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A NOVEL VWF A3-GPI MODIFIED EPCS TO ENHANCE ITS ADHESION ABILITY TO DAMAGED VESSEL SEGMENT-COLLEGEN

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Tan Hu, Huang lan. Department of Internal Cardiology of Xin Qiao Hospital, the Third Military Medical University, ChongQing in China

Objectives This study was designed to investigate the relationship between heme oxygenase (HO)/carbon monoxide (CO) system and nitric oxide synthase (NOS)/nitric monoxide (NO) system in diet-induced atherosclerosis.
Methods The rabbits serving as animal models were divided into six groups. The normal control group (n=8) were fed with normal sterile diet while the Cholesterol group (n=8) were fed with 1% cholesterol diet. The other four groups (L-arg group, L-NAME group, Hame group and ZnPP group; n=8) were fed with 1% cholesterol diet plus L-arginine (L-arg), NG-Nitro-L-arginine Methyl Ester (L-NAME), hame-L-lysinate and zinc protoporphyrin IX (ZnPP-IX) respectively. The treatment lasted for 10 weeks.

Results Aortic plaques area in chol group was 40.2±8.9%. The decrease of aortic NO production and NOS expression was associated with increase of CO production (p<0.01) and HO-1 activity (p<0.01) in chol group when compared with controls. Aortic plaque areas reduced distinctly (26.6±9.2%) as well as the up-regulation of aortic CO production (p<0.01) and HO-1 activity (p<0.01) was significant when hame group was compared to chol group. On the other hand, in comparison with controls, NOS expression and NO production in hame group decreased significantly (p<0.01), but no apparent difference was drew between chol group and hame group. When Compared with chol group, aortic cNOS activity and NO production increased obviously and aortic plaques area (28.1±7.7%) was greatly reduced (p<0.01) in L-arg group. Reversely, HO-1 expression and CO production in L-arg group decreased distinctly when compared with those in control group, but the results in L-arg group were similar to those in chol group. The aortic c-myc and c-fos expressions in both hame group and L-arg group reduced significantly when compared with those in chol group, while they were similar to those in ZnPP group and L-NAME group.

Conclusions Our study demonstrates that HO/CO system can attenuate the development of atherosclerosis through its regulation and compensation to the NOS/NO systems in diet-induced atherosclerosis.