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

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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.