Perilipin Gene 1237 T>C Polymorphism is not Associated with Obesity Risk in Northern Chinese Han Adults[1]

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Objective To identify the association between *PLIN* 1237 polymorphism and obesity in Chinese Han adults. **Methods** A total of 994 adults (157 obese subjects, 322 overweight subjects, and 515 normal controls) were recruited from two rural communities. *PLIN* 1237 polymorphism was genotyped by polymerase chain reaction–restriction-fragment-length-polymorphism (PCR-RFLP). Association between *PLIN* polymorphisms and obesity status was estimated by ordinal logistic regression. **Results** The three genotypes of *PLIN* 1237 were detected with a percentage of 54.3%, 37.1%, and 8.6% in TT, TC, and CC genotypes, respectively. For the *PLIN* 1237 polymorphism locus, the frequency of alleles T and C was 0.73 and 0.27, respectively. The *PLIN* 1237 polymorphisms were in Hardy-Weinberg equilibrium. *PLIN* 1237 polymorphism was not associated with obesity. The odds ratio for overweight or obesity for the CC+TC genotype was 0.8 (0.4, 1.4) in women (P=0.4) and 0.6 (0.3, 1.3) in men (P=0.2) after adjustment for age, education, household income and alcohol consumption, smoking, and physical activity. **Conclusion** Chinese Han adults have a lower frequency of variant-allele C in *PLIN* 1237. *PLIN* 1237 T>C polymorphism is not significantly associated with obesity in northern Chinese adults.

Key words: Perilipin; Polymorphism; Obesity

INTRODUCTION

It is well known that genetic variations contribute more to the current epidemic of obesity, which is associated with a variety of adverse health outcomes[1-3]. Perilipin coats intracellular lipid droplets and modulates the turnover of stored fat[4-9]. Recent studies showed that perilipin gene (*PLIN*) plays a critical role in the regulation of triacylglycerol deposition and mobilization[5,10]. Several *PLIN* single-nucleotide-polymorphisms (SNPs) are associated with obesity or its related clinical traits[11-17]. The absence of *PLIN* in mice can result in lean phenotypes and reversed obesity[4,18]. Furthermore, elevated expression of *PLIN* has been reported in adipose tissue of obese subjects[16-17]. There is evidence that *PLIN* might be a candidate gene for human obesity.

PLIN 1237 T>C polymorphisms associated with obesity in the American, Spaniard, Malays, Indian, as well as Chinese in Singapore are inconsistent[17,19-21]. Little is known about the specific *PLIN* variants and their function in Chinese people. This study was to investigate the association between *PLIN* 1237 polymorphisms and obesity, as well as some metabolic measures related to energy homeostasis in Chinese Han adults.

MATERIELS AND METHODS

Subjects and Study Design

A total of 998 Han subjects (age $\geqslant$18) were

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recruited by two-stage cluster random sampling at two rural communities in Henan Province from August 2005 to April 2006. The present analysis was restricted to the 994 subjects with their body mass index (BMI) successfully measured. This study was approved by the Ethics Committees of Zhengzhou University. Informed consent was obtained from all participants. Data on demographic and anthropometric characteristics were collected using an interviewer-administered questionnaire. Body fat was evaluated using body weight, BMI, waist circumference, hip circumference, and waist/hip ratio. Obesity was defined as BMI of 28 kg/m^2 or higher, overweight as BMI of 24 or higher and less than 28. Blood pressure (BP) was measured with a calibrated mercurial sphygmomanometer following the procedures recommended by the American Heart Association. Hypertension was defined as an average systolic BP (SBP)≥140 mmHg, an average diastolic BP (DBP) ≥90 mmHg, and/or self-reported current treatment for hypertension with antihypertensive medication.

Biochemical Measurements

After the subjects were fasted for 12 h, blood samples were drawn on disodium EDTA for measurement of glucose level and on non-EDTA for measurement of total cholesterol (TC), triglyceride (TG), high density lipoprotein-cholesterol (HDL-C), low density lipoprotein-cholesterol (LDL-C), apolipoprotein A1 (apoA1), apolipoprotein B (apoB) with standardized automated enzymatic methods. Diabetes were defined as glucose ≥ 7.0 mmol/L, hypercholesteremia as total cholesterol ≥ 5.69 mmol/L, hypertriglyceridemia as triglycerides ≥1.70 mmol/L, low HDL-C as HDL-C≤0.91 mmol/L, high LDL-C as HDL-C≥3.61 mmol/L, high apoA1 as apoA1 ≥ 1.6 mmol/L, low apoB as apoB ≤ 0.60 mmol/L.

DNA Isolation and Genotyping

Genotyping was carried out using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP). Genomic DNA was isolated from whole blood using a blood genome DNA extraction kit (TAKARA). Genomic DNA was available for all 991 subjects. DNA fragments encompassing the targeted SNP (accession number in Genbank rs1052500) were amplified by PCR using the primers (Forward: 5′-GGAGAAGAAACAGA-GGTTCAGAG-3′, Reverse: 5′-ATCCTGGGAC- TGATGGCTGG-3′), which was carried out in a 25 μL reaction volume containing 70 μmol/L of each dNTP, 0.5 μmol/L of each primer, 1.5 mmol/L magnesium chloride, and 1.5 U of Qiagen Hotstar Taq polymerase (TAKARA). PCR cycling conditions were 95 ℃ for 10 min followed by 36 cycles of 95 ℃ for 30 sec, 63 ℃ for 30 sec, and 72 ℃ for 1 min, a final extension phase of 10 min at 72 ℃ was included at end of the protocol. The PCR products (705 bp) were incubated for 10-12 h at 37 ℃ with 5U each of Mspal I (Ferments) in a 20 μL reaction volume, and separated by 2% agarose gel electrophoresis. The quality control for genotyping was established, and 20% of samples were repeated at random to verify the reproducibility.

Statistical Analysis

All numerical variables were shown as median (P_{25}, P_{75}) for skewed distributions and categorical variables were shown as *n* (%). Chi-square test was used to analyze categorical data. Differences in continuous variables among gender or genotypes were evaluated by Mann-Whitney-Wilcoxon test or Kruskal-Wallis rank test for skewed distributed data, and *t* test or ANOVA for normal distributed data. Hardy-Weinberg Equilibrium test was compared by Stata programs genhwi (http://www.stata.com/stb/stb48/sg110/genhw.hlp) using a Chi-square goodness-of-fit test[19]. Men and women were analyzed separately for gender difference in obesity -related risk factors. Association between *PLIN* 1237 polymorphism and obesity status was assessed by multivariable ordinal logistic regression analysis to adjust for age, gender, age&gender, education, household income, smoking, and alcohol consumption. Odds ratios (OR) with 95% confidence intervals (CI) were given to estimate the relative risk. All statistical analyses were done with Stata version 10.0, except for power analysis which was computed by nQuery Advisor 7.0. $P<0.05$ was considered statistically significant.

RESULTS

The characteristics of demographic, anthropometric, biochemical, and lifestyle factors are shown in Table 1. The prevalence of overweight and general obesity was 32.5% and 15.8%, respectively. Gender differences in the prevalence of abdominal obesity and obesity-related measures were significant.

Genotye distributions for *PLIN* 1237 were in Hardy-Weinberg equilibrium in three groups (Table 2, $P>0.05$). Genotype TT was most common. No difference was found in genotypic and allelic frequencies among subjects with normal weight, overweight, and obesity in women and men ($P>0.05$). The frequency of major allele T and minor allele C was 0.73 and 0.27, respectively.

TABLE 1

Descriptive Characteristics of Participants by Gender (n=994)

	Women	Men	*P* Value
Age (Years)	51 (39, 60)	48 (38, 58)	0.045
Marital Status			
Married	511 (88)	363 (88)	0.94
Single	68 (12)	49 (12)	
Education (Years)			
⩽9	514 (89)	352 (85)	0.09
>9	63 (11)	60 (15)	
Household Income (Yuan/Month)			
<200	257 (46)	188 (46)	0.67
200-300	112 (20)	90 (22)	
⩾300	190 (34)	130 (32)	
Alcohol Use			
Never User	558 (96)	198 (48)	<0.001
Current/Former User	21 (4)	214 (52)	
Smoking Status			
Never User	575 (99)	143 (35)	<0.001
Current /Former User	4 (1)	269 (65)	
Body Weight	57.5 (52.0,65.5)	65.6 (59.3,73.0)	<0.001
BMI (Kg/m^2)	24.0 (21.8,6.8)	23.6 (21.3,26.3)	0.030
Waist Circumference (cm)	81.0 (73.2,90.4)	82.5 (75.0,91.1)	0.035
Waist-to-hip Ratio	0.86 (0.82,0.92)	0.89 (0.84,0.95)	<0.001
Total Cholesterol (mmol/L)	5.0 (4.3,6.0)	4.8 (4.0, 5.5)	<0.001
Triglycerides (mmol/L)	1.4 (1.0, 2.0)	1.2 (0.9, 1.7)	0.012
HDL-C (mmol/L)	1.5 (1.3, 1.8)	1.4 (1.2, 1.6)	<0.001
LDL-C (mmol/L)	2.8 (2.2, 3.5)	2.7 (2.2, 3.3)	0.017
Apoprotein A1 (mmol/L)	1.6 (1.4, 1.8)	1.5 (1.3, 1.6)	<0.001
Apoprotein B (mmol/L)	1.0 (0.8, 1.3)	0.9 (0.7, 1.2)	0.009
Glucose (mmol/L)	5.5 (5.0, 6.2)	6.2 (5.0, 6.2)	0.886
Obesity (BMI⩽28)	103 (17.8)	54 (13.1)	0.118
Overweight (24⩽BMI＜28)	187 (32.3)	134 (32.7)	0.137
Abdominal Obesity	307 (53.0)	174 (42.0)	0.001
Diabetes Mellitus	72 (12.5)	48 (11.5)	0.647
Hypertension	143 (24.7)	110 (26.7)	0.472
Hypercholesteremia	171 (29.6)	80 (19.2)	<0.001
Hypertriglyceridemia	191 (33.1)	109 (26.2)	0.019

Note. Data of numerical variables were shown as median (P_{25}, P_{75}), *P* values refelecting the differences in gender were assessed by Mann-Whitney-Wilcoxon test; Data of categorical variables were shown as a percentage *n* (%), *P* values were assessed by Chi-square test. BMI: body mass index; SD: standard deviation; HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol. Missing values were less than 0.5% in all variables except for household income (27 participants or 3% were missing).

The association between *PLIN* 1237 variations and obesity risk was also examined by multivariable ordinal logistic regression. As listed in Table 3, the odds ratio for overweight or obesity for the CC+TC genotype was 0.8 (0.4, 1.4) in women (*P*=0.4) and 0.6 (0.3, 1.3) in men (*P*=0.2) after adjustment for age, education, household income, and alcohol consumption, smoking, and physical activity. No significant interaction occurred between *PLIN* genotypes and gender in the outcome variables (data not shown).

Table 4 show the impact of *PLIN* 1237 variations on anthropometric and metabolic variables. No significant association was found between *PLIN* 1237 variations, anthropometric and metabolic variables.

TABLE 2

Allele/Genotype Distribution in *PLIN* 1237 by Obesity Status and Gender

Group	Genotype Frequency *n* (%)			Allele Frequency *n* (%)	
Women	TT	TC	CC	T	C
Normal	161 (56)	101 (35)	25 (9)	423 (74)	151 (26)
Overweight	101 (55)	73 (39)	11 (6)	275 (74)	95 (26)
Obese	55 (54)	40 (39)	7 (7)	150 (74)	54 (26)
χ^2	1.972			0.06	
P	0.741			0.97	
Men	TT	TC	CC	T	C
Normal	120 (54)	79 (35)	25 (11)	319 (71)	129 (28)
Overweight	71 (53)	54 (40)	9 (7)	196 73)	72 (27)
Obese	29 (54)	19 (35)	6 (11)	77 (71)	31 (29)
χ^2	2.462			0.327	
P	0.651			0.849	

Note. Hardy-Weinberg equilibrium test was compared with Stata programs genhwi. χ^2_{all}=3.188, *P*=0.074; χ^2_{women}=0.676, *P*=0.411; χ^2_{men}=3.188, *P*=0.076). Estimated disequilibrium coefficient (D) was 0.011, 0.007, and 0.018 for all participants, women and men, respectively.

TABLE 3

Obesity Risk (Normal, Overweight, Obesity) Associated with *PLIN* 1237 Gene (Unadjusted and Adjusted Estimations; Co-dominant and Recessive Models)

	Women				Men			
Model	Unadjusted OR[#] (95% CI)	*P*[#]	Adjusted OR[Δ] (95% CI)	*P*[Δ]	Unadjusted OR[#] (95% CI)	*P*[#]	Adjusted OR[Δ] (95% CI)	*P*[Δ]
PLIN 1237 (T>C)								
Co-dominant Model								
TT	1		1		1		1	
TC	0.8 (0.4, 1.5)	0.43	0.8 (0.4, 1.5)	0.51	0.8 (0.4, 1.5)	0.48	0.7 (0.3, 1.4)	0.29
CC	1.1 (0.8, 1.6)	0.44	1.1 (0.8, 1.6)	0.45	1.1 (0.7, 1.6)	0.73	1.2 (0.8, 1.7)	0.50
Recessive Model								
TT	1		1		1		1	
CC+TC	0.7 (0.4, 1.2)	0.20	0.8 (0.4, 1.4)	0.40	0.8 (0.4, 1.5)	0.48	0.6 (0.3, 1.3)	0.21

Note. OR: odds ratio; CI: confidence interval. [#]Crude ordinal logistic regression model. [Δ]Multivariate ordinal logistic model adjusted for age, education, household income and alcohol consumption, smoking, and physical activity (alcohol consumption and smoking were not included in the model for women).

TABLE 4

Association between *PLIN* 1237 Genotypes and Body Fat Measures and Metabolism-related Measures

	Women			Men		
	TT	TC	CC	TT	TC	CC
Mean Body Weight (Kg)	57.5 (52.3,64.0)	58.5 (52.0,65.0)	57.0 (48.5,61.0)	65.5 (59.5,73.0)	66 (58.8,72.5)	64.0 (59.1,72.3)
BMI (Kg/m^2)	23.9 (21.9,26.8)	24.3 (21.8,26.9)	23.4 (21.3,25.2)	23.7 (21.5,26.3)	23.6 (20.9,26.4)	23.3 (21.0,25.9)
Waist Circumference (cm)	81.1 (73.2,89.4)	82.1 (73.5,91.2)	76.9 (72.6,84.0)	83.1 (75.0,93.2)	82.3 (74.3,89.0)	81.3 (76.7,90.6)
Waist-to-hip Ratio	0.87 (0.82,0.92)	0.87 (0.82,0.92)	0.84 (0.80,0.89)	0.89 (0.84,0.95)	0.89 (0.84,0.93)	0.90 (0.86,0.95)
Glucose (mmol/L)	5.5 (5.0, 6.2)	5.6 (5.0, 6.3)	5.6 (5.2, 6.3)	5.6 (5.1, 6.2)	5.4 (5.0, 6.0)	5.4 (5.0, 6.4)
Triglycerides (mmol/L)	1.4 (0.9, 2.0)	1.3 (1.0, 2.0)	1.4 (1.0, 2.0)	1.3 (0.9, 1.9)	1.1 (0.8, 1.6)	1.1 (0.9, 1.8)
Total Cholesterol(mmol/L)	5.1 (4.3, 6.0)	5.0 (4.2, 5.9)	4.8 (4.3, 6.1)	4.8 (4.0, 5.6)	4.7 (4.1, 5.3)	4.8 (4.2, 5.5)
HDL-C (mmol/L)	1.5 (1.3, 1.8)	1.5 (1.3, 1.7)	1.6 (1.4, 1.8)	1.4 (1.2, 1.6)	1.4 (1.2, 1.5)	1.3 (1.2, 1.5)
LDL-C (mmol/L)	2.8 (2.3, 3.5)	2.8 (2.2, 3.4)	2.9 (2.2, 3.6)	2.7 (2.2, 3.4)	2.9 (2.2, 3.2)	2.7 (2.3, 3.3)
Apoprotein A1 (mmol/L)	1.6 (1.4, 1.8)	1.6 (1.3, 1.8)	1.6 (1.4, 1.9)	1.5 (1.3, 1.7)	1.4 (1.3, 1.6)	1.5 (1.3, 1.6)
Apoprotein B (mmol/L)	1.0 (0.8, 1.3)	1.0 (0.8, 1.2)	1.0 (0.8, 1.3)	0.9 (0.7, 1.2)	0.9 (0.8, 1.1)	1.0 (0.8, 1.2)

Note. Data of variables are shown as median (P_{25}, P_{75}). No statistical significance by genotype was found in men and women ($P>0.05$).

DISCUSSION

We conducted a genetic study for on the association between *PLIN* 1237 gene and obesity risk, as well as some anthropometric and metabolic variables in 994 northern Chinese Han adults. The most interesting finding in this study is that the frequency of variant-allele C (0.27) was lower in Chinese Han adults than in Indian of Singapore (0.47), Mediterranean Spanish (0.39), American white (0.34), Malaysian of Singapore (0.33), and was similar to that in Chinese of Singapore (0.30), Korean (0.26)[17,20-21]. The allele frequencies for the *PLIN* 1237 polymorphism differed among ethnic groups and might be ethnically related to the differences in the prevalence of obesity.

No association was found between *PLIN*1237 polymorphism and obesity risk, which is consistent with the reported data[14]. Since a large number of subjects (*n*=994) were enrolled in our study, its statistical power was sufficient to detect the major associations (power>80%). *PLIN* 1237 T＞C is located at exon 8, where alternative splicing occurs during *PLIN* transcription resulting in several perilipin isoforms. Perilipin isoforms might function with a different efficiency in protecting the stored body fat from protein kinase A-mediated lipolysis. In recent years, a significant association between polymorphisms in *PLIN* and obesity or diabetes has been reported in different ethics[11-12,18,22]. Higher *PLIN* mRNA levels with a higher percent of body fat and BMI have been observed in white women[17]. *PLIN* 1237 variant-allele C (genotype CC + TC) is associated with a higher obesity risk. Ethnic differences in distributions of *PLIN* 1237 polymorphisms may contribute to this inconsistent conclusion, which needs further investigation in a large multiethnic population.

The distribution of body fat in men and women is different. Some *PLIN* SNPs (6209T>C, 11482G>A, 13041A>G, and 14995A>T) are associated with obesity in women only, suggesting that women probably are more sensitive to the genetic effects than men[12,17]. However, we did not find any significant interactions between gender and *PLIN* 1237 genotypes. Moreover, two other *PLIN* SNPs (A13041G and A14995T) are significantly associated with the indices of obesity and metabolic variables in women[12], but such an association was not observed in our study.

The inconsistence in studies on the association between *PLIN* 1237 polymorphisms and obesity are caused by many factors. Firstly, there was an ethic difference or genetic diversity in subjects of our study. It has long been noted that different race/ethnic groups experience dramatic susceptibility differences to obesity[12,14-15,17,23-24]. The allele frequencies for the *PLIN* 1237 polymorphisms in Chinese Han individuals are different from other ethnics. Secondly, the absence of association in our study might be due to these polymorphisms with synonymous mutations (NCBI dbSNP database) and could not change the amino acids in the protein. Only those *PLIN* polymorphisms modifying the structure of functional active site may be closely associated with complicated phenotypic measurements such as obesity. Since these subjects may be genetically predisposed to obesity due to the influence of other gene variations, we could not exclude the influences of other *PLIN* SNPs that evaded our sight.

In summary, 1237 T>C polymorphism of *PLIN* is significantly associated with obesity in Chinese northern adults. Due to the restricted study sites and a relatively small sample size, further studies are needed to draw a definite conclusion on the risk of obesity.

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CONTRIBUTORS

All authors made an equal contribution to the study, YANG J X undertook the analysis and interpreted the results and wrote the paper. LU J, YU D H, XU G H, and YANG J X commented on the paper. YU D H conceived and supervised the work and edited the paper. YANG J X surpervised data analysis.

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