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

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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 (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 CARDIAL PROTECTIVE EFFECTS OF DIFFERENT DOSAGE ATORVASTATIN IN PATIENTS WITH STABLE ANGINA AFTER PERCUTANEOUS CORONARY INTERVENTION

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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/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, 8 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 $> 3 \times$ 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 ENDOTHELIUM BY MOBILISING ENDOTHELIAL PROGENITOR CELL

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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 Variectomy 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 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 reendothelialization was calculated after 7 days. To trace EPCs in vivo, 1×10^6 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 $0.42 \pm 0.135\%$ (n=6), $1.47 \pm 0.38\%$ (n=5) (ovariectomy+E2); $0.13 \pm 0.024\%$ (n=6), $0.25 \pm 0.024\%$ (n=6) (ovariectomy); $0.43 \pm 0.16\%$ (n=6), $0.65 \pm 0.21\%$ (n=4) (non-ovariectomy); $0.12 \pm 0.019\%$ (n=6), $0.25 \pm 0.062\%$ (n=6) (ovariectomy+E2+LY) and $0.12 \pm 0.019\%$ (n=6), $0.24 \pm 0.067\%$ (n=6) (ovariectomy+E2+ICI). Area of re-endothelialization were (ovariectomy, $28.33 \pm 13.49\%$, n=5) vs (ovariectomy+E2, $69.53 \pm 14.14\%$, n=5) vs (non-ovariectomy, $83.11 \pm 7.94\%$, 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/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

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Objective 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 Ficoll density gradient and stimulated by CRP

with different concentrations (5, 25, 50, 100 µg/ml) and different exposure time (50 µg/ml CRP cocubated 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 ($32.22 \pm 2.80\%$, $49.94 \pm 5.58\%$, $74.82 \pm 3.24\%$ and $90.82 \pm 2.88\%$; $p < 0.005$ vs control, respectively). 50 µg/ml CRP stimulated CD14⁺ monocytes for various times (6, 12, 24 and 48 h) and also increased time-dependently the expression of TLR4 protein ($29.80 \pm 2.70\%$, $47.44 \pm 4.41\%$, $81.71 \pm 2.92\%$ and $50.57 \pm 3.34\%$; $p < 0.005$ 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) and MD2 mRNA (146%, 236%, 311% and 416%; $p < 0.005$ 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 mRNA (162%, 264%, 354% and 208%; $p < 0.005$ vs control, respectively) and MD2 mRNA (147%, 241%, 311% and 190%; $p < 0.005$ vs control, respectively). The release of TNFα, IL-6 and MMP-9 in the supernatants of monocytes treated with CRP increased dose-dependently. TLR4 inhibitor of high dose (30 µg/ml) could block the release of TNFα, IL-6 and MMP-9 mediated by TLR4 and MD2 upregulated by CRP completely.

Conclusion CRP can active the signal transduction of TLR4 on CD14⁺ monocyte, and induced the production of TNFα, IL-6 and MMP-9. Our finding illustrates that CRP, as pathogen associated molecular (PAMP), may induce innate immune response in vitro by 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 cocubated 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 ($32.22 \pm 2.80\%$, $49.94 \pm 5.58\%$, $74.82 \pm 3.24\%$ and $90.82 \pm 2.88\%$; $p < 0.005$ 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 ($29.80 \pm 2.70\%$, $47.44 \pm 4.41\%$, $81.71 \pm 2.92\%$ and $50.57 \pm 3.34\%$; $p < 0.005$ vs control, respectively). Atorvastatin (1.0, 2.5, 5.0, 7.5 and 10 µmol/l) inhibited dose-dependently the expression of TLR4 protein induced by CRP 50 µg/ml for 24 h [68.17%, 52.43%, 27.72%, 17.46% and 9.99%; $p < 0.005$ vs control (80.39%), respectively], and restrained dose-dependently the expression of TLR4 mRNA ($p < 0.005$ vs control, respectively) and MD2 mRNA ($p < 0.005$ 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-inflammatory 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.

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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