density gradient centrifugation. The capacity of HDL to delay LDL oxidation was determined by conjugated diene method, while HDL antimigration on monocyte was examined by Transwell cell culture.

Results Compared with continue-smoking group, plasma CRP and MMP-9 in CHD patients were significantly decreased in smoking cessation group (CRP: 12.576 ng/ml vs 14.794 ng/ml, $p=0.041$; MMP-9: 2.307 ng/ml vs 3.469 ng/ml, $p=0.015$), however, IL-6 and PON-1 were not different (IL-6: 5.848 pg/ml vs 5.910 pg/ml, $p=0.68$; PON-1: 0.17 U/ μ l vs 0.16 U/ μ l, $p=0.42$). Oxidation lag time and oxidation rate peaking time that reflect the ability of HDL to delay LDL oxidation were significantly prolonged (Oxidation lag time: 151.36 min vs 126.65 min, $p=0.028$; oxidation rate peaking time: 192.25 min vs 170.58 min, $p=0.037$). Monocyte migration and chemotaxis were obviously reduced by HDL (33 vs 46, $p<0.01$).

Conclusion Smoking cessation for CHD patients reduces inflammatory response as well as enhances the antioxidation and anti-inflammation capacity of HDL.

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EFFECTS OF SMOKING CESSATION ON HDL FUNCTION IN CORONARY HEART DISEASE PATIENTS

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Aim Smoking cigarette enhances the development of atherosclerosis, whereas smoking cessation could reduce cardiovascular events in CHD patients. Our study intended to clarify the effects of smoking cessation on inflammatory response and high density lipoprotein function in CHD patients.

Methods Thirty six male diagnosed CHD patients with smoking history at least 10 years were recruited, while 36 healthy male smokers were selected as controls. These two groups of subjects were randomised into continue-smoking subgroup and smoking cessation subgroup, and followed up for 90 days. Plasma PON-1 activity, plasma content of MMP-9, CRP and IL-6 were detected by ELISA. Plasma HDL was separated by