Original Article

Association of the adiponectin gene (*ADIPOQ*) +45 T > G polymorphism with the metabolic syndrome among Han Chinese in Sichuan province of China

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The metabolic syndrome is a cluster of abnormalities characterized by obesity, impaired glucose metabolism, hypertension or elevated blood pressure, and dyslipidemia. The aim of this study was to investigate the association of the adiponectin gene (*ADIPOQ*) +45 T > G polymorphism with the metabolic syndrome among Han Chinese in Sichuan province of China. A case-control design was used including 116 patients with the metabolic syndrome and 108 unrelated controls, matched on age and gender. The *ADIPOQ* +45G allele (TG+GG) had a significant association with risk of the metabolic syndrome (odds ratio=1.88, 95% confidence interval: 1.03-3.44, p=0.039) adjusted for education, physical activity, family history of related diseases, smoking and drinking, compared with subjects with TT genotype. The association between the *ADIPOQ* +45 T>G polymorphism and the metabolic syndrome was independent of multiple confounders.

Key Words: the metabolic syndrome, *ADIPOQ*, polymorphism, case-control study, China

INTRODUCTION

The metabolic syndrome (MS) is a cluster of physiological and biochemical abnormalities, including four major components: obesity, especially central obesity; impaired glucose metabolism such as type 2 diabetes mellitus (T2DM), impaired fasting glucose, or impaired glucose tolerance (IGT); hypertension or elevated blood pressure; and dyslipidemia with hypertriglyceridemia and/or hypoalphalipoproteinemia.[1] MS is associated with the development of T2DM and of cardiovascular disease (CVD),[2] as well as with an increased all-cause mortality.[3]

Adiponectin is a relatively abundant serum protein secreted by adipocytes.[4] Plasma levels of adiponectin is lower in people with obesity,[5] hypertension,[6] T2DM and coronary heart disease (CHD),[7] compared to those in healthy subjects. Some studies showed that the level of adiponectin is correlated with insulin resistance,[8] and with glucose and lipid metabolism.[9] Furthermore, adiponectin has been reported to be independently associated with MS.[10,11]

The adiponectin gene (*ADIPOQ)* is located on chromosome 3q27, it consists of three exons and two introns,[12] and has been linked to CHD,[13] T2DM[14] and MS.[15] The +45 T > G polymorphism is one of the most common variants of the exon 2 of the *ADIPOQ* gene and it is reported to be associated with serum levels of adiponectin, insulin sensitivity, obesity and T2DM.[16-18] However, whether the +45 T > G polymorphism is associated with MS still remains unclear.[16, 19]

Previous studies showed a low prevalence of hypertension among Yi farmers[20,21] in remote mountainous areas of southwestern China where the largest Yi community is located. In addition, Wang *et al* [22] reported that the age- and sex-adjusted prevalence of MS was 9.9 times higher in Yi migrants (23.8%) and 6.3 times higher in Han peo-

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Manuscript received 16 May 2011. Initial review completed 15 August 2011. Revision accepted 26 September 2011.

ple (15.2%) than in Yi farmers (2.4%) and assumed, therefore, lifestyle strongly influenced development of MS in Han and Yi Chinese. In addition, it is well known that MS is also influenced by genetic factors. This study was therefore designed to examine the possible association of the *ADIPOQ* +45 T > G polymorphism with MS among Han Chinese in Sichuan province of China to further explore the mechanisms that may affect MS in Han Chinese.

MATERIALS AND METHODS

Participants classification and definition

A case-control study comprising of 224 Han Chinese aged over 20 years living in Xichang City, southwestern China was carried out based on a cross-sectional survey from 2007 to 2008.[22] Both parents of the study participant should be of Han ethnicity, and inhabits of the city for more than five years. The participants included 116 patients of MS and 108 unrelated controls matched with regard to age (± 3 years) and gender.

According to the Chinese Diabetes Society in 2004,[23] individuals satisfying three or more of the following components were diagnosed as having the MS: (1) body mass index (BMI) ≥25.0 kg/m^2; (2) serum levels of triglyceride ≥150 mg/dL (1.7 mmol/L) and/or serum levels of high-density lipoprotein-cholesterol (HDL-C) < 35 mg/dL (0.9 mmol/L) in males and <39 mg/dL (1.0 mmol/L) in females; (3) blood pressure ≥140/90 mmHg and/or history of treatment for previously diagnosed hypertension; (4) fasting plasma glucose (FPG) ≥110 mg/dL (6.1 mmol/L) and/or two-hour plasma glucose (2hPG) ≥ 140 mg/dL (7.8 mmol/L) after meals and/or history of previously diagnosed T2DM.

This study was approved by the institutional review board of the Institute of Basic Medical Sciences, Chinese Academy of Medical Sciences, Beijing. Informed consent was obtained from all participants.

Measurements

To evaluate demographic, socioeconomic and lifestyle information, the participants were interviewed by local trained physicians with a questionnaire that collected data including age, gender, ethnicity, education, occupation, family history of related diseases, smoking, drinking, as well as information related to diagnosis and treatment for hypertension and diabetes. Education for this study was categorized into three levels, ie, illiteracy or primary school, high school and university. Physical activity was categorized based on the participant's occupation, as light, moderate or heavy, respectively. Family history of related diseases was categorized as any of their biological parents or siblings with hypertension, diabetes or dyslipidemia. Smoking was defined as never smoker, or ever smoker if the subject was a current smoker or a former smoker. Drinking was defined as never drinking alcoholic beverages, or ever drinker if the subject was a current drinker or a former drinker.

Blood pressure was measured three consecutive readings after a 10-min resting period in a sitting position to the nearest 2 mmHg on the right arm using a standard mercury sphygmomanometer. Both body height and weight were measured with the participants in light clothing and without shoes. Obesity was assessed by body mass index (BMI, as weight in kilograms divided by the square of height in meters).

Biochemical analysis

Venous blood was obtained after overnight fasting, and processed at the examination center and then shipped to a central clinical laboratory in Beijing where all the samples were kept at -40℃ before analysis. FPG level was measured by the glucose oxidase method. Both of HDL-C and triglyceride were measured by enzymatic methods. All biochemical analyses were performed using Hitachi 7600 automatic analyzer (Boehringer Mannheim, Mannheim, Germany).

Genotyping of the ADIPOQ +45 T > G polymorphism

The *ADIPOQ* +45 T > G polymorphism (rs2241766) was genotyped by polymerase chain reaction (PCR) and restriction fragment length polymorphism (PCR-RFLP). The polymorphism was amplified using the following primers: forward, 5' -GCAGCTCCTAGAAGTAGAC TCTGCTG -3' and reverse, 5' -GCAGGTCTGTGAT GAAAGAGGCC -3' (Yingjun Biotech Co Ltd, Shanghai, China). Polymerase chain reaction was performed in a total volume of 25 μl mixture containing 2.0 μl DNA, 0.5 μl forward primer, 0.5 μl reverse primer, 0.7 μl deoxynucleotide triphosphate (dNTP), 1.8 μl $MgCl_2$, 2.5μl 10×buffer, 0.5μl of Taq DNA polymerase and 16.5 μl of ddH_2O. The amplification conditions were as follows: 94℃ for 5 min, followed by 35 cycles of 1 min at 94℃, 1 min at 59℃ and 1 min at 72℃, and ending with a single 5 min extension step at 72℃. The PCR products were run on 2% agarose gels electrophoresis to evaluate overall amplification efficiency. Then they were digested with the enzyme SmaI (BioLabs Inc., New England, U.S.) at 25℃ for 4 hours. Reaction mixture contained the PCR products 10 μl, 0.3μl of SmaI, 2μl 10×buffer and 8μl ddH_2O. The digestion products were resolved by 3% agarose gel electrophoresis and viewed under an UV transilluminator. The TT homozygote yielded the 372 bp uncut fragment only, the TG heterozygote yielded the 372, 209 and 163bp fragments, while the GG homozygote yielded the 209 and 163bp fragments.

Statistical analysis

Continuous variables are presented as means ± standard deviation (SD) and difference between the cases and controls was compared using Student's *t*-test for normally-distributed variables or non-parametric Mann-Whitney *U*-test for non-normally-distributed ones. Test for Hardy-Weinberg equilibrium and comparison of allele and genotype distribution between MS cases and controls were assessed by chi-square test. Odds ratios (OR) and their 95% confidence intervals (CI) were calculated using the Mantel-Haenszel method. Multiple logistic regression analysis was performed to estimate an association of the +45 T > G polymorphism with the risk of MS. A p value less than 0.05 was considered statistically significant. All data analyses were performed using SAS version 9.2 software (SAS Institute Inc., Cary, NC, USA).

RESULTS

Demographic characteristics of 116 MS cases and 108 controls are shown in Table 1. Mean age of the cases and controls were 51.6±11.6 years and 51.8±11.5 years, respectively (p=0.890). The cases had significantly higher BMI, systolic blood pressure, diastolic blood pressure, triglyceride and total cholesterol, but lower HDL-C than those in the controls. No significant differences in FPG and low-density lipoprotein-cholesterol (LDL-C) between the two groups were found ($p > 0.05$).

As shown in Table 2, frequencies of the TT, TG and GG genotypes were 61.2%, 34.5% and 4.3% in the cases, and 70.4%, 25.9% and 3.7% in the controls, respectively. Because only five cases and four controls were homozygous for the rare G-allele, we combined them with those having the TG genotype in all statistical analysis. Compared with TT, there was no difference in distribution of the TG, GG and TG+GG genotypic frequencies between cases and controls. In addition, no difference was observed in frequency of allele between the two groups. Frequencies of the genotype were in Hardy-Weinberg equilibrium in both groups.

Results of multiple logistic regression analysis showed that the *ADIPOQ* +45 T > G polymorphism was an independent risk factor for MS (Figure 1). After adjustment for education, physical activity, family history of related diseases, smoking and drinking, carriers of the G allele (TG+GG) had a 1.88-fold higher risk for developing MS (OR=1.88, 95% CI: 1.03-3.44, p=0.039) than subjects with TT genotype.

DISCUSSION

This study showed that the *ADIPOQ* +45 T > G polymorphism was independently associated with MS, and that the G allele had a corresponding significant association with the MS.

MS is a complex disorder and its main pathophysiological mechanism is mediated by insulin resistance and central obesity.[24,25] Insulin resistance is considered to be at the core of the syndrome, while central obesity is its most prevalent clinical manifestation.[26] Both genetic and environmental factors contribute to the development of MS.[27,28] Some studies have provided evidence that the *ADIPOQ* +45 T>G polymorphism is associated with MS components.[16-18] However, a number of studies have mainly focused on the association of the polymorphism with one or two components of MS, such as obesity or/and T2DM. To our knowledge, a limited number of published work has been found on association of the +45 T > G polymorphism with MS. In addition, environmental factors such as smoking, drinking, physical activity and family income might have predominantly contributed to the progression of MS.[22,27] Therefore, a case-control study was designed to adjust confounding effect of environmental factors on MS.

Age and gender which are independently associated with MS may confound an association of the *ADIPOQ* +45 T > G polymorphism with MS.[22] Prevalence of MS varies with gender and increases with increasing age.[22,29] Besides, diagnostic criteria for MS for men and women are different. Therefore, a case-control design is needed

Table1. Demographic characteristics of the study participants †

Characteristics	Cases (n=116)	Controls (n=108)	p-value
Age (years)	51.6±11.6	51.8±11.5	0.890
Body mass index (kg/m^2)	27.2±2.21	21.5±2.69	<.0001
SBP (mmHg)	144±19.1	112±12.0	<.0001
DBP (mmHg)	93.0±12.2	74.4±8.60	<.0001
FPG (mmol/L)	6.34±3.12	5.59±1.77	0.198
Triglyceride (mmol/L)	3.41±4.06	1.15±0.67	<.0001
Total choldesterol (mmol/L)	5.03±0.99	4.45±0.75	<.0001
HDL-C (mmol/L)	0.95±0.24	1.31±0.29	<.0001
LDL-C (mmol/L)	2.54±1.55	2.62±0.64	0.336

† Data indicated as mean ± standard deviation. SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol.

Table2. Distribution of the frequencies of the genotype, combined genotype and allele between cases and controls

Genotype/allele	Cases (n=116) n (%)	Controls (n=108) n (%)	p-value	Odds ratio	95% confidence interval
TT	71 (61.2)	76 (70.4)		1.00	
TG	40 (34.5)	28 (25.9)		1.53†	0.86-2.74
GG	5 (4.3)	4 (3.7)	0.349 *	1.34†	0.35-5.18
TG+GG	45 (38.8)	32 (29.6)	0.149 **	1.51‡	0.86-2.63
T	182 (78.4)	180 (83.3)		1.00	
G	50 (21.6)	36 (16.7)	0.190 ***	1.37§	0.85-2.21

p-value * for the difference in the frequencies of TT, TG and GG genotype between cases and controls
p-value ** for the difference in the frequencies of TT and TG+GG genotype between cases and controls
p-value *** for the difference in the frequencies of the T and G allele between cases and controls
OR†: TG and GG vs. TT
OR‡: TG+GG vs. TT
OR§: G vs. T

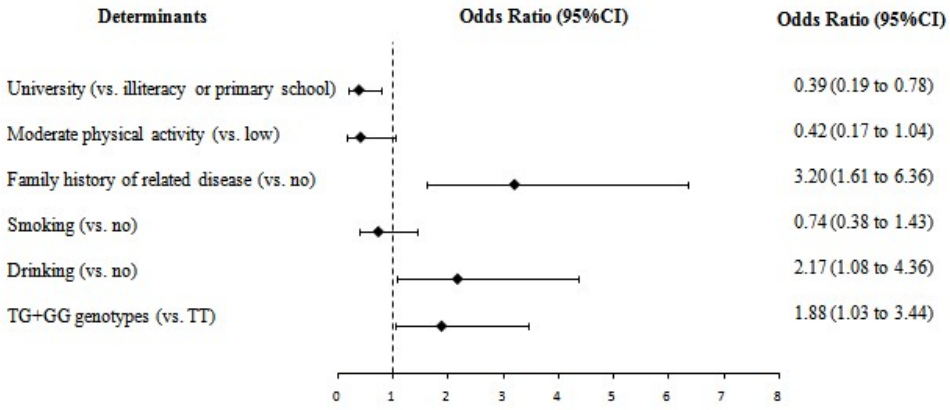

Figure1. Factors related to MS by multiple logistic regression, Han Chinese, China

to match age and gender between MS cases and the controls.

Heid *et al* [19] reported that frequencies of the TG and GG genotypes were 21% and 1%, respectively in healthy Caucasian population. We found that frequencies of TG and GG genotypes were 30% and 4%, respectively in Han Chinese (data not shown). Furthermore, the +45 T > G mutation showed higher frequencies of TG and GG genotypes (41 and 6%, respectively) among Han Chinese in eastern China, compared with our study.[30] This implies that the +45 T > G mutation rate had a different distribution among different ethnic and regional populations. Therefore, case-control studies on an association of the genotypes with MS among different populations may show inconsistent results.

In the present study, after adjusting for environmental factors that may confound expression of the phenotype, such as education, physical activity, family history of related diseases, smoking and drinking, our results showed that the G-allele carriers had an approximately two-fold higher risk for an association with MS in cases than that in controls. This finding was consistent with that found in some of the previous studies. The 45G allele had a significantly increased risk for obesity and reduced insulin sensitivity in Germans,[31] and an increased risk for T2DM in the Japanese[18] and other populations in the STOP-NIDDM trial.[17] However, there are some inconsistent findings showing that the 45G allele appeared to have a protective role on MS, whereas, +45T allele is likely to be a risk factor. The +45T allele is a risk factor for developing obesity and insulin resistance syndrome in Italians.[16,32] Similar results have been reported in populations from Taiwan[33] and Sweden,[34] respectively.

These inconsistent results may depend on several causes. First of all, the *ADIPOQ* gene is only one gene regulating the traits of MS. Moreover, MS is not only associated with genetic factors, but also with environmental factors. Dietary intake is recognized as an important environmental factor in affecting predisposition to MS, and its effect on adiponectin concentrations has been reported in a previous study.[35] In addition, some studies reported that an interaction between genetic predisposition and dietary fat intake may contribute to the development of MS.[36,37] We, therefore, assume that the effect of a single gene variant on MS will be weak.

Some limitations of this study should be interpreted. Firstly, the study has a case-control design, therefore we can't affirm that after the investigation some control subjects did not develop MS. Secondly, plasma level of adiponectin was not measured and an association of adiponectin level with the *ADIPOQ* +45 T>G polymorphism and MS couldn't be analyzed. Thirdly, only one variant within the *ADIPOQ* gene was explored for its association with MS, and not all of the single nucleotide polymorphisms (SNPs) reported in the literature. Therefore, we can't explore the effect of gene-gene interactions on MS. In addition, no detailed information about dietary components was available in this study population, so the effect of gene-diet interactions on the MS can't be identified.

In conclusion, the *ADIPOQ* +45 T>G polymorphism is independently associated with MS, and the G allele has a significant association with the risk of MS among Han Chinese. It is implied that the *ADIPOQ* +45 T>G polymorphism may be a genetic contributor to MS.

ACKNOWLEDGMENTS

This study was supported by a grant (No 30671811) from the National Natural Science Foundation of China. We wish to express our sincere appreciation to all of the staff members of the Liangshan Yi Autonomous Prefectural Center for Disease Control and Prevention, for their help in performing fieldwork. Thanks to all of the study participants.

AUTHOR DISCLOSURES

All authors have no conflicts of interest to declare.

REFERENCES

1. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2005;365:1415-28.
2. Wilson PW, D'Agostino RB, Parise H, Sullivan L, Meigs JB. Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus. Circulation. 2005;112: 3066-72.
3. Trevisan M, Liu J, Bahsas FB, Menotti A. Syndrome X and mortality: a population-based study. Risk Factor and Life Expectancy Research Group. Am J Epidemiol. 1998;148: 958-66.
4. Scherer PE, Williams S, Fogliano M, Baldini G, Lodish HF. A novel serum protein similar to C1q, produced exclusively in adipocytes. J Biol Chem. 1995;270:26746-9.
5. Arita Y, Kihara S, Ouchi N, Takahashi M, Maeda K, Miyagawa J et al. Paradoxical decrease of an adipose-specific

protein, adiponectin, in obesity. Biochem Biophys Res Commun. 1999;257:79-83.
6. Lee HS, Lee M, Joung H. Adiponectin represents an independent risk factor for hypertension in middle aged Korean women. Asia Pac J Clin Nutr. 2007;16:10-5.
7. Koenig W, Khuseyinova N, Baumert J, Meisinger C, Lowel H. Serum concentrations of adiponectin and risk of type 2 diabetes mellitus and coronary heart disease in apparently healthy middle-aged men: results from the 18-year follow-up of a large cohort from southern Germany. J Am Coll Cardiol. 2006;48:1369-77.
8. Berg AH, Combs TP, Du X, Brownlee M, Scherer PE. The adipocyte-secreted protein Acrp30 enhances hepatic insulin action. Nat Med. 2001;7:947-53.
9. Tschritter O, Fritsche A, Thamer C, Haap M, Shirkavand F, Rahe S et al. Plasma adiponectin concentrations predict insulin sensitivity of both glucose and lipid metabolism. Diabetes. 2003;52:239-43.
10. Mi J, Munkonda MN, Li M, Zhang MX, Zhao XY, Fouejeu PC, Cianflone K. Adiponectin and leptin metabolic biomarkers in chinese children and adolescents. J Obes. 2010; 2010:892081.
11. Yu D, Yu Z, Sun Q, Sun L, Li H, Song J et al. Effects of body fat on the associations of high-molecular-weight adiponectin, leptin and soluble leptin receptor with metabolic syndrome in Chinese. PLoS One. 2011;6:e16818.
12. Takahashi M, Arita Y, Yamagata K, Matsukawa Y, Okutomi K, Horie M et al. Genomic structure and mutations in adipose-specific gene, adiponectin. Int J Obes Relat Metab Disord. 2000;24:861-8.
13. Francke S, Manraj M, Lacquemant C, Lecoeur C, Lepretre F, Passa P et al. A genome-wide scan for coronary heart disease suggests in Indo-Mauritians a susceptibility locus on chromosome 16p13 and replicates linkage with the metabolic syndrome on 3q27. Hum Mol Genet. 2001;10:2751-65.
14. Vionnet N, Hani EH, Dupont S, Gallina S, Francke S, Dotte S et al. Genomewide search for type 2 diabetes-susceptibility genes in French whites: evidence for a novel susceptibility locus for early-onset diabetes on chromosome 3q27-qter and independent replication of a type 2-diabetes locus on chromosome 1q21-q24. Am J Hum Genet. 2000; 67:1470-80.
15. Kissebah AH, Sonnenberg GE, Myklebust J, Goldstein M, Broman K, James RG et al. Quantitative trait loci on chromosomes 3 and 17 influence phenotypes of the metabolic syndrome. Proc Natl Acad Sci U S A. 2000;97:14478-83.
16. Menzaghi C, Ercolino T, Di Paola R, Berg AH, Warram JH, Scherer PE, Trischitta V, Doria A. A haplotype at the adiponectin locus is associated with obesity and other features of the insulin resistance syndrome. Diabetes. 2002;51:2306-12.
17. Zacharova J, Chiasson JL, Laakso M. The common polymorphisms (single nucleotide polymorphism [SNP] +45 and SNP +276) of the adiponectin gene predict the conversion from impaired glucose tolerance to type 2 diabetes: the STOP-NIDDM trial. Diabetes. 2005;54:893-9.
18. Hara K, Boutin P, Mori Y, Tobe K, Dina C, Yasuda K et al. Genetic variation in the gene encoding adiponectin is associated with an increased risk of type 2 diabetes in the Japanese population. Diabetes. 2002;51:536-40.
19. Heid IM, Wagner SA, Gohlke H, Iglseder B, Mueller JC, Cip P et al. Genetic architecture of the APM1 gene and its influence on adiponectin plasma levels and parameters of the metabolic syndrome in 1,727 healthy Caucasians. Diabetes. 2006;55:375-84.
20. He J, Klag MJ, Whelton PK, Chen JY, Mo JP, Qian MC, Mo PS, He GQ. Migration, blood pressure pattern, and hypertension: the Yi Migrant Study. Am J Epidemiol. 1991; 134:1085-101.
21. He J, Tell GS, Tang YC, Mo PS, He GQ. Effect of migration on blood pressure: the Yi People Study. Epidemiology. 1991;2:88-97.
22. Wang C, Wei D, Wang B, Zhang J, Zhang K, Ma M et al. Effect of lifestyle on the prevalence of the metabolic syndrome among farmers, migrants with Yi ethnicity and Han population in Sichuan province of China. Asia Pac J Clin Nutr. 2010;19:266-73.
23. Metabolic Syndrome Research Cooperation Group of Chinese Medical Association Diabetes Branch. Recommendation of metabolic syndrome by Chinese Medical Association Diabetes Branch. CJD. 2004;12:156-61.
24. Reaven GM. Pathophysiology of insulin resistance in human disease. Physiol Rev. 1995;75:473-86.
25. Shirai K. Obesity as the core of the metabolic syndrome and the management of coronary heart disease. Curr Med Res Opin. 2004;20:295-304.
26. Alberti KG, Zimmet P, Shaw J. The metabolic syndrome--a new worldwide definition. Lancet. 2005;366:1059-62.
27. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. Arch Intern Med. 2003;163:427-36.
28. Szabo DEF, Goumidi L, Bertrais S, Phillips C, Macmanus R, Roche H, Planells R, Lairon D. Prediction of the metabolic syndrome status based on dietary and genetic parameters, using Random Forest. Genes Nutr. 2008;3:173-6.
29. Shi Z, Hu X, Yuan B, Hu G, Pan X, Holmboe-Ottesen G. Coexistence of anaemia and the metabolic syndrome in adults in Jiangsu, China. Asia Pac J Clin Nutr. 2008;17:505-13.
30. Fengzhen H. The association of adiponectin gene SNP+45,SNP+276 with metabolic syndrome and plasma adiponectin concentration. Shandong: Shandong University; 2008.
31. Stumvoll M, Tschritter O, Fritsche A, Staiger H, Renn W, Weisser M, Machicao F, Haring H. Association of the T-G polymorphism in adiponectin (exon 2) with obesity and insulin sensitivity: interaction with family history of type 2 diabetes. Diabetes. 2002;51:37-41.
32. Ronconi V, Turchi F, Rilli S, Di Mattia D, Agostinelli L, Boscaro M, Giacchetti G. Metabolic syndrome in primary aldosteronism and essential hypertension: relationship to adiponectin gene variants. Nutr Metab Cardiovasc Dis. 2010;20:93-100.
33. Yang WS, Tsou PL, Lee WJ, Tseng DL, Chen CL, Peng CC et al. Allele-specific differential expression of a common adiponectin gene polymorphism related to obesity. J Mol Med. 2003;81:428-34.
34. Ukkola O, Ravussin E, Jacobson P, Sjostrom L, Bouchard C. Mutations in the adiponectin gene in lean and obese subjects from the Swedish obese subjects cohort. Metabolism. 2003; 52:881-4.
35. Qi L, Rimm E, Liu S, Rifai N, Hu FB. Dietary glycemic index, glycemic load, cereal fiber, and plasma adiponectin concentration in diabetic men. Diabetes Care. 2005;28: 1022-8.
36. Warodomwichit D, Shen J, Arnett DK, Tsai MY, Kabagambe EK, Peacock JM et al. ADIPOQ polymorphisms, monounsaturated fatty acids, and obesity risk: the GOLDN study. Obesity (Silver Spring). 2009;17:510-7.
37. Ntalla I, Dedoussis G, Yannakoulia M, Smart MC, Louizou E, Sakka SD, Papoutsakis C, Talmud PJ. ADIPOQ gene-polymorphism rs1501299 interacts with fibre intake to af-

fect adiponectin concentration in children: the GENe-Diet Attica Investigation on childhood obesity. Eur J Nutr. 2009;48:493-7

Original Article

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中國四川省漢族居民脂聯素基因+45 T > G 多態性與代謝綜合征的關聯

代謝綜合征是多種異常組分集聚在同一個體的臨床綜合征，異常組分主要包括肥胖、糖代謝受損、高血壓或血壓升高以及血脂紊亂。本研究的目的是探討中國四川省漢族居民脂聯素基因+45 T > G 多態性與代謝綜合征的關聯。應用病例-對照研究的方法，以確診的 116 例代謝綜合征患者為病例組，按照年齡和性別進行頻數匹配，選取 108 例非代謝綜合征且無血緣關係者作為對照組。研究結果顯示，在調整了教育程度、體力勞動強度、相關疾病家族史、吸煙和飲酒情況後，與攜帶 TT 基因型的人相比，脂聯素基因第 45 位的 G 等位基因(TG+GG)是代謝綜合征的危險因子(OR=1.88，95%CI=1.03-3.44，*p*=0.039)。此結果表明，控制干擾因素後，脂聯素基因+45 T > G 多態性仍是代謝綜合征的獨立危險因素。

關鍵字：代謝綜合征、脂聯素基因、基因多態性、病例-對照研究、中國