INTERACTIONS OF SEVERAL GENETIC POLYMORPHISMS AND CIGARETTE SMOKING ON BLOOD PRESSURE LEVELS

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Objectives Although the relationship between cigarette smoking and cardiovascular diseases is well established, the interactions of some single nucleotide polymorphisms (SNPs) and cigarette smoking on blood pressure levels are still limited. The present study was undertaken to detect nine SNPs in different lipid-related genes and their interactions with cigarette smoking on blood pressure levels in the Bai Ku Yao, an isolated subgroup of the Yao minority in China.

Methods Genotyping of ATP-binding cassette transporter A1 (ABCA-1) V825I (rs2066715), acyl-CoA:cholesterol acyltransferase-1 (ACAT-1) rs1044925, low density lipoprotein receptor (LDL-R) Ava II, hepatic lipase gene (LIPC) −250G>A (rs2070895), endothelial lipase gene (LIPC) 584C>T (rs2000813), methylenetetrahydrofolate reductase (MTHFR) 677C>T (rs1801133), proprotein convertase subtilisin-like kexin type 9 (PCSK9) E670G (rs505151), peroxisome proliferator-activated receptor delta (PPARD) +294T>C (rs2016520) and Scavenger receptor class B type 1 (SCARB1) rs5888 was performed using PCR and restriction fragment length polymorphism in 935 non-smokers and 845 smokers. The interactions of nine SNPs and cigarette smoking on blood pressure levels were detected by factorial regression analysis after controlling for potential confounders.

Results The genotypic frequencies of ACAT-1 and LIPG, the allelic frequencies of ABCA-1, and the genotypic and allelic frequencies of LDL-R, LIPC, PPARD and SCARB1 were different between non-smokers and smokers (p<0.05–0.001). The levels of pulse pressure (PP) among the genotypes of ABCA-1, and the levels of systolic blood pressure (SBP), diastolic blood pressure (DBP) and PP among the genotypes of LIPC were different in non-smokers (p<0.05–0.001). The levels of SBP among the genotypes of ABCA-1, ACAT-1, LIPG and PCSK9, the levels of DBP among the genotypes of ACAT-1, LDL-R, LIPC, PCSK9 and PPARD, and the levels of PP among the genotypes of LIPC, LIPG, MTHFR and PCSK9 were different in smokers (p<0.05–0.001). The SNPs of ABCA-1, ACAT-1 and PCSK9 were shown interactions with cigarette smoking on SBP levels (p<0.05–0.001), the SNPs of ACAT-1, LDL-R, MTHFR and PCSK9 were shown interactions with cigarette smoking on DBP levels (p<0.05–0.01), and the SNPs of ABCA-1, LIPC, PCSK9 and PPARD were shown interactions with cigarette smoking on PP levels (p<0.05–0.01). Multiple linear regression analysis was also shown that blood pressure levels were associated with the genotypes and/or alleles of several SNPs in the non-smokers and smokers (p<0.05–0.001).

Conclusions The differences in blood pressure levels between the non-smokers and smokers might partly result from different interactions of several SNPs and cigarette smoking.