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Specific Allergic Sensitization in Parents and Their 18 year-old Offspring in the Suburban Detroit Childhood Allergy Study

Rana Tawil Misiak, MD¹, Ganesa Wegienka, PhD¹, Suzanne Havstad, MS¹, Dennis R. Ownby, MD², Christine Cole Johnson, PhD¹, and Edward M. Zoratti, MD¹

¹Henry Ford Hospital, Detroit, MI

²Medical College of Georgia, Augusta, GA

Abstract

Background—Allergic sensitization is elevated among offspring of sensitized parents.

Objective—To evaluate whether 18 year old offspring are likely to have the same allergic sensitizations as their parents.

Methods—18 year old participants in an unselected birth cohort and their parents were tested for total and elevated (≥ 0.35 kU/L) levels of allergen-specific IgE to 6 allergens: *Der f*, dog, cat, grass, ragweed, and *Alternaria*.

Results—In 316 parent-teenager triads, parental sensitization to any of six allergens was associated with teen sensitization to any of those same allergens. An increased risk of matched sensitization (i.e., a teen has an increased risk of being sensitized to the same specific allergen as their parent) was found after adjusting for the spouse's sensitivities and adjusting for other allergens (i.e., the parent had an allergic sensitization but not to the particular allergen under analysis). Risk of maternal matched sensitization with their teen to cat (aOR 2.1, 95% CI 1.0,4.5), grass (aOR 2.5, 95% CI 1.2, 5.2) and *Alternaria* (aOR 2.4, 95% CI 1.1,5.5) was elevated when compared to teens without parental allergen specific sensitization. Similarly, a higher than expected risk of paternal matched sensitization with their teen to dog (aOR 2.7, 95% CI 1.3, 5.9), *Der f* (aOR 2.7, 95% CI 1.4, 5.1) and grass (aOR 2.7, 95% CI 1.5, 5.9) was observed.

Conclusion—Parental allergen-specific IgE increases the likelihood of sensitization to the same allergen in young adult offspring.

Clinical Implications—Older teens are more likely to have sensitization to the same particular allergen as either their mother or father.

Capsule summary—The specific allergic sensitizations of a parent are associated with their teenager's allergic sensitivity.

Keywords

allergen specific IgE

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Reprint requests: Edward M. Zoratti, Henry Ford Health System, One Ford Place 4B, Detroit MI 48202, Phone (313) 876-2662, Fax (313) 876-2094. ezoratt1@hfhs.org. .

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INTRODUCTION

It is well accepted that a family history of atopy is a risk factor for atopy in children.^{1,2} However, with regard to allergic rhinitis or asthma it is unclear whether children will have the same airborne allergen sensitizations as their parents (i.e., a dust mite sensitized parent has a dust mite sensitized child – “matched sensitization”), or simply a greater overall tendency towards sensitization to common allergens (“general sensitization”). In clinical practice, parents often inquire about whether their children will have the same allergies that they do, especially when making decisions about a family pet or another aspect of their home environment. Few studies have been done to evaluate matched allergen specific sensitivities within families.^{3,4}

In addition, parental gender is thought to play a role in the inheritance of atopic illnesses with mothers traditionally thought to play a greater role.^{2,4-7} However, some studies have suggested a significant relationship between persistent allergy in fathers and their children.⁸

The aim of this study was to evaluate the relationship between specific allergen sensitization among triads of mothers, fathers, and their children entering adulthood. Specifically, we examined whether general parental sensitization was associated with general sensitization in 18 year old offspring, along with the more focused investigation of whether a teenage child is at increased risk of developing allergen sensitivities matched to their parents. We also examined the role of parental and child sex in the patterns of allergic sensitization of their teenage children.

METHODS

Study population

The Childhood Allergy Study (CAS) was conducted among members of Health Alliance Plan, a health maintenance organization (HMO), in the suburbs of Detroit, Michigan. CAS is a study to evaluate the environmental determinants of pediatric allergy and asthma. All pregnant Henry Ford Medical Group HAP members that were at least 18 years of age, living in a predefined geographic area, and had an estimated date of confinement between April 15, 1987 and August 31, 1989, were invited to participate in the study. Only full term infants (at least 36 weeks gestation) were included in the study. Therefore, this represents an “unselected birth cohort” other than recruitment based on the common characteristics of membership in a particular HMO, delivery date, and geographic location of the clinic.

Of the 1194 pregnant women who were eligible, 953 women consented to participate. Infants from 106 of these women were not further enrolled in the study because their cord blood was not obtained for analysis. Of the remaining 847 infants, six had cord blood that was believed to be contaminated by maternal blood and six more were determined to be ineligible at subsequent review of eligibility criteria. Mothers of the remaining 835 children were asked to complete interviews annually to discuss the health of the child for the previous year. Annual interviews were conducted through age 6-7 years when children were invited to complete a clinic visit. In May 2005, the oldest children enrolled in CAS reached 18 years of age. In the current study, we contacted all children after their 18th birthday and their parents to obtain information on allergy, asthma, and other health and exposure histories through interviews covering ages 6 through 18 years. After completing the interview, teens and parents were invited to provide a blood sample for measurement of total and specific IgE. There were no exclusions for the blood draw, such as concurrent medication use or viral illness, and samples were collected throughout the year at the convenience of participants and their parents. Teens and parents did not necessarily have their blood drawn on the same day.

We were able to collect samples for both parents and the teen forming 316 teen-mother-father “triads” that serve as the study population for the current analyses. All aspects of this research were approved by the institutional human subjects boards of review of all participating institutions.

Assessment of allergen-specific IgE

Venous blood was collected for assessment of allergen-specific IgE, and stored at -80°C until assayed. Measurements of allergen-specific IgE were performed following the standard manufacturer’s protocols using the Pharmacia UniCAP system (Pharmacia-Upjohn Diagnostic Division, Kalamazoo, MI). Allergen-specific IgE was analyzed for *Dermatophagoides farinae* (*Der f*), dog, cat, grass, ragweed, and *Alternaria alternata* (*Alternaria*). One percent of all assays were repeated in a different assay run on a different day to provide estimates of interassay reliability. The geometric mean coefficient of interassay variation was 5.9% for all 6 allergens. Sensitization was defined as a positive allergen-specific IgE result of ≥ 0.35 kU/L.

Statistical analysis

A Student’s t-test was used to compare mean age in included versus excluded groups. To further compare these groups, chi-square tests were used for categorical variables such as gender and marital status. Differences in total IgE were analyzed using a Wilcoxon rank sum test due to the non-normality of the data.

Odds ratios (ORs) and accompanying 95% confidence intervals (CIs) were calculated to examine associations between parent and teen sensitization. We defined general sensitization to reflect an individual that is sensitized (any allergen specific IgE ≥ 0.35 kU/L) to at least one of the six allergens. A logistic regression model was then built to look at the independent associations of both maternal and paternal general sensitization with teen general sensitization at age 18.⁹ Wald’s test for linear hypotheses was used to directly compare the regression coefficient for maternal sensitization with the regression coefficient for paternal sensitization.

Next, allergen-specific sensitization was defined as sensitivity to each specific allergen (≥ 0.35 kU/L). For each individual, allergen-specific sensitization was defined without regard to that same individual’s sensitization to any of the other five remaining allergens. Therefore, if an individual was determined to have cat sensitization they also, by definition, have general sensitization, however this does not mean that they were necessarily sensitized to any of the five other allergens (dog, ragweed, grass, *Der f*, or *Alternaria*). When the teen was sensitized to the same allergen as their parent, this was defined as matched sensitization.

To address whether parental sensitization to a specific allergen affects their teen’s risk of being sensitized to that same specific allergen we first looked at unadjusted measures of association (ORs with 95% CIs) for each of the six allergens separately. Next, to account for sensitization to the five other allergens in an adjusted model without including ten additional variables (one for each of the five allergens, separately for mother and father), we defined “other” sensitization as a parent being sensitized to at least one of the five other allergens, excluding the specific allergen under analysis. Separate logistic regression models were then built for each of the six allergens to predict allergen-specific teen sensitization using four dichotomous dependent variables: (1) maternal matched sensitization (yes/no); (2) paternal matched sensitization (yes/no); (3) maternal other sensitization (yes/no); and (4) paternal other sensitization (yes/no). Using these models, adjusted ORs and 95% CIs were calculated for each specific allergen. We used stratified analyses to consider gender differences. Statistical significance was defined as $p \leq 0.05$.

We were not able to fully investigate whether genetics or environment is a greater predictor of associated sensitization between parent and teen. However, the data provide a unique opportunity to look within households where parents lived together for all of the teen's life (90% of this sample). To describe the possible role of shared environments for the familial triads, we used the kappa statistic to assess agreement in sensitization between mother and father versus between parent and child. The kappa statistic corrects for chance agreement and tells us how much of the agreement over and above chance has been achieved.¹⁰ Agreement in sensitization between mother and father for any of the allergens could suggest a strong environmental influence on sensitization. These kappa calculations were restricted to 90% (n=284) of the sample where mother, father and teen lived together up to the date of inclusion in the study (at least 18 years). To assess agreement between mother's sensitization and father's sensitization, kappa statistics were calculated for general sensitization along with, separately, sensitization for each of the six specific allergens. We tested whether kappa was different from zero (zero meaning no agreement beyond that expected by chance) and used an alpha level of 0.05 for each test. For comparison purposes we also calculated the kappa statistics for mother and teen sensitization agreement and for father and teen sensitization agreement. A kappa value of 0 represents no degree of agreement beyond chance, 0-0.2 a slight degree of agreement beyond chance, 0.2-0.4 a fair degree of agreement beyond chance, 0.4-0.6 moderate degree of agreement beyond chance, 0.6-0.8 a substantial degree of agreement beyond chance, and a kappa value of 0.8-1 representing an almost perfect degree of agreement beyond chance.¹⁰

RESULTS

Three hundred sixteen teen-parent triads had complete laboratory results. As compared to the remaining 519 (out of 835) that were originally recruited in the 1988-89 study, the included parents were more likely to be married (98% vs. 91%, respectively, $p<0.05$) and more likely to have attended some college (mothers: 73% vs. 55%, $p<0.05$; fathers: 81% vs. 61%, $p<0.05$). The racial composition for both mother and father was similar between included and excluded (mothers: Caucasian 95% vs 91%, $p=0.08$; fathers: Caucasian 94% vs. 90%, $p=0.05$). An additional 354 teens were evaluated at age 18 years, however we were not able to obtain blood samples from both of their parents and therefore they could not be included in this analysis. The 354 teens excluded for incomplete triad samples were similar to the 316 included teens except that the included teens more likely to have married parents who had attended some college (data not shown).

Sample description

Demographic characteristics of the 316 triads are provided in Table 1. The majority of mother-father-teen triads lived together in the same house (n=284, 90%) in a suburban environment. Most parents were Caucasian, had at least some college education, and were married at the time of delivery. Current mean age was 48.0 years for mothers, 50.0 years for fathers, and 18.3 years for teens. Almost half the fathers were sensitized to at least one of the six allergens tested (general sensitization) (n=155, 49.1%), and the mothers were slightly less likely to have general sensitization (n=128, 40.5%), $p<0.05$. Over half of the children had general sensitization (56.0% of females, 60.0% of males). The fathers' total IgE was higher, on average, than the mothers, $p<0.05$. Specific rates for sensitization to each tested allergen are presented in Table 1.

Parent – Teen General Association of Allergen Sensitization

Table 2 (top) shows the results of multiple logistic regression models (1 model for all teens, 1 for only female teens, 1 for only male teens) examining the association between parent general sensitization and teen general sensitization. In all teens, both father and mother were

independently associated with the teens' sensitization status. Total IgE and specific IgE levels to each of the 6 aeroallergens in the teenage participants were correlated to those levels of their mother or father. (Table 1 Online Repository).

The data suggest sensitization is heritable and the degree of heritability may be influenced by the gender of child and parent. **However**, using a Wald's test, we directly compared the regression coefficients for father sensitization with mother's sensitization in the all teen model and no statistically significant difference was found, $p=0.060$. The suggestion of difference was stronger within males ($p=0.08$) as compared to females ($p=0.34$). The odds of teen general sensitization is greatest among sons of men with general sensitization compared to sons without a father with general sensitization (aOR=5.5, 95% CI=2.6, 11.5) even after adjusting for maternal sensitization status. To a lesser degree, maternal general sensitization was also associated with greater odds of general sensitization in their sons compared with male teens whose mothers did not have general sensitization, even after adjusting for the father's sensitization status (aOR=2.3, 95% CI=1.0, 4.9). However among female teens, the association with sensitized mothers was no longer statistically significant (aOR=1.6, 95% CI=0.8, 3.0), while the association with sensitized fathers remained significant (aOR=2.4, 95% CI=1.3-4.6). When this analysis was restricted to triads that resided in the same household for 18 years (see Online Repository) similar trends were seen, although the association between sensitized mothers and female teens was now statistically significant.

Maternal – Paternal versus Parent – Child Associations

In an attempt to describe the possible role of shared environments for the familial triads, we used the kappa statistic to assess agreement in general and matched allergen-specific sensitization between parents versus parent and child. The kappa statistic corrects for chance agreement and describes how much of the agreement over and above chance has been achieved. None of the kappa coefficients measuring the level of agreement between maternal and paternal sensitization to the same specific allergen reached statistical significance (Table 3). In contrast, numerous allergens had statistically significant kappa coefficients between parents and teens, showing slight (kappa 0-0.2) to fair (kappa 0.2-0.4) agreement depending on the allergen. Further, there was no association between mothers' general sensitization with fathers' general sensitization, $p=0.83$ (OR=0.9, 95% CI 0.6, 1.5).

Association of Parent – Teen Matched Allergen – Specific Sensitization

Table 4 includes the logistic regression models examining the specific sensitization relationships within triads. Each row includes a single model for each specific allergen (1 multivariable model with 4 covariates). This model was chosen so that both parent gender and type of sensitization (matched for that allergen or sensitized to something other than that allergen) could be considered jointly and simultaneously in the model. Due to the large number of point estimates presented, these data should be considered descriptive rather than conclusive evidence.

After adjusting for maternal sensitization to the other five allergens as well as all paternal sensitizations, teens whose mothers were sensitized to cat were more likely to be sensitized to cat (aOR=2.1, 95% CI=1.0, 4.5) compared with teens whose mothers were not sensitized to cat. This was also true for grass (aOR 2.5, 95% CI=1.2, 5.2) and *Alternaria* (aOR=2.4, 95% CI=1.1, 5.5). After adjusting for paternal sensitizations to the other five allergens as well as all maternal sensitizations, teens whose fathers were sensitized to dog were more likely to be sensitized to dog (aOR=2.7, 95% CI=1.3, 5.9) compared with teens whose fathers were not sensitized to dog. This was also true for *Der f* (aOR 2.7, 95% CI=1.4, 5.1) and grass (aOR 2.7, 95% CI=1.5, 4.9).

We further used a Wald's test to examine if the regression coefficient for parental matched sensitization differed, statistically, from the regression coefficient for parental other sensitization, within each model. All comparisons were not statistically significant at the 0.05 level with two exceptions. In the all teen model predicting teen sensitization to *Der f*, paternal matched sensitization was significantly greater than paternal other sensitization at $p=0.037$. In the female teens only model predicting teen sensitization to *Alternaria*, maternal matched sensitization was significantly greater than maternal other sensitization at $p=0.034$.

It is unclear whether teen gender modifies patterns of matched sensitization. Although elevated odds ratios were apparent for the majority of allergens, the following reached statistical significance. Female teens were more likely to be sensitized to *Alternaria* if their mother was also sensitized to it (aOR=3.8, 95% CI=1.1, 13.0). Female teens were more likely to be sensitized to the following allergens if their father was also sensitized to the same allergen: *Der f* (aOR=2.6, 95% CI=1.1, 6.1), and grass (aOR=3.2, 95% CI=1.3, 7.6). Male teens were more likely to be sensitized to dog (aOR=5.3, 95% CI=1.8, 16), *Der f* (aOR 2.9, 95% CI=1.1, 7.5) and grass (aOR=3.0, 95% CI=1.2, 7.5) if their fathers were sensitized to the same allergen.

When this analysis was repeated after excluding the 32 families (10%) that did not live together for all 18 years, the results were not significantly different. Similarly, excluding households with pets or tobacco smoke exposure in the first year of life did not substantially modify the results (data not shown).

DISCUSSION

We set out to examine if a parent's specific allergen sensitization was associated with an increased risk of the same allergen sensitization in their teenage offspring. Our data suggest that the inheritance of sensitization has an allergen specific component as well as risk associated with general sensitization of a parent. The increased risk of matched sensitization was not statistically elevated for each allergen. However, maternal sensitization to cat, grass, and *Alternaria* significantly elevated the risk of matched sensitization in their children. Similarly, an increased risk of paternal matched sensitization with their teen was observed for dog, *Der f*, and grass. These associations were observed after adjusting for the spouse's sensitization and after adjusting for other sensitization (i.e., the parent had an allergic sensitization but not to the particular allergen under analysis). These findings, although they should be considered to be descriptive, suggest that specific sensitization may be inherited. The implication of this finding, should it be supported in other large studies, is that when counseling sensitized parents about the possibility of matched sensitization in their children, one could suggest that the child may have similar specific allergen sensitizations, which may have an impact on choices a family makes in the home environment. Since our study participants were in an unselected birth cohort, these findings may be generalizable to other populations.

We feel that the age of 18 years may be an informative time point for study, since in many cases this is the last age that children are residing in the same house with their parents before leaving for other endeavors. One of the strengths of this study is that the 18 year old participant had lived his/her entire life in the same environment as the parents, and thus presumably had similar exposures to them. Although our analysis was not intended to separate the factors of environment versus genetic contributions, we did explore whether a parentally shared environment for at least 18 years could account for matched sensitization among the subjects that were unrelated. If there was a strong environmental influence, it is possible that the mother and father would have matched allergen specific sensitizations at rates greater than what would be expected by chance alone. This was not observed.

However, since the development of allergen sensitization can depend on the age that allergen exposure occurs, it is possible that the lack of a shared environment for the parents until adulthood was the reason we did not observe an effect of the shared environment.

Comparatively, previously published studies have shown both conflicting and similar results. Kelso et al. performed skin prick testing of 12 inhalant allergens among 13 parent-child pairs, 19 sibling-sibling pairs and 32 unrelated pairs. They concluded that first degree relatives with allergic rhinitis were no more likely to be skin test positive to the same allergen extracts than unrelated persons.³ By contrast, we investigated a larger, unselected birth cohort, and were able to look at both mother and father in comparison to their teenager and these characteristics likely contribute to our different conclusions. A recent analysis from Canfield et. al measured specific IgE to four indoor allergens in 161 predominantly Hispanic triads (parents and their 4 year old child) living in New York City neighborhoods with high pediatric asthma hospitalization rates.⁴ They found an association between maternal (but not paternal) specific IgE and IgE in 4 year old offspring (regardless of gender). Our study population differed from theirs in a number of ways. Our population is unselected in regard to family history or ethnicity that would import high risk for allergic disease. Furthermore, our participants are 18 years old and therefore have had more time to develop allergic sensitization. In fact, our teens had higher sensitization rates than their parents. We also assessed more and different allergens, including seasonal outdoor allergens. Finally, our analysis considered the role of other sensitizations in the analyses of risk for each specific allergen.

It is difficult to generate a unified hypothesis to describe the relative contribution of genetic and exposure-related factors that lead to an elevated risk of matched sensitization for one allergen versus another. However, several studies have found that particular allergen sensitivities are associated with certain HLA loci, including ragweed allergen Ra5 and an allele at the HLA-DR loci,¹¹ HLA-DRB1*07 and sensitization to *Der p1* and *Der p 2*,¹² rat allergen Rat n 1 and HLA-DR7,¹³ and cockroach sensitization and HLA-DRB1*0101 (in the Hutterite community in South Dakota) versus HLA-DRB1*0102 (in African-American participants living in Chicago).¹⁴ The complexity of the genetic factors in regard to allergen sensitization was demonstrated by Tovey who found that even though monozygotic twins were more likely than dizygotic twins to be skin prick test positive to house dust mite (HDM), in monozygotic twins, there were substantial differences in the specific HDM allergens that they responded to.¹⁵ Similar findings were seen in regard to *Alternaria*.¹⁶ These examples illustrate the complexity of the genetics of allergic sensitization. While our study did not include HLA typing, future analysis including HLA could further clarify the relationship between genetic heritability and environmental exposure in triggering allergen sensitization.

The role of gender was one we analyzed as well, since a mother's atopic status is typically thought to be of greater significance than a father's. Our findings suggest that with regard to allergic sensitization at age 18, fathers play an equally important role as mothers. Interestingly, except for grass, mothers and fathers had different allergen-specific matched sensitizations with their teens (both indoor and outdoor allergens). It remains to be seen whether this is due to gender-specific differences in levels of exposure or other factors. Sex-specific estimates in the teens were less precise due to reduced sample size, however we did observe different matched sensitizations in female and male teens, which warrants further evaluation. Furthermore, different patterns of matched sensitization were seen with mothers or fathers and their daughters or sons. The theory of maternal imprinting and chromosome 11q put forth by Cookson et. al¹⁷ would help explain the maternal, but not paternal influence. Hunninghake et. al showed that a particular allele of thymic stromal lymphopoietin was associated with decreased IgE to cockroach, but only in female children.

18 Therefore, biologic activity of immunoregulatory proteins may differ by sex and be a potential explanation of different inheritance patterns of allergic sensitization among males and females.

Our population was not diverse with regard to race or geographic location which limits the generalizability of these results to other ethnic groups or geographic locations including inner city or rural environments. In addition, the included teens were more likely to have married parents who had attended some college. It is possible that completion of some college or being married may make a family more likely to participate in a study, which may limit the generalizability of our results. Also, we measured IgE for only six inhalant allergens, so it is possible that other patterns may have been observed had a larger number of allergens been studied. However, these are the most common allergens in southeastern Michigan and rates of sensitization were high in this general-risk cohort. In fact, our rates of sensitization in teens were higher than those described in the recent analysis of the US population in the Third National Health and Nutrition and Examination Survey (NHANES) where ten allergens were evaluated.¹⁹ Furthermore, our analysis considered only specific IgE levels and not coexisting allergic conditions (e.g. asthma, eczema) or whether particular allergic sensitizations were associated with clinical symptoms. We acknowledge that a positive IgE test does not necessarily produce clinical symptoms.²⁰ Finally, our analysis was cross-sectional. A longitudinal analysis of the within-person change in specific allergic sensitizations over time would be interesting for further study.

In sum, our findings show high rates of matched specific allergen sensitization between both mothers and their 18 years old teens and fathers and their 18 year old teens. A child's development of specific allergen sensitization is likely to reflect both exposure to common allergens and a degree of additional risk determined by their parent's specific sensitizations. Further studies that are focused on identifying the specific biologic mechanisms that are operant in the inheritance of allergen sensitization will be important to develop strategies to minimize the risk of clinical allergic disease among children that have atopic parents.

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Abbreviations

OR	odds ratio
aOR	adjusted odds ratio

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Table 1

Cohort demographics and elevated IgE frequencies.

	Teen (N=316)			
	Mother N=316	Father N=316	Female N=166	Male N=150
Caucasian, n (%)	299 (94.6%)	297 (94.0%)	159 (95.8%)	138 (92.0%)
Attended some college [*] , n (%)	230 (72.8%)	255 (80.7%)	----	----
Mean age [†] (s.d.)	48.0 (3.9)	50.0 (4.5)	18.4 (0.5)	18.3 (0.4)
Married at time of pre-delivery [*] , n (%)	309 (97.8%)	309 (97.8%)	----	----
General sensitization [‡] , n (%)	128 (40.5%)	155 (49.1%) ²	93 (56.0%)	90 (60.0%)
Specific sensitization to:				
<i>Der f</i> , n (%)	51 (16.1%)	55 (17.4%)	45 (27.1%)	45 (30.0%)
Dog, n (%)	39 (12.3%)	43 (13.6%)	27 (16.3%)	33 (22.0%)
Cat, n (%)	49 (15.5%)	51 (16.1%)	30 (18.1%)	39 (26.0%)
Grass, n (%)	46 (14.6%)	88 (27.8%) ²	39 (23.5%)	49 (32.7%)
Ragweed, n(%)	58 (18.4%)	74 (23.4%)	47 (28.3%)	55 (36.7%)
<i>Alternaria</i> , n(%)	31 (9.8%)	45 (14.2%)	29 (17.5%)	47 (31.3%) ³
Total IgE (kU/L)	23.0	35.7	40.2	46.6
Geometric mean (95% CI)	(19.9, 26.7)	(30.5, 41.7) ⁴	(31.8, 50.8)	(36.4, 59.7)

^{*} reported in CAS (maternal prenatal interview);

[†] Age as of clinic visit for CAS2 study;

[‡] Defined as specific IgE ≥ 0.35 kU/L. for at least 1 of six allergens;

² Chi square test p<0.05, for comparison of Mother with Father;

³ Chi square test p<0.05, for comparison of male teen vs female teen;

⁴ Wilcoxon rank sum test p<0.05 for comparison of Mother with Father

Table 2

Likelihood of allergic sensitization to any of 6 allergens among children of sensitized parents.

All Families (n=316)	Predictor variables	
Outcome Variable: General sensitization ¹ of Teen	Sensitized ¹ Mother ²	Sensitized ¹ Father ³
	aOR ⁴ (95% CI)	aOR (95% CI)
All Teens (n=316)	1.8 (1.1, 3.0)	3.4 (2.1, 5.5)
Female Teens (n=166)	1.6 (0.8, 3.0)	2.4 (1.3, 4.6)
Male Teens (n=150)	2.3 (1.0, 4.9)	5.5 (2.6, 11.5)

¹ Defined as specific IgE ≥ 0.35 kU/L. for at least 1 of six allergens; each row represents one model;

² reference group is mothers with no sensitization to any of the six allergens;

³ reference group is fathers with no sensitization to any of the six allergens.

Kappa coefficients for families who remained in same household for entire 18 years of teen's life (n=284).

Table 3

	General sensitization ¹	Allergen specific sensitization Allergen:					
		Dog	Cat	Ragweed	Der.f	Grass	Alternaria
Mother – Teen	0.16	0.08	0.19	0.12	0.10	0.14	0.13
Father – Teen	0.31	0.20	0.11	0.15	0.20	0.19	0.05
Mother – Father	–0.01	–0.10	0.04	–0.10	–0.07	–0.08	0.03

¹ Defined as specific IgE≥0.35 kU/L. for at least 1 of six allergens;

² All bolded Kappa coefficients, p<0.05 .

Table 4

Adjusted odds of Teens' sensitization to a specific allergen given parental sensitization (n=316)

Teen sensitized	Multivariable models ³			
	Matched sensitization ¹		Other sensitization ²	
	Mother ⁴ aOR (95% CI)	Father ⁵ aOR (95% CI)	Mother ⁶ aOR (95% CI)	Father ⁷ aOR (95% CI)
All teens (N=316)				
Dog	1.4 (0.6, 3.3)	2.7 (1.3, 5.9)	2.0 (1.1, 3.7)	1.9 (1.0, 3.7)
Cat	2.1 (1.0, 4.5)	1.6 (0.8, 3.3)	1.5 (0.8, 2.8)	1.7 (0.9, 3.2)
Ragweed	1.7 (0.9, 3.4)	1.3 (0.7, 2.4)	1.7 (1.0, 2.9)	2.0 (1.2, 3.5)
<i>Der f</i>	1.8 (0.9, 3.4)	2.7 (1.4, 5.1)	1.4 (0.8, 2.4)	1.0 (0.6, 1.7)
Grass	2.5 (1.2, 5.2)	2.7 (1.5, 4.9)	1.4 (0.8, 2.5)	1.2 (0.7, 2.1)
<i>Alternaria</i>	2.4 (1.1, 5.5)	1.0 (0.5, 2.1)	1.4 (0.8, 2.4)	2.0 (1.1, 3.4)
Female teens only (N=166)				
Dog	1.5 (0.5, 4.7)	1.3 (0.4, 4.3)	2.0 (0.8, 5.0)	1.8 (0.7, 4.5)
Cat	2.4 (0.9, 6.5)	1.1 (0.3, 3.8)	1.7 (0.7, 4.1)	1.4 (0.6, 3.3)
Ragweed	2.0 (0.8, 5.3)	2.3 (1.0, 5.4)	1.5 (0.7, 3.3)	1.6 (0.7, 3.6)
<i>Der f</i>	1.7 (0.7, 4.3)	2.6 (1.1, 6.1)	1.4 (0.7, 3.0)	1.0 (0.5, 2.0)
Grass	2.4 (0.8, 7.4)	3.2 (1.3, 7.6)	1.7 (0.7, 4.0)	0.9 (0.4, 2.1)
<i>Alternaria</i>	3.8 (1.1, 13.0)	1.5 (0.5, 4.5)	0.6 (0.2, 1.4)	1.8 (0.8, 4.3)
Male teens only (N=150)				
Dog	1.2 (0.3, 4.9)	5.3 (1.8, 16.0)	2.2 (0.9, 5.4)	2.2 (0.9, 5.6)
Cat	2.1 (0.7, 6.6)	1.7 (0.6, 4.4)	1.4 (0.6, 3.4)	2.5 (1.1, 5.7)
Ragweed	1.5 (0.6, 3.9)	0.7 (0.3, 1.8)	1.9 (0.9, 4.1)	2.7 (1.2, 5.9)
<i>Der f</i>	1.9 (0.7, 4.9)	2.9 (1.1, 7.5)	1.5 (0.7, 3.2)	1.0 (0.5, 2.2)
Grass	2.3 (0.9, 6.3)	3.0 (1.2, 7.5)	1.5 (0.7, 3.3)	1.3 (0.6, 2.9)
<i>Alternaria</i>	1.6 (0.5, 5.4)	0.6 (0.2, 1.8)	2.6 (1.2, 5.5)	2.5 (1.1, 5.4)

¹ indicates parent is sensitized to same allergen as teen, allergen named in left most column² indicates parent is sensitized to at least one of the other five allergens, not the allergen named in left most column;³ each multivariable model includes the following four dichotomous variables: (1) maternal matched sensitization (yes/no); (2) paternal matched sensitization (yes/no); (3) maternal other sensitization (yes/no); and (4) paternal other sensitization (yes/no)⁴ reference group is mother not sensitized to same allergen as teen⁵ reference group is father not sensitized to same allergen as teen⁶ reference group is mother not sensitized to any of five allergens, excluding the allergen named in the left most column⁷ reference group is mother not sensitized to any of five allergens, excluding the allergen named in the left most column