITH STABLE ANGINA AFTER PERCUTANEOUS CORONARY INTERVENTION

doi:10.1136/hrt.2010.208967.107

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**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/q (group A, n=27), 20 mg/q (group B, n=28) or 40 mg/q (group C, n=27) treatment for 3 days before PCI. CK-MB, cTnl, 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 cTnl 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 cTnl, 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 ENDOTHELIUM BY MOBILISING ENDOTHELIAL PROGENITOR CELL

doi:10.1136/hrt.2010.208967.108

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**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** Varicectomy is performed before treatment of 17-β-oestradiol, 17-β-oestradiol combination with oestrogen receptor agonist-ICI182780 and 17-β-oestradiol with PL3K blockers- LY294002. Then, carotid artery injury was performed and neointima was evaluated by H.E staining. 1 and 3 days later, mobilisation of EPCs was evaluated by FACS as double positive of Sca-1/VEGFR-2. Evans blue was injected and area of reendothelization 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=8) (ovariectomy+E2); 0.15±0.024% (n=6), 0.25±0.024% (n=6) (ovariectomy); 0.43±0.16% (n=6), 0.65±0.21% (n=4) (non-ovariectomy); 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-endothelization were (ovariectomy, 28.33±13.49%, n=5) vs (ovariectomy+E2, 69.53±14.14%, n=5) vs (non-ovariectomy, 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 can induce EPCs mobilisation through ERs/PL3K pathway which is helpful to promote endothelium recovery of injured carotid artery.
with different concentrations (5, 25, 50, 100 μg/ml) and different exposure time (50 μg/ml CRP coincubated for 6, 12, 24 and 48 h). The protein expression of TLR4 was measured by flow cytometry and mRNA expression of TLR4 and MD-2 were tested by quantitative PCR. Measurements of TNFα, IL-6 and MMP-9 in the supernatants of cultured monocytes were performed by ELISA.

**Results** CRP (5, 25, 50 and 100 μg/ml) increased dose-dependently the expression of TLR4 protein (32.22±2.80%, 49.94±5.58%, 74.82±3.24% and 90.82±2.88%; p<0.005 vs control, respectively). 50 μg/ml CRP stimulated CD14+ monocytes for various times (6, 12, 24 and 48 h) and increased dose-dependently the expression of TLR4 protein (32.22±2.80%, 49.94±5.58%, 74.82±3.24% and 90.82±2.88%; p<0.005 vs control, respectively). 50 μg/ml CRP stimulated CD14+ monocytes for various times (6, 12, 24 and 48 h) and increased time-dependently the expression of TLR4 protein (29.80±2.70%, 47.44±4.41%, 51.71±2.92% and 50.57±3.54%; p<0.005 vs control, respectively). CRP (5, 25, 50 and 100 μg/ml) increased dose-dependently the expression of TLR4 mRNA (159%, 211%, 320% and 390%; p<0.005 vs control, respectively). CRP (5, 25, 50 and 100 μg/ml) increased dose-dependently the expression of TLR4 mRNA (162%, 264%, 354% and 208%; p<0.005 vs control, respectively) and restrained dose-dependently the expression of TLR4 mRNA (159%, 211%, 320% and 390%; p<0.005 vs control, respectively). The release of TNFα, IL-6 and MMP-9 in the supernatants of monocytes treated with CRP 50 μg/ml was inhibited dose-dependently by atorvastatin. Atorvastatin 10 μmol/l inhibited mostly the release of TNFα, IL-6 and MMP-9 in the supernatants of monocytes treated with CRP 50 μg/ml (24%, 22.6% and 15.6%, p<0.005 vs baseline, respectively).

**Conclusion** CRP can increase dose-dependently and time-dependently the expression of TLR4 on CD14+ monocyte in human, and the production of TNFα, IL-6 and MMP-9 in CD14+ monocyte. Atorvastatin can inhibit dose-dependently the expression of TLR4 mRNA and protein induced by CRP and the release of TNFα, IL-6 and MMP-9 in CD14+ monocytes in human. Atorvastatin has anti-inflamatory effects and may restrain innate immune response in vitro by inhibition of monocyte Toll-like receptor signalling.

**e0110**  **THE EFFECTS OF ATORVASTATIN ON C-REACTIVE PROTEIN INDUCED TOLL-LIKE RECEPTOR 4 EXPRESSION ON CD14+ MONOCYTE**

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**Objective** To observe the effects of atorvastatin on C-reactive protein (CRP) induced Toll-like receptor 4 expression on CD14+ monocyte in human, and anti-inflammatory effect of atorvastatin.

**Methods** CD14+ monocytes were isolated from blood in healthy volunteers by the Ficoll density gradient and stimulated by CRP with different concentrations (5, 25, 50, 100 μg/ml) and different exposure time (50 μg/ml CRP coincubated for 6, 12, 24 and 48 h). The protein expression of TLR4 was measured by flow cytometry and mRNA expression of TLR4 and MD-2 were tested by quantitative PCR. Measurements of TNFα, IL-6 and MMP-9 in the supernatants of cultured monocytes were performed by ELISA.

**Results** CRP (5, 25, 50 and 100 μg/ml) increased dose-dependently the expression of TLR4 protein (32.22±2.80%, 49.94±5.58%, 74.82±3.24% and 90.82±2.88%; p<0.005 vs control, respectively). CRP 50 μg/ml stimulated CD14+ monocytes for various times (6, 12, 24 and 48 h) and also increased time-dependently the expression of TLR4 protein (29.80±2.70%, 47.44±4.41%, 51.71±2.92% and 50.57±3.54%; p<0.005 vs control, respectively). CRP (5, 25, 50 and 100 μg/ml) increased dose-dependently the expression of TLR4 protein (32.22±2.80%, 49.94±5.58%, 74.82±3.24% and 90.82±2.88%; p<0.005 vs control, respectively). CRP (5, 25, 50 and 100 μg/ml) increased dose-dependently the expression of TLR4 mRNA (159%, 211%, 320% and 390%; p<0.005 vs control, respectively). CRP (5, 25, 50 and 100 μg/ml) increased dose-dependently the expression of TLR4 mRNA (159%, 211%, 320% and 390%; p<0.005 vs control, respectively). The release of TNFα, IL-6 and MMP-9 in the supernatants of monocytes treated with CRP 50 μg/ml was inhibited dose-dependently by atorvastatin. Atorvastatin 10 μmol/l inhibited mostly the release of TNFα, IL-6 and MMP-9 in the supernatants of monocytes treated with CRP 50 μg/ml (24%, 22.6% and 15.6%, p<0.005 vs baseline, respectively).

**Conclusion** CRP can increase dose-dependently and time-dependently the expression of TLR4 on CD14+ monocyte in human, and the production of TNFα, IL-6 and MMP-9 in CD14+ monocyte. Atorvastatin can inhibit dose-dependently the expression of TLR4 mRNA and protein induced by CRP and the release of TNFα, IL-6 and MMP-9 in CD14+ monocytes in human. Atorvastatin has anti-inflamatory effects and may restrain innate immune response in vitro by inhibition of monocyte Toll-like receptor signalling.

**e0111**  **ASSOCIATION BETWEEN MYELOPEROXIDASE -463 G/A GENE POLYMORPHISM AND ITS PLASMA LEVELS WITH RISK OF CORONARY ARTERY DISEASE IN CHINESE POPULATION**

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**Objective** The aim of this study was to investigate whether myeloperoxidase gene polymorphism and its plasma levels were associated with risk of coronary artery disease (CAD) in Chinese population.

**Methods** A case-control study was conducted in Fujian provincial hospital, 157 patients with established CAD (cases) and 78 individuals without angiographically significant CAD (controls) were enrolled. Blood samples were collected to identify the MPO polymorphism and its plasma levels.

**Results** Genotypes were determined in all individuals. The frequencies of three genotypes were significantly different in both group (p<0.05). Plasma MPO levels were significantly greater in patients with CAD than in controls (332.05±167.56 pg/ml vs 277.81±142.68 pg/ml, p<0.05). In the case group, 7(4.5%) were homozygous for AA, 101(64.3%) for GG and 49(31.2%) were heterozygous. Mean MPO plasma levels were 200.10±31.47 pg/ml for AA, 297.43±125.28 pg/ml for AG and 367.66±177.14 pg/ml for GG genotypes. In the case group, the MPO levels with GG were significantly higher than that in individuals with GA(p<0.05) and AA(p<0.05), but with no difference between GA and AA genotype (p>0.05). Plasma MPO levels correlated with its genotype.

**Conclusion** We found association between MPO polymerase and its plasma levels with CAD risk in Chinese population. These findings provide new sights for atherosclerosis diagnosis and risk assessment.

**Acknowledgements** This work was supported by a grant from the Nature Science Foundation of Fujian Province (20060326, X0650036).

**e0112**  **STUDY OF MYELOPEROXIDASE LEVEL AND CD11B/CD18 EXPRESSIONS ON LEUKOCYTES IN PATIENTS WITH CORONARY HEART DISEASE**

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**Objective** Myeloperoxidase (MPO) and CD11b/CD18, markers of leukocyte activation, are involved in the pathogenesis of atherosclerosis. The aim of the study was to investigate the plasma MPO level and CD11b/CD18 expressions on leukocytes in patients with coronary heart disease (CHD).

**Methods** This case-control study included 157 patients with angiographically proven CHD (cases). Controls included 78 subjects with normal coronary angiograms. MPO was measured using an