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±3.34%; 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). 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.

**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.

**GWICC Abstracts 2010**

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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 co-incubated 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±3.34%; 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 (30.39%, 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±51.47 pg/ml for AA, 291.43±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 polymers 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