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

Fengxiu Ouyang, MD, PhD[a], **Rajesh Kumar, MD**[b], **Jacqueline Pongracic, MD**[b], **Rachel E. Story, MD**[b], **Xin Liu, MD, PhD**[a], **Binyan Wang, MD, PhD**[a], **Houxun Xing, MD, MS**[c], **Xue Liu**[c], **Zhiping Li, MD**[c], **Wenbin Zhang, MD**[c], **Yaping Fang, MD**[a,c], **Shanchun Zhang, MD**[a], **Xiping Xu, MD, PhD**[d], and **Xiaobin Wang, MD, ScD**[a]

a*Mary Ann and J. Milburn Smith Child Health Research Program, Department of Pediatrics, Northwestern University Feinberg School of Medicine and Children's Memorial Hospital and Children's Memorial Research Center, Chicago, IL, USA*

b*Division of Allergy and Immunology, Department of Pediatrics, Northwestern University Feinberg School of Medicine and Children's Memorial Hospital, IL, USA*

c*Institute for Biomedicine, Anhui Medical University, Hefei, China*

d*Center for Population Genetics, University of Illinois at Chicago School of Public Health, Chicago, IL*

Abstract

Background—Obesity and allergic diseases have increased dramatically in recent decades. While 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 1,187 rural Chinese twins (653 men) aged 18-39 years, with skin prick tests (SPT), anthropometric and DEXA-assessed adiposity measures, and lipid assessments. Allergic sensitization was defined as positive SPT to ≥1 allergen (9 foods and 5 aeroallergens tested). We applied gender-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—Males had lower percent body fat (%BF) (13.9% vs. 28.8%) but higher rates of allergic sensitization (56.2% vs. 36.7%) than females. Males in the highest %BF quartile were 2.1 times more likely sensitized than the lowest quartile (95%CI 1.3-3.5, P-trend=0.003). In males, the risk of allergic sensitization increased with HDL<40 mg/dl (OR=4.0, 95%CI 1.8-9.2) and higher LDL quartiles (P-trend=0.007). This appeared to be partially explained by shared genetic factors between serum lipid

‡Correspondence and reprint requests should be addressed to: Xiaobin Wang, MD, ScD, Mary Ann and J. Milburn Smith Child Health Research Program, Children's Memorial Hospital and Children's Memorial Research Center; Department of Pediatrics, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA; telephone:312-573-7738, fax:312-573-7825, e-mail: E-mail: xbwang@childrensmemorial.org.

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

Capsule summary: This community-based study in rural China suggests a gender-specific link between adiposity, serum lipids and allergic sensitization. It also suggests that there may be common genetic influences on allergic sensitization and serum lipid levels.

levels and allergic sensitization. In females, lower HDL was associated with increased risk of allergic sensitization.

Conclusions—In this relatively lean Chinese population, higher %BF, lower HDL and higher LDL were associated with greater risk of allergic sensitization, most notable in males. The observed associations between adiposity, serum lipids and allergic sensitization in males appear to be partially explained by common genetic influences on these traits.

Keywords

DEXA; Body mass index; Adiposity; Serum lipids; Sensitization

Introduction

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 epidemiological studies have evaluated the relationship between percent body fat (%BF) and allergic sensitization. This is important since %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, since adiposity is associated with disturbances in lipid profiles, it would be important to determine if lipid profiles are independently associated with allergic sensitization or mediate an association of adiposity with allergic sensitization.[16] The two 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 gender issues.[15]

The role of gender in the association between adiposity and allergic sensitization needs further investigation. Body composition and serum lipid levels vary with gender. Females have higher %BF and higher HDL than males.[17, 18] Similarly, there are gender differences in prevalence of allergic sensitization [8, 19] and in regulation of Th1 and Th2 cytokines.[20] Therefore, it is not surprising that some studies have found associations between BMI and allergic sensitization to be gender dependent. [11]

Most prior 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 which 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 gender-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 utilized the twin design to examine the relative contribution of genetic and environmental factors to any associations observed.

Methods

Study Population

This study utilized data obtained from an ongoing study of metabolic syndrome in a large Chinese twin cohort which 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 eight counties of the Anqing region of China in 1998-2000 and the follow-up study was conducted in 2005-2007. This report used the data obtained at the follow-up survey from the participants aged 18-39 years. Anqing has a total population of 6.1 million (10% urban and 90% rural). For both baseline and follow-up study, 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 exam, skin prick testing (SPT), DEXA scan, blood draw, and questionnaire interview. The latter obtained pertinent epidemiological 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 aged 18-39 years.

Zygosity Ascertainment

Zygosity was determined by microsatellite probes, or 'DNA fingerprinting' techniques, which has 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 (MZ) and 180 pairs (male/male 89, female/female 52, male/female 39) were dizygotic (DZ).

Anthropometric and Adiposity Assessments

Body weight and height were measured during physical examination 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 two components, fat mass index (FMI = BF/height^2) and lean mass index (LMI = (Weight − BF)/height^2).[23]

A standard whole-body scan was performed by DEXA (GE-lunar Prodigy, USA) to measure total body fat and trunk fat (the latter defined as: chest, abdomen and pelvis). [21, 22] %BF was calculated as: %BF = (total BF/body weight) × 100. Percent trunk fat (%TF) was calculated as: %TF = (TF/total BF) × 100.

BMI and %BF were used as surrogate measures of general adiposity, while 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 (TG) were measured by enzymatic methods (Boehringer Mannheim, Mannheim, Germany); high density lipoproteins (HDL) by the same enzymatic method after precipitation

with dextran sulphate/magnesium chloride. Low density lipoprotein (LDL) was calculated using formula: LDL=Total Cholesterol-HDL-TG/5.

Skin prick testing (SPT)

SPT was performed on the volar surfaces of the arms on normal skin using Multi-Test II (Lincoln Diagnostics, Decatur, IL). Participants were tested to 14 allergens, including 5 aeroallergens (*Alternaria tenius*, house dust mite mix [equal parts mixture of *D. farinae* and *D. 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 was considered invalid and thus excluded (n=31) if the saline control was ≥3 mm, the histamine control was <3 mm, or if 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 one aeroallergen or food. Secondary outcomes were "any sensitization to at least one aeroallergen", or "any sensitization to at least one 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 TG) were grouped into gender-specific quartiles for statistical analyses. Serum lipids were also analyzed as binary variables based on clinical cutoff points: HDL < vs. ≥ 40mg/dL in males and < vs. ≥ 50mg/dL in females; LDL < vs. ≥ 100 mg/dL; and TG < vs. ≥ 150mg/dl.[24] BMI was categorized as: BMI <23, 23-24.9 (overweight), and ≥25 (obesity) according to BMI cut offs for obesity espoused by the WHO for Asian populations.[25] High WC was defined as ≥ 90 cm in males, or ≥ 80 cm in females, the cut offs for Asian populations.[26]

We fitted gender-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 significant on univariate testing (Table 1), 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/non-farmer), 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 gender-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 non-sensitized 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 to sensitization to food allergens, separately.

To determine whether the relationship between measures of adiposity, serum lipid levels and allergic sensitization differed between males and females, we tested the interactions between gender and each of the variables (gender × %BF-quartiles, gender × low HDL, and gender × LDL-quartiles) on the outcome of any sensitization. Since the interaction terms were all statistically significant, we presented all data with stratification by gender. We defined two-tailed *p* values <0.05 to be statistically significant. The statistical package SAS (version_9.1 SAS Institute, Cary, North Carolina) was used for all the above analysis.

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 gender-specific median of each variable and low HDL as < 40mg/dL in males and <50mg/dl in females. 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 above phenotypes. We also fitted alternative models where *A*, *C*, or E was equated to zero, i.e. CE, AE, and AC models, respectively. Chi-square goodness of fit and Akaike Information Criterio (AIC) were used for comparison of goodness of fit of the models. We presented the estimates from the best-fitted model, which had the lowest AIC and did not have a significantly worse fit compared to the saturated model (i.e. Chi-square test is not statistically significant with p-value < 0.05). We also fitted the Bivariate Cholesky decomposition models to calculate genetic (r_G), common and non-shared 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 the above 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, 27.4 (7.6) years for women. Participants were generally lean with 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, while 6.8% of men and 18.5% of women had low HDL (<40 mg/dL for males, <50 mg/dL for females). The participants had comparable mean BMI and serum lipid levels to a non-twin adult population from the study area.[29]

Overall 47.4% (n=563) of the participants had positive SPT to at least one tested allergen. The gender-specific prevalence of any sensitization was 56.2% in males and 36.7% in females. Similarly, the gender-specific prevalence of sensitization to aeroallergens was 50.5% in males and 31.5% in females. The prevalence of sensitization to food was relatively low (23.3% in males and 18.0% in females). (online Table E1).

Compared to non-sensitized males, males with any sensitization were slightly taller, more likely to have had a high school education, and had higher levels of total body fat mass, %BF and serum LDL (Table I). They also reported more cockroaches in the home (Table I). After adjusting for age, education, occupation, cockroach in house and smoking status, males 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 males without any sensitization. Among females, there were no significant differences in all the listed variables, including adiposity measures and serum lipids levels, between sensitized and non-sensitized 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}=0.003) among males. Compared to the lowest quartile of %BF, odds ratio (OR) for any sensitization was 1.27 (95% confidence interval (CI): 0.80-2.01) for the 2nd quartile of %BF; 1.66 (1.02-2.69) for the 3rd quartile; and 2.12 (1.28-3.51) for the 4th quartile. No associations between %BF and any sensitization were seen in females.

We observed a non-linear relationship between BMI and any sensitization in males (Table II). Compared to those in the lowest quartile, men in the 2nd quartile had lower risk for any sensitization (OR=0.60, 95%CI: 0.37-0.98), while men in the 3rd and 4th quartiles were at comparable or higher risk of any sensitization, respectively. However, when using clinical cut-points 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 males 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=0.052). 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 two 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 females (Table II).

Similar results were observed for sensitization to food allergens and to aeroallergens in both genders (online Table E2).

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

Relationships of serum HDL, LDL and TG with allergic sensitization

As shown in table III, HDL <40 mg/dL was associated with a four times higher risk of any sensitization (95% CI 1.75-9.20) in males. 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 males (online table E3). In females, an inverse association was observed between quartile of HDL and any sensitization (Table III, p-trend=0.023) and food sensitization (p-trend=0.006) (online table E3). A similar pattern was found for aeroallergen sensitization but was not significant (online Table E3).

In males, 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 (online Table E3). In males, the 3rd and 4th quartile 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 any sensitization compared to the lowest quartiles of LDL (p-trend = 0.007, after adjustment) (Table III). No such associations were noted in females.

No associations were seen when LDL was categorized to <100 vs. ≥100 mg/dL in either gender (all p > 0.05, Table III and online Table E3). No associations were observed between serum TG and sensitization (to foods, to aeroallergens, or any sensitization) in either gender (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 TG levels also increased while HDL levels were lower in both genders (online table E4). Thus it is possible that one of the associations of adiposity or lipid levels with sensitization may be due to 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 were slightly attenuated.

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

We, as described in the methods section, performed gender-specific linear regression analysis, with adiposity and serum lipid measures as a function of allergic sensitization. These findings, as reported in Online Table E5, 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 males (β=1.5 cm, 95%CI 1.0-2.8; p=0.02).

Test for gender differences in the associations

We tested the interaction of gender with %BF quartiles, gender with low HDL, and gender 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 gender. Specifically, as shown in Table E6, p values for gender interaction with %BF were 0.046 to 0.0002 depending on quartiles of %BF; in Table E7, p value for gender interaction with low HDL was <0.0001; and in Table E8, p value for gender interaction with high LDL varied from 0.025 to <0.0001 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 (Table E9). This was reflected by the higher tetrachoric correlation (which measures the within-pair similarity of the binary traits) in MZ twins than in DZ 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 80-90% 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 between %BF, lipids and allergic sensitization in males (Table V, full data in Online Table E10). 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), 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, -0.14, which were not statistically significant. We had also carried out the same analysis in females despite the fact that we did not find any association between %BF, lipid levels and allergic sensitization. As expected, neither genetic nor environmental correlations between %BF, serum lipids and allergic sensitization were found in females.

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 males but not females. Also, this study demonstrated an inverse association between HDL

and allergic sensitization in both genders while LDL was associated with higher risk of allergic sensitization in males, even after adjusting for %BF. These associations can be partially explained by shared common genetic factors which may be involved in both the development of allergic sensitization and the regulation of %BF and serum lipid levels.

Prior epidemiologic studies of BMI and allergic sensitization in adults have yielded equivocal findings. [8, 10, 12] This may be in part due to 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] while BMI > 24.8 was found to be associated with about 1.5-fold higher risk of allergic sensitization in a Finnish study.[10] Consistent with prior studies, we found unstable associations between BMI and allergic sensitization. However, when we evaluated FMI (fat mass index) instead of BMI, a positive FMI-sensitization association was observed in males, after controlling for LMI. In keeping with this finding, our study also demonstrated a persistent positive association between %BF and allergic sensitization in males but not in females. Our BMI data, as well as findings of previous studies may, in part, be a consequence 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 gender 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 gender differences in the production of IgE,[3] T cell polarization[20] and also in lipid profiles.[18] Estrogen increases HDL concentration in females and testosterone decreases HDL in both genders.[18] However, the exact mechanism underlying the gender 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 females did not allow for detection of associations seen in the males. The coefficient of variation (CV) was 51.1% in males, and 20.8% in females.

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 NHANES III.[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 gender.[15] There are similar issues in prior 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 males but not females. No associations were observed between TG and sensitization in either gender, which was consistent with results of previous studies.[14] Differences in the association between lipid levels and allergic sensitization between studies may be partly due to differences between genders and how each of the studies accounted for this difference. We feel, given the marked gender differences, that stratification by gender 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 two traits may result in a concomitant rise of both phenomena in response to environmental changes. Notably, shared- and non-shared environmental correlations between HDL, LDL and allergic sensitization were not statistically significant. It is possible that this might due to 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 80-90% of the variance of low HDL and high LDL, while the individual environmental component (E) explained 9% (95%CI: 2-28%) of the variance of low HDL and 21% (95%CI: 11-36%) of the variance of 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 due in part to 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 while 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 TG allowed us to investigate the relation between adiposity, lipids and sensitization among mostly healthy subjects. This may allow for the elucidation of relationships which may be obscured in predominantly obese populations. Since 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 between %BF, lipids and allergic sensitization. Such an analysis would not be possible in a general population.

The study also has limitations. (1) Our findings may not be generalizable to affluent urban populations or populations with a higher level of obesity. (2) This is a cross-sectional analysis which precludes any temporal or cause-effect conclusions. (3) 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 males. We also found evidence for a common genetic element in this association. With the exception of HDL, no significant associations were found in females. These findings suggest a gender-specific link between 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 findngs may have relevance in understanding novel factors related to the etiology of allergic diseases, and may have implications for disease prevention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Abbreviations

BF
: fat mass

%BF
: percent body fat

BMI
: body mass index

CV
: coefficient of variation

DEXA
: dual-energy X-ray absorptiometry

FMI
: fat mass index

GEE
: generalized estimating equations

HDL
: high density lipoproteins

LDL
: low density lipoprotein

LM
: lean mass

LMI
: lean mass index

OR
: odds ratio

SD
: standard deviation

SE
: standard error

SPT

skin prick tests

%TF

percent trunk fat

TG

triglycerides

WC

waist circumference

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Table I

Characteristics of 1187 Chinese participants aged 18-39 years.

	Male			Female		
	Any Sensitization (n=367)	**Control (n=286)**	**p value**	**Any Sensitization (n=196)**	**Control (n=338)**	**p value**
	Mean (SD)					
Age (years)	25.8 (7.1)	26.5 (7.2)	0.238	27.7 (7.7)	27.3 (7.6)	0.674
Weight (kg)	57.7 (8.6)	56.3 (7.6)	0.053	50.4 (6.6)	49.9 (6.0)	0.365
Height (cm)	164.3 (5.7)	163.2 (5.7)	0.022	152.5 (5.3)	152.7 (5.2)	0.671
BMI (kg/m^2)	21.3 (2.9)	21.1 (2.6)	0.385	21.7 (2.5)	21.4 (2.4)	0.246
Waist circumference (cm)	73.0 (8.6)	72.0 (7.9)	0.190	70.7 (7.5)	70.3 (6.5)	0.624
FMI	3.2 (2.1)	2.9 (1.9)	0.086	6.4 (1.9)	6.2 (1.8)	0.329
LMI	17.4 (1.4)	17.5 (1.3)	0.552	14.5 (1.1)	14.4 (1.3)	0.317
Total fat (kg)	8.8 (5.7)	7.8 (5.1)	0.044	14.9 (4.6)	14.5 (4.1)	0.355
%BF	14.4 (7.1)	13.2 (6.9)	0.049	29.0 (5.7)	28.7 (5.6)	0.483
Trunk fat (kg)	4.8 (3.5)	4.3 (3.2)	0.090	7.7 (2.8)	7.4 (2.5)	0.402
%TF	52.9 (5.9)	53.1 (5.9)	0.603	50.7 (4.3)	50.6 (4.5)	0.750
HDL (mg/dL)	65.1 (19.9)	67.0 (19.1)	0.291	66.5 (20.0)	69.4 (20.2)	0.149
LDL (mg/dL)	71.9 (24.4)	66.8 (24.2)	0.016	70.6 (25.1)	69.8 (25.7)	0.737
TG (mg/dL)	92.3 (66.2)	92.6 (61.9)	0.960	77.6 (35.9)	76.8 (42.3)	0.831
	n (%)					
Age						
18-24	214(58.3)	154(53.8)		91(46.4)	169(50.0)	
25-30	36(9.8)	28(9.8)		9(4.6)	24(7.1)	
30-35	54(14.7)	46(16.1)		46(23.5)	65(19.2)	
35-39	63(17.2)	58(20.3)	0.658	50(25.5)	80(23.7)	0.418
Education						
Primary school & 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)	0.003	34(17.4)	64(18.9)	0.140
Occupation						

	Male			Female		
	Any Sensitization (n=367)	**Control (n=286)**	**p value**	**Any Sensitization (n=196)**	**Control (n=338)**	**p value**
Farmer	27(7.5)	20(7.1)		50(25.5)	73(21.6)	
Others	335(92.5)	263(92.9)	0.970	146(74.5)	265(78.4)	0.353
Pet in household, yes	173(47.9)	121(42.8)	0.220	87(44.4)	151(44.7)	1.000
Farm animal, yes	255(71.8)	201(73.1)	0.794	143(73.3)	232(69.3)	0.370
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)	0.948	44(22.4)	63(18.6)	0.553
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)	0.025	17(8.7)	27(8.0)	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)	0.405	3(1.5)	3(0.9)	0.805

Chi-square test for categorical variables; linear regression model was used to test differences of each continuous variable means between the sensitization and the non-sensitization group within gender.

Table II

Association of adiposity measures with sensitization to any allergens in Chinese men and women aged 18-39 years[1].

Adiposity Measure Quartiles	Any Sensitization			Adiposity measure Quartiles	Any Sensitization		
	n (%)	Adjusted OR (95% CI)	p-value		n (%)	Adjusted OR (95% CI)	p-value
male				**female**			
%BF (n, mean ± SD)							
Q1 (163, 6.9 ± 1.1)	77 (47.2%)	Ref.		Q1 (133, 21.7 ± 2.6)	49 (36.8%)	Ref.	
Q2 (163, 9.9 ± 0.7)	93 (57.1%)	1.27 (0.80,2.01)	0.304	Q2 (134, 27.0 ± 1.3)	45 (33.6%)	0.92 (0.54,1.58)	0.767
Q3 (164, 14.3 ± 2.1)	95 (57.9%)	1.66 (1.02,2.69)	0.041	Q3 (134, 30.6 ± 0.9)	51 (38.1%)	1.12 (0.65,1.93)	0.681
Q4 (163, 24.4 ± 4.4)	102 (62.6%)	2.12 (1.28,3.51)	0.003	Q4 (133, 35.9 ± 3.0)	51 (38.4%)	1.14 (0.66,1.97)	0.639
trend		p=0.003		trend		p=0.509	
BMI (n, mean ± SD)							
Q1 (163, 18.2 ± 0.9)	98 (60.1%)	Ref.		Q1 (133, 18.7 ± 0.9)	47 (35.3%)	Ref.	
Q2 (163, 20.1 ± 0.5)	77 (47.2%)	0.60 (0.37,0.98)	0.041	Q2 (134, 20.5 ± 0.4)	47 (35.1%)	1.06 (0.64,1.76)	0.825
Q3 (164, 21.6 ± 0.6)	90 (54.9%)	0.96 (0.58,1.57)	0.857	Q3 (134, 21.9 ± 0.6)	50 (37.3%)	1.03 (0.60,1.77)	0.922
Q4 (163, 25.1 ± 2.1)	102 (62.6%)	1.39 (0.83,2.33)	0.206	Q4 (133, 24.9 ± 1.7)	52 (39.1%)	1.13 (0.63,2.02)	0.690
trend		p=0.085		trend		p=0.731	
BMI							
<23 (n=512)	279 (54.5%)	Ref.		<23 (n=403)	145 (36.0%)	Ref.	
23-24.9 (n=77)	49 (63.6%)	1.67 (1.00,2.80)	0.049	23-24.9 (n=87)	30 (34.5%)	0.90 (0.53,1.52)	0.684
>=25 (n=64)	39 (60.9%)	1.57 (0.86,2.83)	0.139	>=25 (n=44)	21 (47.7%)	1.56 (0.76,3.21)	0.228
trend		p=0.052		trend		p=0.432	
Model: FMI+LMI [2]							
High FMI (n=327)	198 (60.6%)	1.74 (1.18,2.55)	0.005	High FMI (n=267)	97 (36.3%)	0.97 (0.66,1.44)	0.895
High LMI (n=327)	182 (55.7%)	0.98 (0.67,1.41)	0.896	High LMI (n=267)	103 (38.6%)	1.05 (0.69,1.60)	0.824

[1] all models were adjusted for age (18-24, 25-29, 30-34, 35-39), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/non-farmer), Cockroach in house (no, occasional, some or many) and smoking status (current smoking (yes/no) in male, passive smoking(yes/no) in female).

[2] low vs. high cut at gender-specific median of variables (LMI, FMI)

Table III

Association of serum lipids with sensitization to any allergen in Chinese men and women aged 18-39 years[1].

Serum lipids (mg/dL)	Any Sensitization		
	Cases	Adjusted OR (95% CI)	p-value
Male	n (%)		
HDL			
Q4 (163, 92.3±12.1)	89 (54.6%)	Ref.	
Q3 (161, 70.7 ± 4.2)	94 (58.4%)	1.13 (0.70,1.82)	0.618
Q2 (163, 57.5 ± 3.7)	89 (54.6%)	0.90 (0.55,1.47)	0.676
Q1 (163, 43.1 ± 5.7)	94 (57.7%)	0.99 (0.59,1.64)	0.954
Trend			p=0.735
HDL			
≥ 40 (n=606)	329 (54.3%)	Ref.	
<40 (n=44)	37(84.1%)	4.01 (1.75,9.20)	0.001
LDL			
Q1 (162, 41.7 ± 9.7)	80 (49.4%)	Ref.	
Q2 (163, 60.7 ± 4.4)	83 (50.9%)	1.03 (0.66,1.62)	0.887
Q3 (163, 74.4 ± 4.2)	104 (63.8%)	1.79 (1.11,2.90)	0.017
Q4 (162, 101.8±18.4)	99 (61.1%)	1.66 (1.03,2.67)	0.037
Trend			p=0.007
LDL			
<100 (n=580)	320 (55.2%)	Ref.	
≥ 100 (n=70)	46 (65.7%)	1.69 (0.94,3.03)	0.081
Female			
HDL			
Q4 (131, 96.2±13.5)	40 (30.5%)	Ref.	
Q3 (132, 72.7 ± 3.8)	46 (34.9%)	1.25 (0.72,2.17)	0.431
Q2 (135, 59.5 ± 3.6)	53 (39.3%)	1.51 (0.86,2.66)	0.148
Q1 (133, 45.5 ± 5.8)	57 (42.9%)	1.91 (1.07,3.40)	0.028
Trend			p=0.023
HDL			
≥ 50 (n=433)	153 (35.3%)	Ref.	
<50 (n=98)	43 (43.9%)	1.54 (0.93,2.55)	0.093
LDL			
Q1 (132, 39.9 ± 8.8)	47 (35.6%)	Ref.	
Q2 (133, 60.6 ± 5.3)	48 (36.1%)	0.95 (0.56,1.61)	0.859
Q3 (133, 76.7 ± 4.7)	46 (34.6%)	0.95 (0.56,1.62)	0.852
Q4 (133, 102.9±18.5)	55 (41.4%)	1.27 (0.73,2.24)	0.400
Trend			p=0.439
LDL			
<100 (n=471)	171(36.3%)	Ref.	
≥ 100 (n=60)	25 (41.7%)	1.29 (0.68,2.46)	0.434

mean ± SD (all such values)

[1] all models were adjusted for age (18-24, 25-29, 30-34, 35-39), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/non-farmer), Cockroach in house (no, occasional, some or many) and smoking status (current smoking (yes/no) in male, passive smoking(yes/no) in female).

Table IV

Association of serum lipids and %BF with sensitization to any allergen in Chinese men and women aged 18-39 years[1].

Serum Lipids and %BF	Any Sensitization			
	Male		Female	
	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)	0.625	1.25 (0.72,2.17)	0.430
HDL Q2	0.86 (0.52,1.42)	0.557	1.51 (0.86,2.65)	0.154
HDL Q1	0.93 (0.55,1.57)	0.791	1.88 (1.05,3.38)	0.034
%BF Q1	1.00		1.00	
%BF Q2	1.30 (0.82,2.06)	0.270	0.92 (0.53,1.59)	0.758
%BF Q3	1.69 (1.04,2.75)	0.033	1.09 (0.64,1.89)	0.744
%BF Q4	2.15 (1.29,3.58)	0.003	1.09 (0.62,1.90)	0.764
Model 2				
HDL <40 male; HDL <50 female	3.70 (1.61,8.48)	0.002	1.52 (0.91,2.53)	0.112
%BF Q1	1.00		1.00	
%BF Q2	1.24 (0.78,1.97)	0.357	0.92 (0.54,1.58)	0.771
%BF Q3	1.64 (1.01,2.67)	0.047	1.11 (0.64,1.91)	0.707
%BF Q4	1.94 (1.18,3.21)	0.010	1.11 (0.64,1.92)	0.718
LDL + %BF				
Model 1				
LDL Q1	1.00		1.00	
LDL Q2	1.00 (0.64,1.57)	0.999	0.95 (0.56,1.61)	0.859
LDL Q3	1.70 (1.05,2.76)	0.032	0.95 (0.56,1.63)	0.863
LDL Q4	1.39 (0.85,2.30)	0.192	1.26 (0.72,2.22)	0.423
%BF Q1	1.00		1.00	
%BF Q2	1.29 (0.82,2.05)	0.272	0.90 (0.52,1.56)	0.711
%BF Q3	1.66 (1.02,2.72)	0.043	1.08 (0.63,1.86)	0.771
%BF Q4	1.88 (1.11,3.19)	0.019	1.12 (0.65,1.95)	0.682
Model 2				
LDL> 100	1.41 (0.77,2.59)	0.267	1.26 (0.66,2.41)	0.485
%BF Q1	1.00		1.00	
%BF Q2	1.26 (0.80,1.99)	0.323	0.91 (0.53,1.58)	0.751
%BF Q3	1.65 (1.02,2.68)	0.043	1.09 (0.64,1.87)	0.753

Serum Lipids and %BF	Any Sensitization			
	Male		Female	
	OR (%95_CI)	p value	OR (%95_CI)	p value
%BF Q4	1.94 (1.16,3.24)	0.011	1.14 (0.66,1.99)	0.637

1 all models were adjusted for age (18-24, 25-29, 30-34, 35-39), education (primary school or lower, junior middle school, high school or higher), occupation (farmer/non-farmer), Cockroach in house (no, occasional, some or many) and smoking status (current smoking (yes/no) in male, passive smoking(yes/no) in female).

Table V

Genetic (r_G), common and individual environmental correlations (r_C and r_E) between low HDL, high LDL, high %BF and any sensitization in 149 MZ and 89 DZ **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 (-1.00-1.00)	0.68 (-0.21-0.99)
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 (-0.01-0.58)	--	0.71 (-0.16-0.99)
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 (-1.00-1.00)	1.00 (-1.00-1.00)	-0.12 (-0.56-0.33)
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 (-0.57-0.31)
%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 (-0.55-0.91)	1.00 (-1.00-1.00)	0.03 (-0.35-0.41)
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 (-0.04-0.41)	--	0.03 (-0.35-0.40)

* High and low cut at gender-specific median of each variable (LDL and %BF) except for HDL which use a clinical cut point (HDL < 40 mg/dL).

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 two variables examined.