·研究论文摘要·

[文章编号] 1007-3949(2011)19-03-0276-01

The Case-Control Study Between DDAH2 Polymorphism and Coronary Heart Disease

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[**KEY WORDS**] Dimethylargininase; Genetics; ADMA; Polymorphism; Coronary Heart Disease; Risk Factors

Background and Aim Asymmetric dimethylarginine (ADMA), an endogenous inhibitor of nitric oxide synthase (NOS), has been shown to be an independent predictor of coronary heart disease (CHD). Dimethylarginine dimethylaminohydrolase-2 (DDAH2) promotes the metabolism of ADMA and plays a key role in formation of the atherosclerosis. We hypothesized that genetic variation in DDAH2 gene might alter the susceptibility to CHD. **Methods** We tested our hypothesis in a case-control studies. We used a haplotype-tagging single nucleotide polymorphisms (SNP) approach to identify tag SNP in DDAH2. The SNP were genotyped by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) and ligase detection reaction (LDR)-sequencing in 1650 patients with CHD and 1920 control subjects. **Results** A promoter variant -449C/G (rs805305) and -1415G/A (rs2272592) in DDAH2 was identified in the region containing DDAH2. The frequency of those polymorphism were consistent with the law of Hardy-Weinberg. The frequency of rs805305 CG + GG or G allele was not significantly different between CHD and wild-type genotype (OR: 0.667, 95% CI: 0.374 to 1.187, $P > 0.05$). The frequency of rs2272592 GA + AA or A allele also showed no significant difference between CHD and wild-type genotype (OR: 1.420, 95% CI: 0.899 to 2.242, $P > 0.05$). No association was observed between the DDAH2 variant and CHD. These results was independent of age, gender, hypertension, diabetes and hyperlipidemia. **Conclusions** Our results suggest that although DDAH2/ADMA pathway acts as a critical regulator of coronary atherosclerotic heart disease, the DDAH2 common variant may not predict the susceptibility to CHD in Chinese population.

(Edited by XU Xue-Mei)