Adiposity, serum lipid levels, and allergic sensitization in Chinese men and women

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Background: Obesity and allergic diseases have increased dramatically in recent decades. Although adiposity has been associated with asthma, associations with allergic sensitization have been inconsistent.
Objective: To examine the association of adiposity and lipid profiles with allergic sensitization.
Methods: This study included 1187 rural Chinese twins (653 men) age 18 to 39 years, with skin prick tests, anthropometric and dual-energy x-ray absorptiometry–assessed adiposity measures, and lipid assessments. Allergic sensitization was defined as positive SPT to ≥1 allergen (9 foods and 5 aeroallergens tested). We applied sex-stratified generalized estimating equations to assess the association of adiposity and serum lipids with allergic sensitization, and structural equation models to estimate the genetic/environmental influences on any observed associations.
Results: Men had lower percent body fat (% BF) (13.9% vs. 28.8%) but higher rates of allergic sensitization (56.2% vs 36.7%) than women. Men in the highest %BF quartile were 2.1 times more likely to be sensitized than the lowest quartile (95% CI, 1.3-3.5; *P* trend = .003). In men, the risk of allergic sensitization increased with high-density lipoprotein (HDL) <40 mg/dL (odds ratio = 4.0; 95% CI, 1.8-9.2) and higher low-density lipoprotein quartiles (*P* trend = .007). This appeared to be partially explained by shared genetic factors between serum lipid levels and allergic sensitization. In females, lower HDL was associated with increased risk of allergic sensitization.
Conclusion: In this relatively lean Chinese population, higher %BF, lower HDL and higher LDL were associated with greater risk of allergic sensitization, most notable in men. The observed associations among adiposity, serum lipids, and allergic sensitization in men appear to be partially explained by common genetic influences on these traits. (J Allergy Clin Immunol 2009;123:940-8.)

Key words: *DEXA, body mass index, adiposity, serum lipids, sensitization*

Over recent decades, the worldwide prevalence of obesity,[1] allergic sensitization, and atopic diseases[2,3] has risen dramatically. There is increasing evidence that excess adiposity and metabolic syndrome are associated with chronic systemic inflammation[4] and asthma.[5,6] A recent study reported that there was both a shared genetic and a shared environmental component between obesity and asthma.[7] However, less is known about the relation of adiposity and lipid profiles to allergic sensitization.

To date, most of the few studies examining this topic have used body mass index (BMI) as the measure of adiposity,[8,9] with inconsistent findings.[8-12] No epidemiologic studies have evaluated the relationship between percent body fat (%BF) and allergic sensitization. This is important because %BF as determined by dual-energy x-ray absorptiometry (DEXA) is less affected by the effects of lean muscle mass than BMI.[13]

Similarly, few large-scale epidemiologic studies have examined the association of serum lipids with allergic sensitization.[14,15] Also, because adiposity is associated with disturbances in lipid profiles, it would be important to determine whether lipid profiles are independently associated with allergic sensitization or mediate an association of adiposity with allergic sensitization.[16] The 2 large-scale epidemiologic studies that have examined the association of serum lipids with allergic sensitization had inconsistent findings,[14,15] perhaps related to age[14] and sex issues.[15]

The role of sex in the association between adiposity and allergic sensitization needs further investigation. Body composition and serum lipid levels vary with sex. Females have higher %BF and higher high-density lipoprotein (HDL) than males.[17,18] Similarly, there are sex differences in prevalence of allergic sensitization [8,19] and in regulation of T_H1 and T_H2 cytokines.[20] Therefore, it is not surprising that some studies have found associations between BMI and allergic sensitization to be sex-dependent.[11]

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Abbreviations used
BF: Fat mass
%BF: Percent body fat
BMI: Body mass index
DEXA: Dual-energy X-ray absorptiometry
FMI: Fat mass index
GEE: Generalized estimating equation
HDL: High-density lipoprotein
LDL: Low-density lipoprotein
LMI: Lean mass index
OR: Odds ratio
SPT: Skin prick test
%TF: Percent trunk fat
WC: Waist circumference
WHO: World Health Organization

Most previous studies assessed populations with a high rate of obesity.[12] We chose to examine a lean population of Chinese twins in the midst of economic and nutritional transition. This approach may better delineate associations that could have been obscured in primarily obese populations, and may allow us to determine the extent to which these conditions have common genetic influences.

We evaluated the sex-specific relationships of BMI, waist circumference (WC), DEXA-derived direct measures of adiposity (%BF and percent trunk fat), and serum lipid profiles with allergic sensitization in a large rural Chinese twin cohort of young adults. We also used the twin design to examine the relative contribution of genetic and environmental factors to any associations observed.

METHODS

Study population

This study used data obtained from an ongoing study of metabolic syndrome in a large Chinese twin cohort that was originally designed to study environmental and genetic determinants of complex human diseases including metabolic syndrome. The study protocol was approved by the Institutional Review Boards of Children's Memorial Hospital and the Institute of Biomedicine, Anhui Medical University in Hefei, China.

The baseline study was carried out in 8 counties of the Anqing region of China from 1998 to 2000, and the follow-up study was conducted from 2005 to 2007. This report used the data obtained at the follow-up survey from the participants age 18 to 39 years. Anqing has a total population of 6.1 million (10% urban and 90% rural). For the baseline and the follow-up studies, both twins had to be available and willing to participate. All subjects provided written informed consent before participating in the study. In the baseline study, twins had to be 6 years or older. In the follow-up study, eligible twins had to have participated in the baseline survey. Eligible participants were invited to a central office to complete a physical examination, skin prick tests (SPTs), DEXA scan, blood draw, and questionnaire interview. The questionnaire interview obtained pertinent epidemiologic information, including occupation, education, smoking history (active and passive smoking), presence of pets in household, exposure to farm animals, and presence of mice and cockroaches in the house.

We excluded subjects who had missing data for anthropometric and adiposity measurements (n = 34) and missing or invalid SPT data (n = 31). This report included 1187 (568 twin pairs and 51 who were not paired) from a total of 1252 participants age 18 to 39 years.

Zygosity ascertainment

Zygosity was determined by microsatellite probes, or DNA fingerprinting techniques, which have an accuracy rate exceeding 99%.[21] Of 483 twin pairs (n = 966) in whom zygosity was determined, 303 pairs (male/male 149, female/female 154) were monozygotic, and 180 pairs (male/male 89, female/female 52, male/female 39) were dizygotic.

Anthropometric and adiposity assessments

Body weight and height were measured during physical examination by using standard protocols, without shoes or outerwear, as detailed elsewhere.[21] WC was measured at the level of the umbilicus. BMI was calculated as weight/height^2 (kg/m^2). BMI was also split into its 2 components, fat mass index (FMI = body fat [BF]/height^2) and lean mass index (LMI = [weight – BF]/height^2).[22]

A standard whole-body scan was performed by DEXA (GE-lunar Prodigy, Madison, Wis) to measure total body fat and trunk fat (the latter defined as chest, abdomen, and pelvis).[21,23] %BF was calculated as %BF = (total BF/body weight) × 100. Percent trunk fat (%TF) was calculated as %TF = (trunk fat/total BF) × 100.

Body mass index and %BF were used as surrogate measures of general adiposity, whereas WC and %TF were used as surrogate measures of central adiposity.

Laboratory measurements

Venous blood samples were obtained from participants after a 12-hour overnight fast. Triglycerides were measured by enzymatic methods (Boehringer Mannheim, Mannheim, Germany) and HDLs by the same enzymatic method after precipitation with dextran sulfate/magnesium chloride. Low-density lipoprotein (LDL) was calculated by using formula LDL = total cholesterol − HDL − triglycerides/5.

Skin prick testing

Skin prick testing was performed on the volar surfaces of the arms on normal skin by using the Multi-Test II (Lincoln Diagnostics, Decatur, Ill). Participants were tested for their reactions to 14 allergens, including 5 aeroallergens (*Alternaria tenuis*, house dust mite mix [equal parts mixture of *Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*], cat hair, dog epithelia, cockroach mix [American and German cockroach]) and 9 food allergens (cow milk, egg white, soybean, wheat, peanut, English walnut, sesame seed, fish mix [cod, flounder, halibut, mackerel, tuna], and shellfish mix [clam, crab, oyster, scallops, shrimp]) plus negative (50% glycerinated saline) and positive (histamine, 1.0 mg/mL) controls (Greer, Lenoir, NC). The largest wheal diameter (a) and the perpendicular diameter (b) were measured 15 minutes after application. The mean wheal diameter was calculated as (a + b) / 2. SPT data were considered invalid and thus were excluded (n = 31) if the saline control was ≥3 mm, the histamine control was <3 mm, or the difference of histamine minus saline was <3 mm. A positive SPT was defined as a valid SPT with the mean wheal diameter ≥3 mm than the saline control.

Definition of allergic sensitization

In this study, SPT was used as the measure of allergic sensitization. The primary outcome was any sensitization, defined as positive SPT to at least 1 aeroallergen or food. Secondary outcomes were any sensitization to at least 1 aeroallergen, or any sensitization to at least 1 food.

Statistical analyses

The primary outcome was any sensitization as a binary variable. Adiposity measures (%BF, BMI, %TF, and WC) and serum lipids (LDL, HDL, and triglycerides) were grouped into sex-specific quartiles for statistical analyses. Serum lipids were also analyzed as binary variables on the basis of clinical cutoff points: HDL < vs ≥ 40 mg/dL in men and < vs ≥ 50 mg/dL in women; LDL < vs ≥ 100 mg/dL; and triglycerides < vs ≥ 150 mg/dL.[24] BMI was categorized as BMI <23, 23 to 24.9 (overweight), and ≥25 (obesity) according to BMI cutoffs for obesity espoused by the World Health Organization (WHO) for Asian populations.[25] High WC was defined as ≥90 cm in men or ≥80 cm in women, the cutoffs for Asian populations.[26]

We fitted sex-stratified generalized estimating equation (GEE) logistic regression to examine the association of each adiposity or serum lipid measure with allergic sensitization. We included the following covariates that were either

TABLE I. Characteristics of 1187 Chinese participants age 18 to 39 years

	Men			Women		
	Any sensitization (n = 367)	Control (n = 286)	*P* value	Any sensitization (n = 196)	Control (n = 338)	*P* value
	Mean (SD)					
Age (y)	25.8 (7.1)	26.5 (7.2)	.238	27.7 (7.7)	27.3 (7.6)	.674
Weight (kg)	57.7 (8.6)	56.3 (7.6)	.053	50.4 (6.6)	49.9 (6.0)	.365
Height (cm)	164.3 (5.7)	163.2 (5.7)	.022	152.5 (5.3)	152.7 (5.2)	.671
BMI (kg/m^2)	21.3 (2.9)	21.1 (2.6)	.385	21.7 (2.5)	21.4 (2.4)	.246
Waist circumference (cm)	73.0 (8.6)	72.0 (7.9)	.190	70.7 (7.5)	70.3 (6.5)	.624
FMI	3.2 (2.1)	2.9 (1.9)	.086	6.4 (1.9)	6.2 (1.8)	.329
LMI	17.4 (1.4)	17.5 (1.3)	.552	14.5 (1.1)	14.4 (1.3)	.317
Total fat (kg)	8.8 (5.7)	7.8 (5.1)	.044	14.9 (4.6)	14.5 (4.1)	.355
%BF	14.4 (7.1)	13.2 (6.9)	.049	29.0 (5.7)	28.7 (5.6)	.483
Trunk fat (kg)	4.8 (3.5)	4.3 (3.2)	.090	7.7 (2.8)	7.4 (2.5)	.402
%TF	52.9 (5.9)	53.1 (5.9)	.603	50.7 (4.3)	50.6 (4.5)	.750
HDL (mg/dL)	65.1 (19.9)	67.0 (19.1)	.291	66.5 (20.0)	69.4 (20.2)	.149
LDL (mg/dL)	71.9 (24.4)	66.8 (24.2)	.016	70.6 (25.1)	69.8 (25.7)	.737
Triglycerides (mg/dL)	92.3 (66.2)	92.6 (61.9)	.960	77.6 (35.9)	76.8 (42.3)	.831
	n (%)					
Age						
18-24 y	214 (58.3)	154 (53.8)		91 (46.4)	169 (50.0)	
25-30 y	36 (9.8)	28 (9.8)		9 (4.6)	24 (7.1)	
30-35 y	54 (14.7)	46 (16.1)		46 (23.5)	65 (19.2)	
35-39 y	63 (17.2)	58 (20.3)	.658	50 (25.5)	80 (23.7)	.418
Education						
Primary school and lower	67 (18.5)	54 (19.1)		93 (47.7)	132 (39.1)	
Junior middle school	159 (43.9)	157 (55.5)		68 (34.9)	142 (42.0)	
High school	136 (37.6)	72 (25.4)	.003	34 (17.4)	64 (18.9)	.140
Occupation						
Farmer	27 (7.5)	20 (7.1)		50 (25.5)	73 (21.6)	
Others	335 (92.5)	263 (92.9)	.970	146 (74.5)	265 (78.4)	.353
Pet in household, yes	173 (47.9)	121 (42.8)	.220	87 (44.4)	151 (44.7)	1.000
Farm animal, yes	255 (71.8)	201 (73.1)	.794	143 (73.3)	232 (69.3)	.370
Mice in house						
No	142 (39.2)	110 (38.9)		65 (33.2)	121 (35.8)	
Yes, occasionally	147 (40.6)	113 (39.9)		87 (44.4)	154 (45.6)	
Yes, some or many	73 (20.2)	60 (21.2)	.948	44 (22.4)	63 (18.6)	.553
Cockroach in house						
No	214 (59.1)	195 (69.1)		120 (61.5)	187 (55.5)	
Yes, occasionally	115 (31.8)	71 (25.2)		58 (29.7)	123 (36.5)	
Yes, some or many	33 (9.1)	16 (5.7)	.025	17 (8.7)	27 (8.0)	.284
Passive smoking, yes	212 (59.7)	168 (59.8)	1.000	130 (67.7)	226 (67.9)	1.000
Current smoking, yes	152 (42.0)	129 (45.6)	.405	3 (1.5)	3 (0.9)	.805

χ^2 Test for categorical variables; linear regression model was used to test differences of each continuous variable means between the sensitization and the nonsensitization group within sex.

significant on univariate testing (Table I) or important for allergic sensitization or atopic diseases based on the literature: age (18-24, 25-39, 30-34, and 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (none, occasional, some, or many), and tobacco exposure (passive exposure for women).[3,27] We also tested the linear trends across quartiles of adiposity measures and lipid levels, and across BMI categories. To examine the independent effect of %BF and lipid levels on allergic sensitization, GEE logistic regressions were performed by including both variables simultaneously in the models. As secondary analyses, we also performed sex-specific GEE linear regression analysis, with adiposity and serum lipid measures as a function of allergic sensitization and the previously specified covariates to examine the differences in adiposity and lipid variables between sensitized and nonsensitized groups. This was carried out to evaluate the robustness of our findings. Finally, we also carried out secondary analyses to examine the association of adiposity measures and serum lipids with the subtypes of sensitization, sensitization to aeroallergens and sensitization to food allergens, separately.

To determine whether the relationship among measures of adiposity, serum lipid levels, and allergic sensitization differed between men and women, we tested the interactions between gender and each of the variables (sex × %BF-quartiles, sex × low HDL, and sex × LDL-quartiles) on the outcome of any sensitization. Because the interaction terms were all statistically significant, we presented all data with stratification by sex. We defined 2-tailed *P* values <.05 to be statistically significant. The statistical package SAS (version 9.1; SAS Institute, Cary, NC) was used for all theses analyses.

Finally, taking advantage of our twin design, we estimated the relative contributions of genetic and environmental influences on the observed associations between adiposity measures/serum lipids and allergic sensitization using structural equation modeling.[28] Of note, %BF and LDL were classified into low and high at sex-specific median of each variable and low HDL as <40 mg/dL in men and <50 mg/dL in women. Thus, all the tested phenotypes were binary variables. Specifically, we first fitted a saturated model (ACE model) that allowed for additive genetic (A), common/familial (C), and individual specific (E) environmental components for each of these

TABLE II. Association of adiposity measures with sensitization to any allergens in Chinese men and women age 18 to 39 years*

Adiposity measure quartiles	Any sensitization			Adiposity measure quartiles	Any sensitization		
	n (%)	Adjusted OR (95% CI)	*P* value		n (%)	Adjusted OR (95% CI)	*P* value
Men				**Women**			
%BF, mean ± SD (n)							
Q1 (163, 6.9 ± 1.1)	77 (47.2%)	Reference		Q1 (133, 21.7 ± 2.6)	49 (36.8%)	Reference	
Q2 (163, 9.9 ± 0.7)	93 (57.1%)	1.27 (0.80, 2.01)	.304	Q2 (134, 27.0 ± 1.3)	45 (33.6%)	0.92 (0.54, 1.58)	.767
Q3 (164, 14.3 ± 2.1)	95 (57.9%)	1.66 (1.02, 2.69)	.041	Q3 (134, 30.6 ± 0.9)	51 (38.1%)	1.12 (0.65, 1.93)	.681
Q4 (163, 24.4 ± 4.4)	102 (62.6%)	2.12 (1.28, 3.51)	.003	Q4 (133, 35.9 ± 3.0)	51 (38.4%)	1.14 (0.66, 1.97)	.639
Trend		*P* = .003		Trend		*P* = .509	
BMI, mean ± SD (n)							
Q1 (163, 18.2 ± 0.9)	98 (60.1%)	Reference		Q1 (133, 18.7 ± 0.9)	47 (35.3%)	Reference	
Q2 (163, 20.1 ± 0.5)	77 (47.2%)	0.60 (0.37, 0.98)	.041	Q2 (134, 20.5 ± 0.4)	47 (35.1%)	1.06 (0.64, 1.76)	.825
Q3 (164, 21.6 ± 0.6)	90 (54.9%)	0.96 (0.58, 1.57)	.857	Q3 (134, 21.9 ± 0.6)	50 (37.3%)	1.03 (0.60, 1.77)	.922
Q4 (163, 25.1 ± 2.1)	102 (62.6%)	1.39 (0.83, 2.33)	.206	Q4 (133, 24.9 ± 1.7)	52 (39.1%)	1.13 (0.63, 2.02)	.690
Trend		*P* = .085		Trend		*P* = .731	
BMI							
<23 (n = 512)	279 (54.5%)	Reference		<23 (n = 403)	145 (36.0%)	Reference	
23-24.9 (n = 77)	49 (63.6%)	1.67 (1.00, 2.80)	.049	23-24.9 (n = 87)	30 (34.5%)	0.90 (0.53, 1.52)	.684
>=25 (n = 64)	39 (60.9%)	1.57 (0.86, 2.83)	.139	≥25 (n = 44)	21 (47.7%)	1.56 (0.76, 3.21)	.228
Trend		*P* = .052		Trend		*P* = .432	
Model: FMI + LMI†							
High FMI (n = 327)	198 (60.6%)	1.74 (1.18, 2.55)	.005	High FMI (n = 267)	97 (36.3%)	0.97 (0.66, 1.44)	.895
High LMI (n = 327)	182 (55.7%)	0.98 (0.67, 1.41)	.896	High LMI (n = 267)	103 (38.6%)	1.05 (0.69, 1.60)	.824

Q, Quart-ile.
*All models were adjusted for age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), and smoking status (current smoking [yes/no] in men, passive smoking [yes/no] in women).
†Low vs high cut at sex-specific median of variables (LMI, FMI).

phenotypes. We also fitted alternative models where A, C, or E was equated to zero—that is, CE, AE, and AC models, respectively. χ^2 Goodness of fit and Akaike information criterion were used for comparison of goodness of fit of the models. We presented the estimates from the best fitted model, which had the lowest Akaike information criterion and did not have a significantly worse fit than the saturated model (ie, χ^2 test is not statistically significant with *P* value <.05). We also fitted the bivariate Cholesky decomposition models to calculate genetic (r_G), common, and nonshared environmental correlations (r_C and r_E) between allergic sensitization and adiposity measures/serum lipids. Mx software (http://www.psy.vu.nl/mxbib/) was used for these analyses.

RESULTS

Characteristics of study population

This study included 1187 participants (653 men and 534 women). Mean (SD) age was 26.1 (7.2) years for men and 27.4 (7.6) years for women. Participants were generally lean with a mean (SD) BMI of 21.2 (2.8) in men and 21.5 (2.5) in women. Mean (SD) %BF was 13.9 (7.1) in men and 28.8 (6.0) in women. Twelve percent of men and 16.3% of women were overweight, and 9.8% of men and 8.2% of women were obese, based on the WHO criteria for Asians.[25] Eleven percent of men and the same proportion of women had LDL ≥100 mg/dL, whereas 6.8% of men and 18.5% of women had low HDL (<40 mg/dL for men, <50 mg/dL for women). The participants had mean BMI and serum lipid levels comparable to a nontwin adult population from the study area.[29]

Overall, 47.4% (n = 563) of the participants had positive SPT to at least 1 tested allergen. The sex-specific prevalence of any sensitization was 56.2% in men and 36.7% in women. Similarly, the sex-specific prevalence of sensitization to aeroallergens was 50.5% in men and 31.5% in women. The prevalence of sensitization to food was relatively low (23.3% in men and 18.0% in women; see this article's Table E1 in the Online Repository at www.jacionline.org).

Compared with nonsensitized men, men with any sensitization were slightly taller, were more likely to have had a high school education, and had higher levels of total BF, %BF, and serum LDL (Table I). They also reported more cockroaches in the home (Table I). After adjusting for age, education, occupation, cockroaches in the house, and smoking status, men with any sensitization had a 1.6 higher mean %BF (95% CI, 0.4-2.8), 1.5 cm higher mean WC (95% CI, 0.3-2.8), and 4.9 mg/dL higher mean LDL (95% CI, 0.9-8.9) than men without any sensitization. Among women, there were no significant differences in all the listed variables, including adiposity measures and serum lipid levels, between sensitized and nonsensitized groups.

Relationship of %BF, BMI, and central adiposity with allergic sensitization

As shown in Table II, there was a dose-response relationship between %BF and the risk of any sensitization (P_{trend} = .003) among men. Compared with the lowest quartile of %BF, the odds ratio (OR) for any sensitization was 1.27 (95% CI, 0.80-2.01) for the second quartile of %BF; 1.66 (1.02-2.69) for the third quartile; and 2.12 (1.28-3.51) for the fourth quartile. No associations between %BF and any sensitization were seen in women.

We observed a nonlinear relationship between BMI and any sensitization in men (Table II). Compared with men in the lowest quartile, men in the second quartile had lower risk for any sensitization (OR = 0.60; 95% CI, 0.37-0.98), whereas men in the third and fourth quartiles were at comparable or higher risk of any sensitization, respectively. However, when using clinical cut-points

TABLE III. Association of serum lipids with sensitization to any allergen in Chinese men and women age 18 to 39 years*

	Any sensitization		
Serum lipids (mg/dL)	**Cases**	**Adjusted OR (95% CI)**	***P* value**
Male	**n (%)**		
HDL			
Q4 (163, 92.3 ±0 012.1)	89 (54.6%)	Reference	
Q3 (161, 70.7 ± 4.2)	94 (58.4%)	1.13 (0.70, 1.82)	.618
Q2 (163, 57.5 ± 3.7)	89 (54.6%)	0.90 (0.55, 1.47)	.676
Q1 (163, 43.1 ± 5.7)	94 (57.7%)	0.99 (0.59, 1.64)	.954
Trend			$P = .735$
HDL			
≥40 (n = 606)	329 (54.3%)	Reference	
<40 (n = 44)	37 (84.1%)	4.01 (1.75, 9.20)	.001
LDL			
Q1 (162, 41.7 ± 9.7)	80 (49.4%)	Reference	
Q2 (163, 60.7 ± 4.4)	83 (50.9%)	1.03 (0.66, 1.62)	.887
Q3 (163, 74.4 ± 4.2)	104 (63.8%)	1.79 (1.11, 2.90)	.017
Q4 (162, 101.8 ± 18.4)	99 (61.1%)	1.66 (1.03, 2.67)	.037
Trend			$P = .007$
LDL			
<100 (n = 580)	320 (55.2%)	Reference	
≥100 (n = 70)	46 (65.7%)	1.69 (0.94, 3.03)	.081
Female			
HDL			
Q4 (131, 96.2 ± 13.5)	40 (30.5%)	Reference	
Q3 (132, 72.7 ± 3.8)	46 (34.9%)	1.25 (0.72, 2.17)	.431
Q2 (135, 59.5 ± 3.6)	53 (39.3%)	1.51 (0.86, 2.66)	.148
Q1 (133, 45.5 ± 5.8)	57 (42.9%)	1.91 (1.07, 3.40)	.028
Trend			$P = .023$
HDL			
≥50 (n = 433)	153 (35.3%)	Reference	
<50 (n = 98)	43 (43.9%)	1.54 (0.93, 2.55)	.093
LDL			
Q1 (132, 39.9 ± 8.8)	47 (35.6%)	Reference	
Q2 (133, 60.6 ± 5.3)	48 (36.1%)	0.95 (0.56, 1.61)	.859
Q3 (133, 76.7 ± 4.7)	46 (34.6%)	0.95 (0.56, 1.62)	.852
Q4 (133, 102.9 ± 18.5)	55 (41.4%)	1.27 (0.73, 2.24)	.400
Trend			$P = .439$
LDL			
<100 (n = 471)	171 (36.3%)	Reference	
≥100 (n = 60)	25 (41.7%)	1.29 (0.68, 2.46)	.434

Q, Quartile.
Means ± SDs (all such values).
*All models were adjusted for age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), and smoking status (current smoking [yes/no] in men, passive smoking [yes/no] in women).

for BMI as defined by the WHO for Asian populations,[25] both overweight and obesity appeared to be associated with higher risk of any sensitization in men with OR (95% CI) of 1.67 (1.00-2.80) and 1.57 (0.86-2.83), but no dose-response association was observed (P trend = .052). The OR for any sensitization was 1.62 (95% CI, 1.06-2.48) when combining overweight and obesity in men. Interestingly, when analyzing FMI and LMI, the 2 components of BMI, we found that high FMI was associated with any sensitization (OR = 1.74; 95% CI, 1.18-2.55), but not for LMI (OR = 0.98; 95% CI, 0.67-1.41). None of these adiposity measures were associated with any sensitization in women (Table II).

Similar results were observed for sensitization to food allergens and to aeroallergens in both sexes (see this article's Table E2 in the Online Repository at www.jacionline.org).

No associations between central adiposity (quartile %TF, quartile WC, or high WC) and any sensitization were observed in either sex (data not shown).

Relationships of serum HDL, LDL, and triglycerides with allergic sensitization

As shown in Table III, HDL <40 mg/dL was associated with a 4 times higher risk of any sensitization (95% CI, 1.75-9.20) in men. This pattern was also seen for both food sensitization (OR = 4.50; 95% CI, 1.74-11.65) and aeroallergen sensitization (OR = 3.86; 95% CI, 1.65-9.00) in men (see this article's Table E3 in the Online Repository at www.jacionline.org). In women, an inverse association was observed between quartile of HDL and any sensitization (Table III; P trend = .023) and food sensitization (P trend = .006; Table E3). A similar pattern was found for aeroallergen sensitization but was not significant (Table E3).

In men, higher serum LDL was associated with increased odds of any sensitization (Table III), and also to aeroallergen, but was not significantly associated with food sensitization (Table E3). In men, the third and fourth quartiles of LDL had 1.79-fold (95% CI, 1.11-2.90) and 1.66-fold (95% CI, 1.03-2.67) increased odds of

TABLE IV. Association of serum lipids and %BF with sensitization to any allergen in Chinese men and women age 18 to 39 years*

	Any sensitization			
	Male		Female	
Serum lipids and %BF	OR (95% CI)	*P* value	OR (95% CI)	*P* value
HDL + %BF				
Model 1				
HDL Q4	1.00		1.00	
HDL Q3	1.13 (0.69, 1.86)	.625	1.25 (0.72, 2.17)	.430
HDL Q2	0.86 (0.52, 1.42)	.557	1.51 (0.86, 2.65)	.154
HDL Q1	0.93 (0.55, 1.57)	.791	1.88 (1.05, 3.38)	.034
%BF Q1	1.00		1.00	
%BF Q2	1.30 (0.82, 2.06)	.270	0.92 (0.53, 1.59)	.758
%BF Q3	1.69 (1.04, 2.75)	.033	1.09 (0.64, 1.89)	.744
%BF Q4	2.15 (1.29, 3.58)	.003	1.09 (0.62, 1.90)	.764
Model 2				
HDL <40 male	3.70 (1.61, 8.48)	.002	1.52 (0.91, 2.53)	.112
HDL <50 female				
%BF Q1	1.00		1.00	
%BF Q2	1.24 (0.78, 1.97)	.357	0.92 (0.54, 1.58)	.771
%BF Q3	1.64 (1.01, 2.67)	.047	1.11 (0.64, 1.91)	.707
%BF Q4	1.94 (1.18, 3.21)	.010	1.11 (0.64, 1.92)	.718
LDL + %BF				
Model 1				
LDL Q1	1.00		1.00	
LDL Q2	1.00 (0.64, 1.57)	.999	0.95 (0.56, 1.61)	.859
LDL Q3	1.70 (1.05, 2.76)	.032	0.95 (0.56, 1.63)	.863
LDL Q4	1.39 (0.85, 2.30)	.192	1.26 (0.72, 2.22)	.423
%BF Q1	1.00		1.00	
%BF Q2	1.29 (0.82, 2.05)	.272	0.90 (0.52, 1.56)	.711
%BF Q3	1.66 (1.02, 2.72)	.043	1.08 (0.63, 1.86)	.771
%BF Q4	1.88 (1.11, 3.19)	.019	1.12 (0.65, 1.95)	.682
Model 2				
LDL >100	1.41 (0.77, 2.59)	.267	1.26 (0.66, 2.41)	.485
%BF Q1	1.00		1.00	
%BF Q2	1.26 (0.80, 1.99)	.323	0.91 (0.53, 1.58)	.751
%BF Q3	1.65 (1.02, 2.68)	.043	1.09 (0.64, 1.87)	.753
%BF Q4	1.94 (1.16, 3.24)	.011	1.14 (0.66, 1.99)	.637

Q, Quartile.
*All models were adjusted for age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), and smoking status (current smoking [yes/no] in men, passive smoking [yes/no] in women).

any sensitization compared with the lowest quartiles of LDL (*P* trend = .007 after adjustment; Table III). No such associations were noted in women.

No associations were seen when LDL was categorized to <100 vs ≥100 mg/dL in either sex (all $P > .05$; Tables III and E3). No associations were observed between serum triglycerides and sensitization (to foods, to aeroallergens, or any sensitization) in either sex (data not shown).

Relationships of %BF and serum lipids with allergic sensitization after mutual adjustment of these variables

With increasing %BF quartile, serum LDL and triglyceride levels also increased, whereas HDL levels were lower in both sexes (see this article's Table E4 in the Online Repository at www.jacionline.org). Thus it is possible that one of the associations of adiposity or lipid levels with sensitization may be a result of the correlation between adiposity and lipids. We further investigated whether the associations of %BF and lipids with any sensitization were still present after the %BF and lipids were adjusted for one another and other covariates (Table IV). The pattern of associations was similar to that assessed by modeling %BF and lipids without mutual adjustment, but the magnitude of the associations was slightly attenuated.

Secondary analysis to test of robustness of these associations by linear regression modeling

As described in Methods, we performed sex-specific linear regression analysis, with adiposity and serum lipid measures as a function of allergic sensitization. These findings, as reported in this article's Table E5 in the Online Repository at www.jacionline.org, show the same relationships as our primary analysis, confirming the robustness of our findings. The only difference in this linear analysis with increased power was the positive association between waist circumference and any sensitization in men (β = 1.5 cm; 95% CI, 1.0-2.8; $P = .02$).

Test for sex differences in the associations

We tested the interaction of sex with %BF quartiles, sex with low HDL, and sex with LDL quartiles in relation to allergic sensitization. We found that the relationships between allergic sensitization and each of the variables (%BF, HDL, and LDL) differed significantly by sex. Specifically, as shown in this

TABLE V. Genetic (r_G), common (r_C), and individual environmental correlations (r_E) between low HDL, high LDL, high %BF, and any sensitization in 149 monozygotic and 89 dizygotic male twin pairs*

Trait	A	C	E	r_G	r_C	r_E
HDL-sensitization						
ACE models						
Any sensitization	0.65 (0.60-0.80)	0.00 (0.00-0.06)	0.35 (0.29-0.44)			
Low HDL	0.46 (0.25-0.46)	0.45 (0.00-0.66)	0.10 (0.02-0.28)	0.51 (0.17-0.51)	-1.00 (-1.00 to 1.00)	0.68 (−0.21 to 0.99)
AE models						
Any sensitization	0.65 (0.47-0.80)	—	0.35 (0.24-0.53)			
Low HDL	0.91 (0.74-0.98)	—	0.09 (0.03-0.26)	0.31 (−0.01 to 0.58)	—	0.71 (−0.16 to 0.99)
LDL-sensitization						
ACE models						
Any sensitization	0.65 (0.09-0.80)	0.01 (0.00-0.49)	0.34 (0.20-0.53)			
High LDL	0.47 (0.00-0.81)	0.33 (0.00-0.76)	0.20 (0.10-0.35)	0.34 (−1.00 to 1.00)	1.00 (-1.00 to 1.00)	-0.12 (−0.56 to 0.33)
AE models						
Any sensitization	0.66 (0.47-0.80)	—	0.34 (0.20-0.53)			
High LDL	0.81 (0.68-0.90)	—	0.19 (0.10-0.32)	0.33 (0.11-0.55)	—	-0.14 (−0.57 to 0.31)
%BF-sensitization						
ACE model						
Any sensitization	0.65 (0.07-0.80)	0.01 (0.00-0.50)	0.35 (0.20-0.54)			
High %BF	0.74 (0.17-0.89)	0.06 (0.00-0.57)	0.20 (0.11-0.36)	0.17 (−0.55 to 0.91)	1.00 (-1.00 to 1.00)	0.03 (−0.35 to 0.41)
AE model						
Any sensitization	0.65 (0.47-0.80)	—	0.35 (0.20-0.53)		—	
High %BF	0.80 (0.65-0.89)	—	0.20 (0.11-0.35)	0.19 (−0.04 to 0.41)	—	0.03 (−0.35 to 0.40)

The genetic (A), common environment (C), and individual specific environment (E) components for each phenotype were similar to those from univariate genetic models, but they were not identical because bivariate analysis included covariance between 2 variables examined.
*High and low cut at sex-specific median of each variable (LDL and %BF), except for HDL, which uses a clinical cut-point (HDL < 40 mg/dL).

article's Table E6 in the Online Repository at www.jacionline.org, *P* values for sex interaction with %BF were .046 to .0002 depending on quartiles of %BF; in this article's Table E7 in the Online Repository at www.jacionline.org, the *P* value for sex interaction with low HDL was <.0001; and in this article's Table E8 in the Online Repository at www.jacionline.org, the *P* value for sex interaction with high LDL varied from .025 to <.0001 depending on quartiles of serum LDL.

Genetic and environmental contributions to observed associations

Individually, allergic sensitization, high %BF, low HDL, and high LDL were phenotypes that were influenced by genetic and environmental factors (see this article's Table E9 in the Online Repository at www.jacionline.org). This was reflected by the higher tetrachoric correlation (which measures the within-pair similarity of the binary traits) in monozygotic twins than in dizygotic twins for any sensitization (0.67 vs 0.30), HDL (0.91 vs 0.69), LDL (0.79 vs 0.57), and %BF (0.80 vs 0.43). The heritability estimate from the best fitted model (AE model) was 65% for any sensitization and 80% for high %BF. Genetic (A) and common environment (C) components together explained about 80% to 90% of the variance of low HDL and high LDL.

We further examined the degree to which genetic and environmental influences contributed to the observed associations among %BF, lipids, and allergic sensitization in men (Table V; full data in this article's Table E10 in the Online Repository at www.jacionline.org). Bivariate Cholesky decomposition models revealed some marginal and some statistically significant genetic correlations for allergic sensitization and low HDL (r_G = 0.31; 95% CI, –0.01, 0.58), allergic sensitization and high %BF (r_G = 0.19; 95% CI, –0.04, 0.41), and allergic sensitization and high LDL (r_G = 0.33; 95% CI, 0.11, 0.55). This indicates that these paired traits might share some common genetic factors. The corresponding environmental correlations were 0.71, 0.03, and –0.14, which were not statistically significant. We had also carried out the same analysis in women despite the fact that we did not find any association among %BF, lipid levels, and allergic sensitization. As expected, neither genetic nor environmental correlations among %BF, serum lipids, and allergic sensitization were found in women.

DISCUSSION

To our knowledge, this is the first study to examine directly the association of %BF as measured by DEXA with allergic sensitization. We found %BF was associated with allergic sensitization in men but not women. Also, this study demonstrated an inverse association between HDL and allergic sensitization in both sexes, whereas LDL was associated with higher risk of allergic sensitization in men, even after adjusting for %BF. These associations can be partially explained by shared common genetic factors that may be involved in both the development of allergic sensitization and the regulation of %BF and serum lipid levels.

Previous epidemiologic studies of BMI and allergic sensitization in adults have yielded equivocal findings.[8,10,12] This may be in part a result of the use of BMI, a surrogate measure of adiposity, as opposed to a direct measure of adiposity. For example, no association was found between BMI and allergic sensitization in rural Australia,[12] whereas BMI >24.8 was found to be associated with about a 1.5-fold higher risk of allergic sensitization in a Finnish study.[10] Consistent with previous studies, we found unstable associations between BMI and allergic sensitization. However, when we evaluated FMI instead of BMI, a positive FMI-sensitization association was observed in men, after controlling for LMI. In keeping with this finding, our study also demonstrated a persistent positive association between %BF and allergic sensitization in men but not in women. Our BMI data as well as findings of previous studies may be a consequence in part of the limitation of BMI as a general adiposity measure. This underscores the importance of direct adiposity measures in evaluating the relationship between adiposity and allergic sensitization.

The sex differences in the effects of adiposity and serum lipid profiles on allergic sensitization were pronounced in this study. There are a number of potential explanations for this finding. From a biologic standpoint, previous studies have found sex differences in the production of IgE,[3] T-cell polarization,[20] and lipid profiles.[18] Estrogen increases HDL concentration in women, and testosterone decreases HDL in both sexes.[18] However, the exact mechanism underlying the sex differences of these associations cannot be fully explained by the current literature. From a methodologic standpoint, it is also possible that the limited variation of %BF in the women did not allow for detection of associations seen in the men. The coefficient of variation was 51.1% in men and 20.8% in women.

In this study, we found an inverse association between HDL and allergic sensitization. In previous studies, both positive and negative associations between HDL and sensitization have been reported.[14,15] HDL was found to be associated with a lower risk of allergic sensitization in children but not in adults in the Third National Health and Nutrition Examination Survey.[14] In contrast, higher HDL was associated with greater risk of allergic sensitization in adults in a German study, but the association disappeared after controlling for age and sex.[15] There are similar issues in previous studies of the association between LDL and allergic sensitization.[14,15] Higher LDL levels were associated with a lower prevalence of allergic sensitization in the German study,[15] but no association was found in NHANES III.[14] In our study, a positive association of LDL quartiles and allergic sensitization was observed in men but not women. No associations were observed between triglycerides and sensitization in either sex, which was consistent with results of previous studies.[14] Differences in the association between lipid levels and allergic sensitization between studies may be partly a result of differences between sexes and how each of the studies accounted for this difference. We feel, given the marked sex differences, that stratification by sex is the most prudent approach.

Using our unique twin study design, we showed that common genetic factors may contribute to the observed associations between %BF, HDL, LDL, and allergic sensitization. Shared genetic influences between 2 traits may result in a concomitant rise of both phenomena in response to environmental changes. Notably, shared and nonshared environmental correlations among HDL, LDL, and allergic sensitization were not statistically significant. It is possible that this might be a result of the limited number of twin pairs (n = 238) with data available on zygosity in addition to the variables of interest (SPT, adiposity, and serum lipid levels). In ACE models, we have limited power to determine relative importance of genetic (A) and common environmental (C) effects on low HDL and high LDL. The A+C component explained about 80% to 90% of the variance of low HDL and high LDL, whereas the individual environmental component (E) explained 9% (95% CI, 2% to 28%) of the variance in low HDL and 21% (95% CI, 11% to 36%) of the variance in high LDL. Taken together, it appears that both genetic and environmental factors may have also played a role in determining the magnitude of the observed associations.

Our findings may have important public health implications. These results provide a potential explanation for the phenomenon of increasing prevalence of allergic sensitization or allergic diseases in Asian immigrants commensurate with length of stay in Westernized countries regardless of age at arrival.[30] This increase might be in part a result of that transition to a Westernized nutrition and lifestyle from an original Asian lifestyle, and environment increases the prevalence of obesity and increases the risk of low HDL, high LDL, and allergic sensitization in Asian immigrants,[31] especially those genetically susceptible to both low HDL (or high LDL) and allergic sensitization.

Previous studies have suggested that adiposity predisposes to asthma, an atopic disease, but not vice versa.[5] However, the associations between asthma and adiposity may not be the same as those between sensitization and adiposity. In this cross-sectional study, body fat was measured in adulthood, whereas sensitization might have occurred in childhood. Another possible explanation of our findings is that allergic sensitization increases the risk of adiposity or abnormalities in lipid profiles. Further longitudinal studies are needed to evaluate the temporal relationship of these phenotypes.

This study has several strengths. First, body composition (such as %BF) was measured by DEXA, a technique that can accurately assess total BF.[13] This community-based sample with a relatively low prevalence of obesity and high LDL and triglycerides allowed us to investigate the relation among adiposity, lipids, and sensitization in mostly healthy subjects.

This may allow the elucidation of relationships that may be obscured in predominantly obese populations. Because these are clinically asymptomatic subjects, our findings were less likely confounded by lipid-lowering medication use. Finally, our twin design offers the opportunity to examine whether genetic influences contribute to the associations among %BF, lipids, and allergic sensitization. Such an analysis would not be possible in a general population.

The study also has limitations. First, our findings may not be generalizable to affluent urban populations or populations with a higher level of obesity. Second, this is a cross-sectional analysis that precludes any temporal or cause-effect conclusions. Third, this quantitative genetic study only provides estimates of the degree to which genes influence variation in each trait between subjects. This design does not identify specific genes or address associated mechanisms. Further studies are needed to determine which specific genes, environmental factors, or gene-environmental interactions contribute to the correlation of allergic sensitization, serum lipid levels, and adiposity.

In summary, in this lean Chinese population, higher %BF, lower HDL, and higher LDL were associated with increased risk of allergic sensitization in men. We also found evidence for a

common genetic element in this association. With the exception of HDL, no significant associations were found in women. These findings suggest a sex-specific link among adiposity, serum lipids, and allergic sensitization. Continued follow-up of this cohort may help determine the temporal relationships between adiposity, serum lipids, and allergic sensitization. These findings may have relevance in understanding novel factors related to the etiology of allergic diseases, and may have implications for disease prevention.

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Clinical implications: Higher %BF and serum lipid disturbances (lower HDL and higher LDL) are associated with an increased risk of allergic sensitization in a sex-specific manner.

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TABLE E1. Prevalence of sensitization to food and aeroallergen by sex among 1187 Chinese participants age 18 to 39 years

Sensitization	Men, n (%)	Women, n (%)	*P* value
Any food	152 (23.3)	96 (18.0)	.031
Shellfish	103 (15.8)	56 (10.5)	.010
Peanut	70 (10.7)	41 (7.7)	.091
Soy	23 (3.5)	13 (2.4)	.359
Egg white	22 (3.4)	12 (2.2)	.328
Sesame	19 (2.9)	7 (1.3)	.094
Fish	15 (2.3)	7 (1.3)	.300
Walnut	15 (2.3)	6 (1.1)	.192
Wheat	7 (1.1)	7 (1.3)	.913
Milk	3 (0.5)	6 (1.1)	.329
Any aeroallergen	330 (50.5)	168 (31.5)	<.001
Dust mite	258 (39.5)	133 (24.9)	<.001
Cockroach	253 (38.7)	114 (21.3)	<.001
Alternaria	55 (8.4)	30 (5.6)	.080
Dog epithelia	18 (2.8)	9 (1.7)	.300
Cat hair	10 (1.5)	8 (1.5)	1.000
Any sensitization	367 (56.2)	196 (36.7)	<.001

TABLE E2. Association of adiposity measures with sensitization to food, aeroallergen in Chinese men and women age 18 to 39 years*

	Sensitization to aeroallergen			Sensitization to food		
Adiposity measures	**n (%)**	**Adjusted OR (95% CI)**	***P* value**	**n (%)**	**Adjusted OR (95% CI)**	***P* value**
Men						
%BF						
Q1 (low)	71 (45.2%)	1.00		32 (27.1%)	1.00	
Q2	82 (54.0%)	1.19 (0.74, 1.89)	.474	41 (36.9%)	1.41 (0.77, 2.60)	.268
Q3	85 (55.2%)	1.61 (0.98, 2.64)	.062	41 (37.3%)	1.79 (0.97, 3.30)	.062
Q4 (high)	92 (60.1%)	2.09 (1.25, 3.51)	.005	38 (38.4%)	1.96 (1.03, 3.75)	.041
Trend			.004			.028
BMI						
Q1 (low)	89 (57.8%)	1.00		46 (41.4%)	1.00	
Q2	70 (44.9%)	0.61 (0.37, 0.99)	.047	31 (26.5%)	0.49 (0.27, 0.92)	.027
Q3	77 (51.0%)	0.91 (0.55, 1.52)	.716	32 (30.2%)	0.72 (0.39, 1.32)	.287
Q4 (high)	94 (60.7%)	1.43 (0.85, 2.43)	.180	43 (41.4%)	1.31 (0.70, 2.46)	.391
Trend			.086			.370
BMI						
<23	247 (51.5%)	1.00		118 (33.6%)	1.00	
23-24.9	45 (61.6%)	1.78 (1.06, 3.00)	.029	21 (42.9%)	1.78 (0.94, 3.40)	.079
≥25	38 (60.3%)	1.72 (0.94, 3.14)	.077	13 (34.2%)	1.29 (0.57, 2.93)	.536
Trend			.024			.250
Model: FMI + LMI						
High FMI		1.74 (1.17, 2.58)	.006		1.74 (1.08, 2.81)	.023
High LMI		0.96 (0.66, 1.41)	.844		0.94 (0.59, 1.49)	.793
Women						
%BF						
Q1 (low)	41 (32.8%)	1.00		23 (21.5%)	1.00	
Q2	43 (32.6%)	1.07 (0.60, 1.88)	.826	24 (21.2%)	0.99 (0.52, 1.90)	.982
Q3	43 (34.1%)	1.14 (0.65, 2.03)	.646	22 (21.0%)	1.04 (0.52, 2.05)	.915
Q4 (high)	41 (33.3%)	1.10 (0.62, 1.98)	.739	27 (24.8%)	1.25 (0.63, 2.48)	.519
Trend			.695			.519
BMI						
Q1 (low)	41 (32.3%)	1.00		24 (21.8%)	1.00	
Q2	40 (31.5%)	1.05 (0.62, 1.79)	.854	22 (20.2%)	0.90 (0.48, 1.84)	.849
Q3	46 (35.4%)	1.07 (0.60, 1.91)	.807	24 (22.2%)	0.90 (0.47, 1.89)	.864
Q4 (high)	41 (33.6%)	1.03 (0.55, 1.92)	.921	26 (24.3%)	1.00 (0.48, 2.10)	.995
trend			.908			.991
BMI						
<23	128 (33.2%)	1.00		70 (21.3%)	1	
23-24.9	23 (28.8%)	0.79 (0.44, 1.41)	.429	15 (20.8%)	0.85 (0.43, 1.67)	.634
≥25	17 (42.5%)	1.42 (0.66, 3.05)	.371	11 (32.4%)	1.60 (0.65, 3.95)	.304
Trend			.706			.534
Model: FMI + LMI						
High FMI		0.93 (0.61, 1.40)	.727		0.97 (0.60, 1.56)	.893
High LMI		1.06 (0.68, 1.64)	.804		0.80 (0.45, 1.41)	.443

Q, Quartile.

*All models were adjusted for age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), and smoking status (current smoking [yes/no] in men, passive smoking [yes/no] in women).

TABLE E3. Association of serum lipids with sensitization to food and aeroallergen in Chinese men and women age 18 to 39 years*

	Sensitization to aeroallergen			Sensitization to food		
Serum lipids (mg/dL)	**Case n (%)**	**Adjusted OR (95% CI)**	***P* value**	**Case n (%)**	**Adjusted OR (95% CI)**	***P* value**
Men						
HDL						
Q4 (high)	81 (52.3%)	1.00		31 (29.5%)	1.00	
Q3	87 (56.5%)	1.16 (0.71, 1.88)	.548	40 (37.4%)	1.38 (0.74, 2.57)	.306
Q2	79 (51.6%)	0.89 (0.54, 1.46)	.641	37 (33.3%)	1.04 (0.55, 1.99)	.900
Q1 (low)	82 (54.3%)	0.95 (0.56, 1.60)	.846	44 (38.9%)	1.37 (0.71, 2.64)	.342
Trend			.606			.537
HDL						
HDL ≥40	297 (51.7%)	1.00		135 (32.8%)	1.00	
HDL <40	32 (82.1%)	3.86 (1.65, 9.00)	.002	17 (70.8%)	4.50 (1.74, 11.65)	.002
LDL						
Q1 (low)	68 (45.3%)	1.00		36 (30.5%)	1.00	
Q2	76 (48.7%)	1.12 (0.71, 1.76)	.630	34 (29.8%)	0.92 (0.50, 1.68)	.777
Q3	95 (61.7%)	1.95 (1.19, 3.19)	.008	43 (42.2%)	1.61 (0.87, 2.98)	.128
Q4 (high)	90 (58.8%)	1.80 (1.11, 2.93)	.017	39 (38.2%)	1.44 (0.75, 2.74)	.270
Trend			.003		1.18 (0.96, 1.45)	.117
LDL						
LDL <100	288 (52.6%)	1.00		135 (34.2%)	1.00	
LDL ≥100	41 (63.1%)	1.70 (0.93, 3.10)	.086	17 (41.5%)	1.48 (0.69, 3.17)	.310
Women						
HDL						
Q4 (high)	37 (28.9%)	1.00		13 (12.5%)	1.00	
Q3	38 (30.7%)	1.11 (0.63,1 .98)	.716	25 (22.5%)	2.10 (0.97, 4.58)	.061
Q2	45 (35.4%)	1.39 (0.78, 2.49)	.260	25 (23.4%)	2.17 (0.97, 4.90)	.061
Q1 (low)	48 (38.7%)	1.72 (0.94, 3.15)	.077	33 (30.3%)	3.17 (1.43, 7.03)	.005
Trend			.059			.006
HDL						
HDL ≥50	131 (31.9%)	1.00		70 (20.0%)	1.00	
HDL <50	37 (40.2%)	1.53 (0.90, 2.59)	.117	26 (32.1%)	1.90 (1.05, 3.46)	.035
LDL						
Q1 (low)	44 (34.1%)	1.00		19 (18.3%)	1.00	
Q2	41 (32.5%)	0.85 (0.49, 1.47)	.565	23 (21.3%)	1.22 (0.61, 2.45)	.569
Q3	36 (29.3%)	0.81 (0.46, 1.43)	.462	22 (20.2%)	1.09 (0.54, 2.21)	.802
Q4 (high)	47 (37.6%)	1.16 (0.65, 2.07)	.616	32 (29.1%)	1.74 (0.87, 3.49)	.117
Trend			.692			.158
LDL						
LDL <100	148 (33.0%)	1.00		81 (21.3%)	1	
LDL ≥100	20 (36.4%)	1.18 (0.60, 2.30)	.628	15 (30.0%)	1.50 (0.74, 3.07)	.261

Q, Quartile.

*All models were adjusted for age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), and smoking status (current smoking [yes/no] in men, passive smoking [yes/no] in women).

TABLE E4. Mean (SD) percent body fat (%BF), BMI, and serum lipid profiles by quartiles (Q) of %BF

	Quartiles of %BF				
	Q1	**Q2**	**Q3**	**Q4**	***P* trend***
Men					
N	163	163	164	163	
Age (y)	26.1 (7.3)	23.3 (6.0)	26.6 (7.3)	28.4 (7.1)	.0012
BMI (kg/m^2)	19.5 (1.5)	19.7 (1.5)	21.2 (1.7)	24.6 (2.5)	<.0001
%BF	6.9 (1.1)	9.9 (0.7)	14.3 (2.1)	24.4 (4.4)	<.0001
HDL (mg/dL)	68.4 (20.0)	65.6 (20.3)	67.1 (19.9)	62.4 (17.5)	.0386
LDL (mg/dL)	61.7 (17.1)	64.4 (22.3)	68.5 (22.1)	84.0 (28.5)	<.0001
Trigylicerides (mg/dL)	68.3 (31.4)	74 (28.4)	86.1 (44.3)	141.4 (97.5)	<.0001
Women					
N	133	134	134	133	
Age (y)	29.1 (7.4)	26.6 (7.3)	25.8 (7.7)	28.3 (7.8)	.3993
BMI (kg/m^2)	19.6 (1.7)	20.7 (1.6)	21.6 (1.9)	24.0 (2.3)	<.0001
%BF	21.7 (2.6)	27.0 (1.3)	30.6 (0.9)	35.9 (3.0)	<.0001
HDL (mg/dL)	71.2 (20.2)	70.0 (20.7)	68.0 (19.0)	64.3 (20.3)	.0136
LDL (mg/dL)	63.4 (20.6)	69.3 (23.3)	72.8 (25.0)	74.9 (30.8)	.0013
Triglycerides (mg/dL)	69.6 (39.5)	68.4 (27.5)	78.2 (31.4)	92.2 (52.9)	.0002

*Linear test for trend for each variable across quartiles of %BF. GEEs were applied.

TABLE E5. Association of sensitization with the measures of adiposity measure and serum lipid levels on the basis of linear regression*

	Male		Female	
	β (95% CI)	*P* value	β (95% CI)	*P* value
	%BF			
Nonsensitized	Reference		Reference	
Any sensitization	1.60 (0.43, 2.77)	.008	0.50 (−0.58, 1.58)	.362
	BMI (kg/m2)			
Nonsensitized	Reference		Reference	
Any sensitization	0.42 (-0.02, 0.86)	.063	0.22 (−0.26, 0.70)	.373
	WC (cm)			
Nonsensitized	Reference		Reference	
Any sensitization	1.54 (0.25, 2.82)	.020	0.20 (−1.11, 1.50)	.768
	%TF			
Nonsensitized	Reference		Reference	
Any sensitization	−0.06 (−0.99, 0.86)	.892	0.20 (−0.64, .04)	.642
	HDL (mg/dL)			
Nonsensitized	Reference		Reference	
Any sensitization	−0.96 (−4.23, 2.32)	.566	−3.20 (−7.16, 0.75)	.112
	LDL (mg/dL)			
Nonsensitized	Reference		Reference	
Any sensitization	4.90 (0.93, 8.88)	.016	0.71 (−4.41, 5.82)	.787
	Triglycerides (mg/dL)			
Nonsensitized	Reference		Reference	
Any sensitization	0.22 (−10.66, 11.10)	.969	−0.13 (−7.30, 7.05)	.973

*All models were adjusted for age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), and smoking status (current smoking [yes/no] in men, passive smoking [yes/no] in women).

TABLE E6. The interaction between quartiles (Q1-Q4) of %BF and sex in the association of %BF with any sensitization

	Any sensitization		
	OR	**95% CI**	***P* value**
%BF			
Q1 (low)	1.00		
Q2	1.31	0.84, 2.05	.2396
Q3	1.59	0.99, 2.57	.0559
Q4 (high)	2.10	1.28, 3.47	.0036
Q1 * sex	0.58	0.34, 0.99	.0455
Q2 * sex	0.39	0.23, 0.68	.0008
Q3 * sex	0.40	0.23, 0.70	.0014
Q4 * sex	0.33	0.18, 0.59	.0002

Q, Quartile.
Sex: 0 = male, 1 = female.

$$y = \beta_0 + \beta_1 * Q_2 + \beta_2 * Q_3 + \beta_3 * Q_4 + \beta_4 * Q_1 * sex + \beta_5 * Q_2 * sex + \beta_6 * Q_3 * sex + \beta_7 * Q_4 * sex + \text{covariates}$$

Covariates in the model were age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), smoking status (yes/no), and passive smoking (yes/no).

TABLE E7. The interaction between low HDL (HDL <40 male; <50 female) and sex in the association of low HDL with any sensitization

	Any sensitization		
	OR	**95% CI**	***P* value**
HDL			
Normal	1.00		
Low	3.98	1.72, 9.22	.0013
Normal HDL * sex	0.42	0.30, 0.59	<.0001
Low HDL * sex	0.15	0.06, 0.39	<.0001

Sex: 0 = male, 1 = female.

$$y = \beta_0 + \beta_1 * lowHDL + \beta_2 * normalHDL * sex + \beta_3 * lowHDL * sex + \text{covariates}$$

Covariates in the model were age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), smoking status (yes/no), and passive smoking (yes/no).

TABLE E8. The interaction between quartiles (Q1-Q4) of LDL and sex in the association of LDL with any sensitization

	Any sensitization		
	OR	**95% CI**	***P* value**
LDL			
Q1 (low)	1.00		
Q2	1.10	0.70, 1.71	.6857
Q3	1.83	1.14, 2.96	.0129
Q4 (high)	1.80	1.12, 2.88	.0146
Q1 * sex	0.55	0.33, 0.93	.0252
Q2 * sex	0.47	0.27, 0.81	.0073
Q3 * sex	0.29	0.17, 0.50	<.0001
Q4 * sex	0.41	0.23, 0.72	.002

Q, Quartile.
Sex: 0 = male, 1 = female.

$$y = \beta_0 + \beta_1 * Q_2 + \beta_2 * Q_3 + \beta_3 * Q_4 + \beta_4 * Q_1 * sex + \beta_5 * Q_2 * sex + \beta_6 * Q_3 * sex + \beta_7 * Q_4 * sex + \text{covariates}$$

Covariates in the model were age (18-24, 25-29, 30-34, 35-39 years), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/nonfarmer), cockroach in house (no, occasional, some or many), smoking status (yes/no), and passive smoking (yes/no).

TABLE E9. Estimates of genetic (A), common environment (C), and individual specific environment (E) effects on any sensitization, low HDL, high LDL and high %BF from univariate structural equation models in 149 monozygotic and 89 dizygotic male twin pairs*

	Intrapair correlation (SE)†		Parameter estimates			Test of model fit		
Models	**Monozygotic**	**Dizygotic**	**A (95% CI)**	**C (95% CI)**	**E (95% CI)**	χ^2	***P* value**	**AIC**
Any sensitization								
ACE	0.67 (0.09)	0.30 (0.16)	0.65 (0.06-0.79)	0.00 (0.00-0.51)	0.35 (0.21-0.54)	—	—	—
AE			0.65 (0.46-0.79)	—	0.35 (0.21-0.54)	0.00	—	–2.00
CE			—	0.53 (0.36-0.67)	0.47 (0.33-0.64)	4.53	0.033	2.53
Low HDL								
ACE	0.91 (0.06)	0.69 (0.19)	0.43 (0.00-0.97)	0.47 (0.00-0.92)	0.09 (0.02-0.28)	—	—	—
AE			0.91 (0.75-0.98)	—	0.09 (0.02-0.25)	1.16	0.282	–0.84
CE			—	0.85 (0.67-0.94)	0.15 (0.06-0.33)	1.85	0.174	–0.15
High LDL								
ACE	0.79 (0.06)	0.57 (0.13)	0.44 (0.00-0.88)	0.35 (0.00-0.78)	0.21 (0.11-0.36)	—	—	—
AE			0.81 (0.67-0.90)	—	0.19 (0.10-0.33)	1.52	0.218	–0.48
CE			—	0.72 (0.58-0.82)	0.28 (0.18-0.42)	2.82	0.093	0.82
High %BF								
ACE	0.80 (0.06)	0.43 (0.14)	0.73 (0.16-0.89)	0.06 (0.0-0.58)	0.21 (0.11-0.36)	—	—	—
AE			0.80 (0.65-0.89)	—	0.20 (0.11-0.35)	0.04	0.834	–1.96
CE			—	0.67 (0.53-0.79)	0.33 (0.21-0.47)	6.39	0.011	4.39

AIC, Akaike information criterion (a global measure of goodness of fit of models).
The best fitting models are in boldface.
*High and low cut at sex-specific median of each variable (LDL and %BF) except for HDL, which used a clinical cut-point (HDL <40 mg/dL).
†Tetrachoric correlation (SE).

TABLE E10. Contribution of genes (C_{GCP}), common (C_{CCP}), and unique (C_{UCP}) environment to the correlation among lipids, %BF, and any sensitization in 149 monozygotic and 89 dizygotic male twin pairs*

Trait	A	C	E	r_G	r_C	r_E	r_{TP}	C_{GCP}	C_{CCP}	C_{UCP}
HDL-sensitization										
ACE models										
Any sensitization	0.65 (0.60-0.80)	0.00 (0.00-0.06)	0.35 (0.29-0.44)							
Low HDL	0.46 (0.25-0.46)	0.45 (0.00-0.66)	0.10 (0.02-0.28)	0.51 (0.17 to 0.51)	−1.00 (−1.00 to 1.00)	0.68 (−0.21 to 0.99)	0.41	0.28	0.00	0.13
AE models										
Any sensitization	0.65 (0.47-0.80)	—	0.35 (0.24-0.53)							
Low HDL	0.91 (0.74-0.98)	—	0.09 (0.03-0.26)	0.31 (−0.01 to 0.58)	—	0.71 (−0.16 to 0.99)	0.37	0.24	—	0.13
LDL-sensitization										
ACE models										
Any sensitization	0.65 (0.09-0.80)	0.01 (0.00-0.49)	0.34 (0.20-0.53)							
High LDL	0.47 (0.00-0.81)	0.33 (0.00-0.76)	0.20 (0.10-0.35)	0.34 (−1.00 to 1.00)	1.00 (-1.00 to 1.00)	−0.12 (−0.56 to 0.33)	0.22	0.19	0.06	−0.03
AE models										
Any sensitization	0.66 (0.47-0.80)	—	0.34 (0.20-0.53)							
High LDL	0.81 (0.68-0.90)	—	0.19 (0.10-0.32)	0.33 (0.11-0.55)	—	-0.14 (−0.57 to 0.31)	0.20	0.24	—	−0.04
%BF-sensitization										
ACE model										
Any sensitization	0.65 (0.07-0.80)	0.01 (0.00-0.50)	0.35 (0.20-0.54)							
High %BF	0.74 (0.17-0.89)	0.06 (0.00-0.57)	0.20 (0.11-0.36)	0.17 (−0.55 to 0.91)	1.00 (-1.00 to 1.00)	0.03 (−0.35 to 0.41)	0.15	0.12	0.02	0.01
AE model										
Any sensitization	0.65 (0.47-0.80)	—	0.35 (0.20-0.53)		—					
High %BF	0.80 (0.65-0.89)	—	0.20 (0.11-0.35)	0.19 (−0.04 to 0.41)	—	0.03 (−0.35 to 0.40)	0.15	0.14	—	0.01

r_G, Genetic correlation between 2 phenotypes; r_C, common environmental correlation; r_E, unique environmental correlation between 2 phenotypes; r_{TP} phenotype correlation between %BF/serum lipid and allergic sensitization. $\mathbf{r}_{TP=}\ \mathbf{r}_{G*}\sqrt{A_1}*A_2+\ \mathbf{r}_{C*}\sqrt{C_1}*C_2+\ \mathbf{r}_{E*}\sqrt{E_1}*E_2$; $C_{GCP=}\ \mathbf{r}_{G*}\sqrt{A_1}*A_2$; $C_{CCP=}\ \mathbf{r}_{C*}\sqrt{C_1}*C_2$; $C_{UCP}\ =r_{E*}\sqrt{E_1}*E_2$.
The A,C, and E were similar to those from univariate genetic models, but they were not identical because bivariate analysis included covariance between 2 variables examined.
*High and low cut at sex-specific median of each variable (LDL and %BF) except for HDL, which used a clinical cut-point (HDL <40 mg/dL).