EFFECTS OF FABP2 ALA54THR GENE POLYMORPHISM ON OBESITY AND METABOLIC SYNDROME IN MIDDLE-AGED KOREAN WOMEN WITH ABDOMINAL OBESITY

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SUMMARY

Objectives: Asians (including Chinese, Japanese and Koreans), who generally have a relatively smaller body size and a lower mean body mass index (BMI), have a relatively higher risk of developing android-type obesity than westerners. Substitution of alanine for threonine (Ala54Thr) on the FABP2 gene (rs 1799883) is related to insulin resistance and obesity. However, few studies have examined this substitution in Koreans, and the number of Korean subjects in those studies is limited. For this reason, we investigated the differences between the FABP2 Ala54Thr polymorphism and obesity, hemodynamic variables, blood lipid profile results, and insulin resistance among middle-aged Korean women with abdominal obesity.

Methods: We studied 243 middle-aged community-dwelling Korean women with abdominal obesity from Gyeonggi Province, Republic of Korea, who had no history of taking chronic medications. We examined each subject (n = 243) for the presence of FABP2 Ala54Thr polymorphism using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP). Subjects were also examined for obesity hemodynamic variables (n = 243), lipid profiles (n = 142), and insulin resistance (n = 142).

Results: Of the 243 subjects, 117 had AA (“normal”) homozygotic genotype, 100 had AT heterozygotic genotype, and 26 had TT homozygotic genotype for the FABP2 Ala54Thr polymorphism. The AT heterozygotic individuals had a significantly higher mean waist-to-hip ratio, abdominal fat area, and visceral fat area than individuals with other genotypes. TT homozygotic individuals had higher mean triglyceride and fasting glucose levels than individuals with other genotypes.

Conclusions: The results of this study show that the FABP2 Ala54Thr polymorphism was associated with central obesity and obesity-related metabolic syndrome among middle-aged Korean women.

Key words: abdominal obesity, fatty acid binding protein-2, fatty acid binding protein-2 polymorphism, insulin resistance, middle-aged women

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INTRODUCTION

In 2000, approximately 1.2 billion people worldwide were overweight or obese, and 2.5 million deaths were caused by obesity-related illnesses; without appropriate intervention, this number could have reached 5 million (1). Obesity is classified into android-type obesity (apple-shaped obesity) and gynoid-type obesity (pear-shaped obesity). In android-type obesity, the fat mass occurs in the abdominal area and is more likely to be associated with metabolic syndrome and insulin resistance than gynoid-type obesity, in which the fat mass usually accumulates in the thigh area (2, 3).

Interestingly, Asians (including Chinese, Japanese and Koreans), who generally have a relatively smaller body size and a lower mean body mass index (BMI), have a relatively higher risk of developing android-type obesity than westerners (4, 5). Therefore, in the absence of treatment, Asians, including Koreans, have a higher risk of disorders associated with obesity-related metabolic syndrome, including diabetes, dyslipidemia, hypertension, cardiovascular disease, and cancer (6–8).

Metabolic syndrome is associated with insulin resistance; patients with metabolic syndrome often have poor dietary habits and a sedentary lifestyle. Non-insulin-dependent diabetes and obesity have a genetic link (9). Considerable effort has been made to identify the genes associated with insulin resistance. Only one gene affects insulin sensitivity (10). Current candidate genes include the insulin receptor substrate-1 (IRS-1), β3-adrenergic receptor (β3-AR), uncoupling protein-1 (UCP-1), glucose transporter (GLUT), and fatty acid binding protein-2 (FABP2) (11–13). Pima Indians suggest that the FABP2 gene is associated with insulin sensitivity (14).

To date, nine FABP genes have been shown to be expressed in various tissue types: a 1-kilobase (kb) sequence upstream from the start codon in the regulatory region, three indel polymorphisms, and four single nucleotide polymorphisms (SNPs). One of the nine FABP genes, the FABP2 gene (3.4 kb), is located on chromosome 7.
4q28-31 and has four exons (700 base pairs (bp) and three introns (2,650 bp) (15). In FABP2, substitution of alanine for threonine (Ala54Thr, rs 1799883), resulting from substitution of adenine for guanine, is associated with insulin resistance (16). FABP2, a relatively small protein (15 kilodalton), is involved in the absorption of fatty acids and intracellular transportation and is widely distributed in epithelial cells of the villi in the small intestine (17, 18). The FABP2 for long-chain fatty acids is doubled when in FABP2 gene has an Ala54Thr polymorphism (16). This increase in fat absorption and free fatty acids (FFAs) may cause insulin resistance by increasing intracellular oxidation (16).

The Ala54Thr polymorphism increases triglyceride (TG) secretion and free fatty acid transport in vitro (19). Moreover, the Thr54 allele is associated with a higher level of fasting insulin, insulin resistance, and 2-h blood glucose level (20). Furthermore, with excess absorption of fatty acids, the thr54 variant considers skeletal muscles a priority and uses fatty acids rather than glucose for energy; this leads to an increase in glucose levels (21).

Studies of Japanese, Pima Indians, and some Caucasians found that FABP2 Ala54Thr polymorphism is associated with insulin resistance, but studies among populations from England, Finland, Wales, and some European countries found no association, suggesting differences among races (10, 14, 16, 22, 23). Previous studies on FABP2 polymorphisms and energy metabolism in the body found no differences in BMI, blood lipid concentrations, or fasting glucose levels between individuals with FABP2 Ala54Thr polymorphism and those with other genotypes. Homeostasis model assessment (HOMA) found that basal insulin concentrations and insulin resistance index are both higher in the presence of the FABP2 Ala54Thr polymorphism (24, 25). Although there are no differences in metabolic rates among individuals with these alleles, the Thr54 allele results in a significantly higher level of fat oxidation than the Ala54 allele, indicating that the FABP2 Ala54Thr polymorphism may cause insulin resistance through changes in fat metabolism among Koreans (25). However, few studies have examined changes in fat metabolism in Koreans, and studies that evaluated Koreans had few subjects. Therefore, this study aimed to examine the differences between FABP2 Ala54Thr polymorphism and obesity variables (weight, waist circumference, waist-to-hip ratio, BMI, percent body fat, total abdominal area, visceral area, subcutaneous area, visceral area/subcutaneous area ratio, and maximum volume of oxygen consumed), hemodynamic variables (systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, and cardiac stress), blood lipid profile, and insulin resistance among middle-aged Korean women with abdominal obesity.

MATERIALS AND METHODS

Participants

We recorded the age, history of hormone replacement therapy, physical activity level, waist circumference, weight status, smoking status, and current drug use among participants of a health promotion programme from September 2006 to December 2007 in Gyeonggi Province, Republic of Korea. In this study, 243 middle-aged women who had a waist circumference > 80 cm were enrolled (1). Because of mechanical errors, blood samples of 101 study subjects were not examined for lipid profile and insulin resistance, but were examined for FABP2 Ala54Thr polymorphism. Therefore, we examined 243 subjects for the presence of the FABP2 Ala54Thr polymorphism using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP), obesity variables, and hemodynamic variables, but studied only 142 subjects for blood lipid profile and insulin resistance.

All participants gave informed consent prior to participation in this study. This study was approved by the institutional ethics review board of the College of Sport Science, Sungkyunkwan University, and was conducted according to the principles of the Helsinki Declaration.

FABP2 Polymorphism Analysis

A fasting venous blood sample was obtained from each subject. The DNA was extracted using DNA extraction kit (Qiagen, Hilden, Germany). PCR-RFLP (26) was then conducted using the GeneAmp PCR System 9700 (Applied Biosystems, Foster City, CA, USA) with 2 μL of DNA template, 15 μL of 2 × PCR master mix (Promega, Madison, WI, USA), 0.25 μL of sense primer (5′-CTACCGAGTTTTCTTCCACC-3′), 0.25 μL of antisense primer (5′-AATTAAACCATCCAATGAAATAGC-3′), and 12.5 μL of distilled water. Fragments were amplified as follows: 5 min of denaturation at 94 °C; 25 cycles of denaturation for 30 s at 94 °C, annealing for 30 s at 55 °C, extension for 30 s at 72 °C, and a final extension for 30 s at 72 °C. Four units of the restriction enzyme HhaI (New England BioLabs, Beverly, MA, USA) was added to the PCR product and incubated for 1.5 h at 37 °C. Electrophoresis of the sample was conducted at 85 volts for 120 min on 2% agarose gel and stained with ethidium bromide. Different DNA fragment sizes were observed: two bands at 200 and 175 bp, indicating the AA (“normal”) genotype; three bands at 375, 200, and 175 bp indicating the AT genotypes; and a single band at 375 bp indicating the TT genotype (Fig. 1).

![Fig. 1.](image-url)
BMI and Blood Pressure

Heights and weights were determined for each study subject and BMI was calculated kg/m\(^2\) as the weight in kilograms divided by the height in meter squared. The percent body fat was measured using an eight-polar bioelectrical impedance analysis (X-Scan Body Composition Analyzer, JAWON Medical Co., Seoul, Korea) (27, 28). Waist circumference at the umbilicus and hip circumference at the widest area between the superior and inferior iliac crests were measured using a measuring tape and the waist-to-hip ratio (WHR) was then calculated. Abdominal, subcutaneous, and visceral fat areas were measured at the Korean Health Management Association, Suwon City, Korea by computed tomography at the fourth lumbar level (SCT-7800TE; SHIMADZU, Kyoto, Japan) (29, 30). Resting blood pressure was measured twice at 5 min interval using an automatic sphygmomanometer (JAWON Medical Co., Seoul, Korea), and the mean value was then calculated and recorded. Cardiac stress (index) was defined as systolic blood pressure (mm Hg) ÷ heart rate (beats per minute).

Blood and Blood Lipid Profile

Venous blood was collected from brachial veins after fasting for 10–12 h. Collected blood was immediately centrifuged, and the serum was then stored at −80°C until use. The serum samples were examined for total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and TGs using a Vitros DT60II Chemistry System (Johnson & Johnson, NY, USA). Blood HDL-C levels were analyzed following the precipitation of cholesterol with apolipoprotein B using magnesium chloride and dextran sulfate. Low-density lipoprotein cholesterol (LDL-C) levels were calculated using the following formula: LDL-C = TC – (HDL-C+(TG/5)) (31).

Oral Glucose Tolerance Test

The Oral Glucose Tolerance Test (OGTT) was performed on each subject as follows: a 21-gauge polyethylene catheter was inserted in the brachial vein, and 75 g of glucose was injected (32). Venous blood was collected 10 min before the glucose injection and then at 30, 60, 90, and 120 min after injection. The collected venous blood was centrifuged and stored at −80°C. Blood glucose levels were analyzed using the Vitros DT60II Chemistry System (Johnson & Johnson, NY, USA). Blood HDL-C levels were analyzed following the precipitation of cholesterol with apolipoprotein B using magnesium chloride and dextran sulfate. Low-density lipoprotein cholesterol (LDL-C) levels were calculated using the following formula: LDL-C = TC – (HDL-C+(TG/5)) (31).

AUC = ((M2+M1)/2)+((M3+M2)/2)+((M4+M3)/2)+((M5+M4)/2),

where M1 is the fasting glucose level, M2 is the glucose level at 30 min, M3 is the glucose level at 60 min, M4 is the glucose level at 90 min, and M5 is the glucose level at 120 min.

The HOMA index was calculated from the fasting serum glucose and insulin levels using the following formula (34):

\[ \text{HOMA index} = \frac{\text{glucose level (mM)} \times \text{insulin level (µU/mL)}}{22.5}. \]

Cardiorespiratory Fitness

Cardiorespiratory fitness (maximal oxygen consumption, VO\(_{\text{max}}\)) was measured by a treadmill exercise test at 1.7 mph and a 10% of grade using Bruce Protocol (35) with an increase of 0.8–0.9 mph and 2% of grade every 3 min, and breath-by-breath type was applied using gas analyzer (TrueOne Metabolic Cart; Parvo Medics, Sandy, UT, USA) and a wireless heart rate analyzer (Polar a5, Polar, Finland). Oxygen consumption per minute was measured at 85% of the maximum predicted heart rate to determine cardiorespiratory fitness (36). Cardiorespiratory fitness followed the recommendations of the book Advanced Fitness Assessment and Exercise Prescription (37).

Statistical Analysis

All data were expressed as means ± standard deviations and were analyzed by descriptive statistics. Test of normality for all measured values was conducted using one-sample Kolmogorov-Smirnov Test. Homoscedasticity test was conducted using Levene variance F-test. In this study, all variables at p > 0.05 were accepted appropriate for model. Comparisons of each variable with respect to the FABP2 Ala54Thr polymorphisms were made using one-way analysis of variance and Tukey’s post-hoc testing. Statistical calculations were performed using SPSS, version 18.0 (Chicago, IL, USA). Statistical significance was set at p < 0.05.

RESULTS

Association of FABP2 Ala54Thr Polymorphism

Of the 243 participants (48.1%), 117 had the AA genotype (“normal”) on PCR-RFLP, 100 (41.2%) had the AT genotype, and 26 (10.7%) had the TT genotype (Table 1). The relative incidences of the A and T alleles were 68.7% and 33.3%, respectively.

Differences between FABP2 Ala54Thr Genotypes and Obesity Factors and Cardiorespiratory Fitness

The WHR, total abdominal (TA) area, and visceral fat area were statistically significantly associated with distribution of FABP2 Ala54Thr genotypes (p < 0.05). However, there were no statistically significant differences in age, weight, BMI, percent body fat, subcutaneous area, visceral fat area/subcutaneous fat area, and VO\(_{\text{max}}\) (p > 0.05) (Table 2).

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**Table 1. Relative frequencies of FABP2 Ala54Thr genotypes in study population (N = 243)**

<table>
<thead>
<tr>
<th>Independent/Dependent</th>
<th>Genotype frequency</th>
<th>Allele frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ala/Ala</td>
<td>Ala/Thr</td>
</tr>
<tr>
<td>Total</td>
<td>117 (48.1%)</td>
<td>100 (41.2%)</td>
</tr>
</tbody>
</table>
Differences between FABP2 Ala54Thr Genotypes and Blood Pressure, Heart Rate and Cardiac Stress

The systolic blood pressure showed statistically significant differences among FABP2 Ala54Thr genotypes (p < 0.05). However, there were no statistically significant differences in diastolic blood pressure, mean arterial pressure, heart rate, and cardiac stress (p > 0.05) (Table 3).

### Table 3. Comparison of baseline values in blood pressure, heart rate and cardiac stress among FABP2 Ala54Thr genotypes

<table>
<thead>
<tr>
<th>Independent/Dependent</th>
<th>Genotype</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ala/Ala (n = 117)</td>
<td>Ala/Thr (n = 100)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>45.9 ± 7.2</td>
<td>47.1 ± 7.6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>64.1 ± 7.2</td>
<td>64.1 ± 7.2</td>
</tr>
<tr>
<td>WHR (%)</td>
<td>0.93 ± 0.05*</td>
<td>0.95 ± 0.05a</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.6 ± 2.6</td>
<td>25.8 ± 2.6</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>33.2 ± 2.9</td>
<td>33.3 ± 3.0</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>114.6 ± 14.5</td>
<td>119.9 ± 14.0</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>70.8 ± 10.1</td>
<td>73.1 ± 10.1</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>84.8 ± 11.2</td>
<td>88.1 ± 11.0</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>73.7 ± 10.4</td>
<td>71.9 ± 10.7</td>
</tr>
<tr>
<td>Cardiac stress (index)</td>
<td>8422.4 ± 1824.9</td>
<td>8638.6 ± 1779.9</td>
</tr>
</tbody>
</table>

WHR – waist-to-hip ratio; BMI – body mass index; TA area – total abdominal area; V/S ratio – visceral fat area/subcutaneous fat area. Cardiorespiratory fitness was measured as the maximum volume of oxygen consumption (VO\textsuperscript{2}max) during graded treadmill exercise.

*p < 0.05, **p < 0.01, one-way analysis of variance

Differences between FABP2 Ala54Thr Genotypes and Blood Lipid Profiles and Insulin Resistance Markers

The TG and glucose showed statistically significant differences among FABP2 Ala54Thr genotypes (p < 0.05). However, there were no statistically significant differences in TC, LDL-C, HDL-C, insulin, and HOMA (p > 0.05) (Table 4).

### Table 4. Comparison of baseline values in blood lipid levels and insulin resistance markers among FABP2 Ala54Thr genotypes

<table>
<thead>
<tr>
<th>Independent/Dependent</th>
<th>Genotype</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ala/Ala (n = 72)</td>
<td>Ala/Thr (n = 57)</td>
</tr>
<tr>
<td>TC (mg/dL)</td>
<td>196.3 ± 41.2</td>
<td>192.9 ± 42.6</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>134.8 ± 37.9</td>
<td>127.6 ± 32.4</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>41.6 ± 11.4</td>
<td>42.2 ± 14.1</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>111.3 ± 37.9a</td>
<td>106.0 ± 46.6</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>97.0 ± 15.6</td>
<td>95.0 ± 16.5aa</td>
</tr>
<tr>
<td>Insulin (μU/ml)</td>
<td>6.5 ± 3.3</td>
<td>7.0 ± 3.8</td>
</tr>
<tr>
<td>HOMA (index)</td>
<td>1.5 ± 0.8</td>
<td>1.6 ± 0.9</td>
</tr>
</tbody>
</table>

TC – total cholesterol; LDL-C – low density lipoprotein cholesterol; HDL-C – high density lipoprotein cholesterol; TG – triglyceride; HOMA index = (glucose [mM] \times insulin [μU/mL])/22.5

*p < 0.05, **p < 0.01, one-way analysis of variance

*p < 0.05, Ala/Ala < Ala/Thr, Ala/Thr < Thr/Thr; Tukey’s post-hoc testing
DISCUSSION

Obesity and its negative consequences are rapidly increasing worldwide. Genetic studies must concentrate on polymorphisms in the gene encoding FABP, a protein involved in fat synthesis and metabolism, to identify factors that are correlated with obesity. The absorption of fat is one of the most important factors underlying metabolic syndrome. Polymorphisms in the intestinal FABP promoter, resulting in transcriptional activation, lead to changes in body composition, and a missense polymorphism in codon 54(Ala/Thr) has been linked to insulin resistance (38). FABP2 Ala54Thr polymorphism increases the absorption of fatty acids via food intake; indeed, FABP2 harbouring the Thr54 allele has twice the affinity for fatty acids compared with FABP2 harbouring the Ala54 allele. This polymorphism inhibits insulin activation by increasing fatty acid oxidation (16, 39).

Thus, in this study, we investigated the genetic influence of FABP2 Ala54Thr on obesity indices, hemodynamic variables, and insulin resistance in disease-free, middle-aged Korean women with abdominal obesity and a waist circumference greater than 80 cm. Based on the genetic analysis of FABP2 in 243 women, we found that 117 women had the AA homozygotic genotype, 100 had the AT heterozygotic genotype, and 26 had the TT homozygotic genotype. Of these, the relative frequencies of A and T alleles were 68.7% and 33.3%, respectively, similar to the frequencies seen in Pima Indians (29% T), Caucasian (31% T), and Japanese (35% T) populations (16, 23). The relative frequency of FABP2 Ala54Thr polymorphism in our study showed the Hardy-Weinberg equilibrium, indicating that these patients were a representative sample of the population.

The Ala54Thr polymorphism in FABP2 has been shown to be associated with high BMI and percent body fat in Canadian Indians (40) and excessive accumulation of intra-abdominal fat in Japanese men (23). Furthermore, obesity index-related genetic sensitivity has been linked to FABP2 Ala54Thr polymorphism, as reported by Takakura et al. (41) and Albala et al. (42). Based on these previous studies, we expected to observe a correlation between total obesity indices and the FABP2 genotype. However, we did not observe significant differences in weight, BMI, percent body fat, or waist circumference among the FABP2 genotypes, as reported in previous studies (15, 26, 43–44). To account for this discrepancy, Yun et al. (25) examined the relationship between resting metabolic rate (RMR) and the FABP2 Ala54Thr polymorphism in Korean men and found no difference in the RMR among FABP2 genotypes. However, Takakura et al. suggested that the Thr54 allele increases the obesity rate by lowering the RMR (41); thus, the results of studies on FABP2 Ala54Thr polymorphism and obesity are inconsistent and vary depending on the race, ethnicity, and characteristics of the participants.

In our study, we found no genetic link between the FABP2 Ala54Thr polymorphism and weight, percent body fat, or BMI in Korean women with abdominal obesity. However, individuals with the FABP2 AT heterozygotic genotype exhibited significantly higher central obesity indices, except for waist circumference, such as WHR, whole-abdominal area, and visceral fat. In other words, increases in WHR, whole-abdominal area, and visceral fat area in individuals with the Thr allele carriers were significantly larger than those in individuals with the AA homozygotic genotype, suggesting that the polymorphism in FABP2 was an important factor affecting genetic sensitivity to central obesity in middle-aged Korean women with abdominal obesity. When examining VO2 max per minute during the graded treadmill exercise, a predictor of cardiorespiratory fitness, we found no association between the FABP2 Ala54Thr polymorphism and cardiorespiratory fitness, as suggested by Weiss et al. (15).

Then, we examined the role of FABP2 Ala54Thr polymorphism in determining an individual’s predisposition for hypertension by regression, after accounting for the increased systolic blood pressure in individuals with the TT homozygotic genotype and individual differences in age and body composition (weight, BMI, percent body fat, and WHR). Although not statistically significant, systolic blood pressure was positively associated with waist circumference. This indicated that the FABP2 Ala54Thr polymorphism was associated with a relatively higher risk of hypertension in middle-aged Korean women with abdominal obesity. Although previous studies have not reported an association between the FABP2 Ala54Thr polymorphism and hypertension, individuals with the Thr54 allele were assumed to have significantly higher blood TG and glucose levels, vessel elasticity, secretion of related proteins, and secretion of cytokines, causing vessel inflammation and subsequent increases in other cardiovascular factors (45–46), predisposing such individuals to obesity. In a study by Georgopoulos et al. (47), the Veterans Affairs HDL Intervention Trial compared dyslipidemia between men with or without type 2 diabetes. They found that the Thr54 polymorphism is associated with a 2- to 3.5-fold higher risk of cardiovascular disease in men with type 2 diabetes than in men without diabetes.

Baier et al. (16) conducted an OGTT in 760 Pima Indians and found that the FABP2 Ala54Thr polymorphism was significantly associated with insulin resistance and in vitro data also suggested that FABP2 containing Thr54 had a two-fold increase in affinity for long-chain fatty acids than FABP2 containing Ala54. Thus, Thr54 increases the absorption of fatty acids in the intestinal canal. Consequently, excessive release of fatty acids and increased insulin resistance are induced by systemic circulation and excessive influx of fatty acids in peripheral tissue. Similarly, Mitchell et al. (48) conducted an OGTT in Mexican-American individuals and reported that the FABP2 Thr54 allele increased insulin secretion at 120 min and significantly increased the risk of insulin resistance based on the HOMA index. However, there was no association between fasting insulin or HOMA index and FABP2 genotype among European populations in England or Finland (49, 50).

Nevertheless, our study had some limitations. First, the study lacked statistical power because of the inadequate number of participants and because blood samples from only a portion of the study population were examined to determine the associations between genetic or environmental factors and between the FABP2 Ala54Thr polymorphism and obesity. The inclusion of more participants would likely reveal a significant relationship between the FABP2 Ala54Thr polymorphism and obesity. Second, although other characteristics of the study participants, e.g., proportion of obese participants, diabetes, history of heart disease, age, or medications taken may have affected the results, we did not control for these variables. This may have biased the results. Third, this study did not include a control (nonobese) group; it is difficult to determine whether the polymorphism affected the development of central obesity in a homogeneous population because variations in the parameters were limited. Fourth, this
study did not investigate dietary intake. However, we believe that this study is valuable because it focused on a specific group – Korean middle-aged women with abdominal obesity and showed that FABP2 Ala54Thr polymorphism was associated with central obesity indices, such as WHR, total abdominal area, visceral fat, systolic blood pressure, blood TG, and glucose levels.

CONCLUSION

The results in this study showed that the FABP2 Ala54Thr polymorphism increased the risk of central obesity and obesity-related metabolic syndrome in Korean middle-aged women.

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Conflict of Interests

None declared

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