Original article

Total and specific IgE associations between New York City Head Start children and their parents

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Background: Allergy and asthma risk share strong inherited components; however, the relative importance of maternal and paternal atopy in predicting child atopy remain unclear.
Objective: We sought to identify relationships between parents' and children's total and specific IgE levels within family units as predictors of allergic risk in children.
Methods: Total and allergen-specific IgE (to dust mite, cockroach, mouse, and cat) were determined by means of ImmunoCap (Phadia, Inc, Portage, Mich) in a sample of families participating in New York City Head Start programs. Regression models were developed to determine the associations of parents' and children's total IgE levels and sensitization patterns.
Results: Blood specimens were collected from 161 family triads of mother, father, and child (83 boys and 78 girls). At a mean age of 4 years, boys had significantly higher total IgE levels than girls. Boys' total IgE levels were highly correlated with both mothers' ($P < .002$) and fathers' ($P = .002$) total IgE levels; girls' total IgE levels were not. Unlike total IgE levels, specific IgE levels among both boys and girls were associated with their mothers' specific IgE levels. Dust mite sensitization among mothers was predictive of children's sensitization to each of the 4 aeroallergens.
Conclusion: The strong associations between parents' and children's IgE levels suggest that assessment of parents' total and locally relevant allergen-specific IgE levels might have value in predicting atopy in children of preschool age. (J Allergy Clin Immunol ■■■■;■■■:■■■-■■■.)

Key words: *Allergy, asthma, familial, total IgE, specific IgE, Hispanic, Head Start, children*

Abbreviations used
ETS: Environmental tobacco smoke
M-F-C triad: Mother-father-child triad

The development of asthma in children is strongly associated with atopy.[1-3] The determinants of a child's risk of sensitization to airborne allergens remain incompletely characterized. Clinically, family history of asthma or allergic disease is one of the features most commonly used to estimate a child's atopic risk. Although evidence supports the predictive value of this indicator (in particular of maternal history),[4-6] its reliability remains disputed.[7] A small number of studies have demonstrated the predictive value of a parent's serum IgE level for children's atopy. Among 468 children at a mean age of 7 years in Tucson, Arizona, Burrows et al[8] found a highly significant association between the children's total IgE levels and those of both mothers and fathers. More recently, in a study of 545 infants in Taiwan, maternal, but not paternal, total IgE levels were correlated with infant total IgE levels at 6 months of age.[9]

The implications of parental sensitization to specific allergens with respect to children's atopy remain largely unexplored. Multiple studies have established an association between parental allergic history and a child's sensitization by means of skin prick testing[6,10,11] and, conversely, between parental sensitization and a child's symptoms.[12] However, very few have documented a direct relationship between parents' and children's sensitizations. The comparison of allergen-specific sensitization between a child and parent has been facilitated by the development and validation[13] in the last decade of the Pharmacia ImmunoCAP method of IgE quantitation. In a Belgian study of 600 families using this method, specific IgE response to any of 7 common inhalant allergens among fathers was found to predict allergic sensitization (specific IgE to any of 3 inhalant or 2 food allergens) among their 1-year-old infants, although the adjusted odds ratio was statistically significant only for the girls.[14]

Generation of specific IgE to a given allergen depends not only on local exposure levels but also on inherited factors, including HLA genetics.[15-18] Certain allergen-specific sensitizations are particularly associated with respiratory disease,[19,20] but the clinically relevant allergens vary across geographic regions.[21] To date, no study has examined whether sensitization to particular allergens in parents can predict sensitization to the same allergens in their children. Moreover, although the sum of specific IgE levels to highly relevant indoor inhalant allergens has recently been shown to predict wheeze and compromise in lung function,[22] no study has examined the relationship between parents' and children's specific IgE concentrations. We have addressed these questions in a sample of children at a mean age of 4 years

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Supported by HL068236 and the National Institutes of Environmental Health Sciences P30 ES009089 and by a grant from the Flight Attendants' Medical Research Institute.
Disclosure of potential conflict of interest: S. M. Canfield has received research support from the Flight Attendants' Medical Research Institute and the National Institutes of Health. J. S. Jacobson has received research support from the National Cancer Institute, the Lance Armstrong Foundation, and MetroPlus. M. S. Perzanowski has received research support from the National Institutes of health. R. B. Mellins has received research support from the National Institutes of Health and the Children's Health Foundation, Inc, Norfolk, Va. G. L. Chew has received research support from the National Institutes of Health. I. F. Goldstein has received research support from the National Institutes of Health. R. M. Zemble has declared that he has no conflict of interest.
Received for publication March 17, 2007; revised February 4, 2008; accepted for publication March 3, 2008.

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0091-6749/$34.00
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doi:10.1016/j.jaci.2008.03.011

living in New York City neighborhoods with high pediatric asthma hospitalization rates.[23] The children in this study are predominantly of Hispanic background. They therefore represent a rapidly growing subgroup of the US population[24] and one for which studies of predictors of allergy remain scarce.[25]

METHODS

Subjects and recruitment

The study population has previously been described.[23] Briefly, participants were recruited from 50 Head Start centers in northern Manhattan, the Bronx, and Brooklyn. Informed consent was obtained from parents who responded to a questionnaire about demographic and allergy symptom–related factors. On the basis of the survey, subjects were grouped by national origin as follows: Mexican, Dominican, Puerto Rican, other/mixed Hispanic, black, white, and other non-Hispanic (detailed demographic and clinical characteristics have been reported[23]). A total of 517 families who agreed to have the study team collect data in their homes received visits. During the first home visit, phlebotomy was performed on each child and on available parents. This study focuses on families in which sera were obtained from the child and both parents. The study was approved by the Health Sciences Institutional Review Board of Columbia University.

IgE analysis

All blood specimens were received in the laboratory within 2 hours of collection. All processed serum specimens were stored frozen at −80°C. Serum total IgE and allergen-specific IgE determinations were made with a UniCap100 automated IgE analyzer (Phadia, Inc, Portage, Mich). For the purposes of analysis, tests yielding an undetectable IgE level were assigned an arbitrary value midway between zero and the detection limit (1 IU/mL for total IgE and 0.17 IU/mL for specific IgE).

Serum was assayed for total IgE and specific IgE to 4 inhalant allergens strongly associated with asthma in urban minority populations[21]: house dust mite *(Dermatophagoides farinae)*, German cockroach, cat dander, and mouse urinary protein. Sensitization was defined as a specific IgE level of 0.35 kU/L or greater. Preliminary analyses of specific IgE to 2 other inhalants, dog dander and *Alternaria alternata*, in the initial 200 subjects determined that fewer than 1% of subjects were sensitized to either allergen (data not shown). Every *Alternaria* species– or dog-sensitized individual also showed additional sensitizations (commonly to dust mite or cockroach). Hence we did not include these allergens in the remainder of the IgE analysis.

Statistical analysis

The analyses reported here were performed on study participants for whom complete triads of mother, father, and child serum were available (M-F-C triads). Natural log transformation was used to normalize the distribution of total IgE levels. We used *t* tests for pairwise comparisons. Boys and girls were compared with respect to the proportion sensitized to specific allergens by using χ^2 tests of statistical significance. We developed linear regression models of the association of children's and parents' log-transformed total IgE levels. Correlations between children's and parents' summed specific IgE levels were examined with the Spearman rank sum test. To explore possible parent-child associations in specific allergen sensitization or seroatopy (defined as the presence of any specific IgE of $\geq$0.35 kU/L), the children were divided into 4 groups; those without a sensitized parent served as the referent population for the other 3 groups: children whose mothers (but not fathers) were sensitized, those whose fathers (but not mothers) were sensitized, and those whose fathers and mothers were sensitized. In all regression analyses the following variables were evaluated as potential effect modifiers by means of stratification of the triad sample: age, sex, environmental tobacco smoke (ETS) exposure, and birth order. In addition, the following variables were evaluated as potential confounders by observing the effect of inclusion of each, individually, on the regression results: national origin, mother's educational attainment, ETS exposure, and environmental allergen exposure. Allergen levels, including cockroach, mouse, and cat dander, were evaluated in dust collected from the subject's bed and kitchen by using standard methods.[26] Childhood pet exposure was not included as a potential confounder because fewer than 5% of triad families reported pet (cat, dog, rodent, or other furry animal) exposure during the child's life.

RESULTS

Demographic characteristics and sensitization rates among Head Start families

Among the 161 M-F-C triads, the mean age of the children was 4.0 years (Table I). The sample included 83 boys and 78 girls. The majority (>80%) of participating families were of Hispanic origin (Mexican, Dominican, Puerto Rican, or other/mixed Hispanic); about 10% were non-Hispanic black.

After log transformation, total IgE values for children, mothers, and fathers were normally distributed. Geometric mean total IgE levels were higher among male participants (fathers and boys) than among female participants (mothers and girls), and within each sex, the adults had higher geometric means than the children (Table I). Total IgE values were not associated with national origin (data not shown).

Parents were more likely than children to be sensitized to dust mite, cockroach, and cat, but sensitization to mouse was most prevalent among boys. Consistent with previous studies of children at this age,[27] the prevalence of sensitization to each allergen and to any allergen was higher among boys than among girls (Table I and see Fig E1 in the Online Repository at www.jacionline.org).

Correlation of children's with parents' IgE levels

Linear regression analysis demonstrated a correlation between the log-transformed children's total IgE levels and those of both parents (Table II). Stratification to identify potential effect modifiers of this relationship revealed that the parent-child correlation was statistically significant for children aged 48 months or older but not for younger children and for boys but not for girls. The β values for the association between the child and either parent did not differ between the 38 ETS-exposed and the 123 unexposed children, although the correlation was significant only for the latter. Finally, there were no trends detected in the strength of the parent-child total IgE correlation with change in birth order.

The correlations of parents' and children's total IgE levels were slightly stronger in regression models that included national origin or ETS exposure (see Table E1 in the Online Repository at www.jacionline.org). Inclusion in the model of cockroach, mouse, or cat allergen levels found in dust samples collected in the children's homes had a minimal effect on the overall correlation or the β values for mothers and fathers.

We examined the relationship between parents' and children's inhalant allergen sensitization by looking for correlations in the sum of the specific IgE concentrations for dust mite, cockroach, mouse, and cat. The summed specific IgE values of the children were correlated with those of their mothers but not those of their fathers (Table III). The contribution of the mothers' summed specific IgE levels to those of the children (indicated by the Spearman rho value) was essentially equivalent for boys and girls and varied only modestly when stratified by age. No substantial change in the strength of the mother-child correlation was seen with ETS exposure or with birth order.

Association of parents' with children's atopy

We examined the correlation between children and parents with respect to the number of allergens to which each individual

TABLE I. Total IgE levels and distribution of allergen-specific sensitization in mother-father-child triads

	Boys (n = 83)		Girls (n = 78)		Fathers (n = 161)		Mothers (n = 161)		*P* value*
Mean age (y)	3.9 (0.5)		4 (0.5)						.6
Geometric mean total IgE (kU/L), range	70.4 (<2-2643)		41.6 (<2-1778)		91.2 (2.2->5000)		57.4 (2.0->5000)		.03
	n	%	n	%	n	%	n	%	*P* value†
Total IgE >100 kU/L	38	45.8	23	29.5	70	43.5	56	34.8	.04
Total IgE >150 kU/L	26	31.3	16	20.5	57	35.4	46	28.6	.2
Sensitized (specific IgE ≥0.35 kU/L) to:									
Dust mite	10	12.0	9	11.5	32	19.9	31	19.3	1
Cockroach	18	21.7	10	12.8	65	40.4	55	34.2	.2
Mouse	11	13.3	4	5.1	8	5.0	6	3.7	.1
Cat	10	12.0	2	2.6	23	14.3	17	10.5	.03
Any of the 4 allergens	25	31.1	18	23.1	78	48.4	72	44.7	.4

*Student *t* test, comparison of boys with girls.
†χ^2 Test, comparison of boys with girls.

TABLE II. Linear regression models of the associations of children's Ln(total IgE) with their mothers' and fathers' Ln(total IgE), within strata*

				Mothers		Fathers	
Stratified on:	Stratum	*r*	n	β	*P* value	β	*P* value
—	All	0.383	161	.244	.002	.230	.003
Age of child (mo)							
	<48	0.273	85	.082	.5	.072	.5
	≥48	0.570	76	.382	<.001	.383	.001
Sex of child							
	Girls	0.361	78	.127	.3	.077	.5
	Boys	0.509	83	.341	.001	.341	.002
No. of smokers in residence							
	0	0.409	123	.278	.002	.245	.006
	≥1	0.563	38	.275	.1	.223	.3
No. of older siblings							
	0	0.431	64	.252	.046	.312	.016
	1	0.519	51	.344	.014	.206	.2
	≥2	.488	46	.201	.2	.219	.1

*All models are adjusted for national origin.

TABLE III. Correlations of children's summed specific IgE levels with mothers' and fathers' summed specific IgE levels

			Mothers		Fathers	
Stratified on:	Stratum	n	Spearman rho*	*P* value	Spearman rho	*P* value
—	All	161	0.352	<.001	0.101	.2
Age of child (mo)						
	<48	85	0.332	.002	0.137	.2
	≥48	76	0.374	.001	0.063	.6
Sex of child						
	Girls	78	0.362	.001	0.121	.3
	Boys	83	0.369	.001	0.086	.4
No. of smokers in residence						
	0	123	0.350	<.001	0.160	.1
	≥1	38	0.336	.04	−0.075	.7
No. of older siblings						
	0	64	0.378	.002	0.256	.04
	1	51	0.398	.004	−0.215	.1
	≥2	46	0.333	.024	0.250	.1

was sensitized as an indicator of overall atopic risk. This index behaved very much like the summed specific IgE values. The association between children and their mothers (but not their fathers) reached statistical significance (see Table E2 in the Online Repository at www.jacionline.org). The mother-child correlation yielded modestly higher rho values for older than for younger children, for boys than for girls, and for ETS-unexposed than exposed children. There were no trends detected in this correlation with change in birth order.

We next examined the relationship between parental and child seroatopy, which was defined as the presence of detectable specific IgE to any of the allergens examined. Logistic regression analysis revealed that children whose mothers alone were seroatopic bore a 3- to 4-fold increased risk of seroatopy relative to children of nonseroatopic parents (Table IV). In stratified analyses the association of mothers' with children's seroatopy was significant for girls but not for boys and for children with one older sibling but not for children without older siblings. Children whose fathers alone were seroatopic differed little from children with no seroatopic parents.

When national origin was included in the seroatopy regression models, the associations between children's and mothers' seroatopy (mother alone or both mother and father) were modestly stronger (see Table E3 in the Online Repository at www.jacionline.org). When both national origin and ETS exposure were taken into account, the associations were even stronger. Including allergen exposure in the models did not affect the associations between children's and parents' seroatopy.

Predictive value of individual allergen sensitizations

To examine the associations between children's and parents' sensitizations to individual allergens, we used logistic regression models comparing children whose parents were sensitized (mother, father, or both) with those whose parents were not sensitized on an allergen-by-allergen basis (Table V). Mothers' sensitization to dust mite was associated with children's sensitization to each of the 4 allergens examined. Fathers' sensitization alone was generally not a significant predictor of children's sensitization. However, this analysis suggested that children both of whose parents were sensitized to dust mite were at increased risk for either dust mite or cockroach sensitization relative to children whose mothers alone were sensitized. The sensitization of both parents to cockroach was also associated with the child's sensitization to cockroach, as well as to mouse. These associations were evident in both unadjusted models and those adjusted for national origin. The inclusion of ETS exposure or allergen

TABLE IV. Relative odds of children's seroatopic status* given parents' seroatopic status within strata of other variables

Stratified on:	Parents' status	OR	95% CI	Parents' status	OR	95% CI	Parents' status	OR	95% CI
—	**Overall (n = 161)**								
	M–F–	1.0	Referent						
	M+F–	3.5	1.2-9.9†						
	M–F+	1.0	0.3-3.3						
	M+F+	5.0	1.8-14.0‡						
Age (mo)	**<48 (n = 85)**			**≥48 (n = 76)**					
	M–F–	1.0	Referent	M–F–	1.0	Referent			
	M+F–	3.2	0.7-15.0	M+F–	3.9	0.9-17.0			
	M–F+	1.0	0.2-5.5	M–F+	1.1	0.2-5.6			
	M+F+	7.0	1.5-32.0†	M+F+	3.6	0.9-15.0			
Sex	**Girls (n = 78)**			**Boys (n = 83)**					
	M–F–	1.0	Referent	M–F–	1.0	Referent			
	M+F–	15.0	1.6-134.0†	M+F–	1.6	0.4-6.4			
	M–F+	4.9	0.5-52.0	M–F+	0.4	0.1-2.1			
	M+F+	13.0	1.5-122.0†	M+F+	3.8	1.0-14.0†			
Smokers in residence	**0 (n = 123)**			**≥1 (n = 38)**					
	M–F–	1.0	Referent	M–F–	1.0	Referent			
	M+F–	3.5	0.9-13.0	M+F–	6.7	0.8-56.0			
	M–F+	1.4	0.4-5.9	M–F+	0.4	0.1-4.8			
	M+F+	8.3	2.3-30.0‡	M+F+	1.4	0.2-9.2			
Older siblings	**0 (n = 64)**			**1 (n = 51)**			**≥2 (n = 46)**		
	M–F–	1.0	Referent	M–F–	1.0	Referent	M–F–	1.0	Referent
	M+F–	0.8	0.1-5.1	M+F–	7.5	1.5 = 38.0†	M+F–	—	
	M–F+	—§		M–F+	0.9	0.2 = 4.6	M–F+	—	
	M+F+	4.4	0.9=20.0	M+F+	1.2	0.1 = 15.0†	M+F+	—	

OR, Odds ratio.
*Children were grouped by parental seroatopy (any specific IgE level ≥0.35 kU/L): neither parent seroatopic *(M-F-)*, mother alone or father alone seroatopic (*M+F−* and *M-F+*, respectively), or both parents seroatopic *(M+F+)*.
†$P < .05$.
‡$P < .01$.
§Indeterminate.

exposure levels in the regression models had no substantial effect on the associations (data not shown).

DISCUSSION

The studies reported here examine the associations between children's IgE levels at age 4 years and those of their parents in a sample of 161 families (mother, father, and child) recruited from New York City neighborhoods with high rates of asthma. We examined both total and allergen-specific IgE levels, focusing on a set of indoor allergens with high rates of sensitization among children in New York City. Although total and specific IgE levels are both commonly used as indicators of atopic tendency, we found that these 2 measures yielded distinct patterns of association between parent and child.

A small number of studies have recently highlighted the predictive value of parental total IgE levels.[8,9,28] Among our study families, both mothers' and fathers' total IgE levels were correlated with those of their children. The β value for the mother-child correlation was modestly higher than that for the father-child correlation, which is consistent with the findings of Burrows et al[8,28] among preadolescent children in Tucson, Arizona.

Total IgE level has been reported to increase steadily over the first 10 years of life,[29] and reflecting this trend, the older children in our sample (above median age) had a modestly higher geometric mean total IgE level than the younger children (64 vs 47 IU/mL, $P = .2$), despite the relatively narrow age range of the study sample (36-60 months). Examining the effect of age on the association of parents' with children's total IgE levels, we found that a correlation was evident for older, but not for younger, children (Table II). This age effect might explain the differences between our findings and those of Liu et al,[9] who found only a weak correlation of total IgE levels in 6-month-olds with their mothers ($r = 0.14$).

During the first 10 years of childhood, total IgE levels increase in parallel among girls and boys; however, boys reach a given median IgE level approximately 2 years before girls.[29] Consistent with this report, the boys in our sample had a higher geometric mean total IgE level than the girls (Table I), and this sex difference had a significant effect on the parent-child total IgE relationships: the correlation of children's total IgE levels with that of their parents was demonstrable for boys but not for girls. The effects of age and sex on the total IgE associations suggest that the correlation of parents' with children's total IgE levels might be more readily demonstrable among children as their IgE levels approach those of adults. In this regard it will be of interest to reassess the association of parents' with girls' total IgE levels as the girls continue to mature and IgE levels increase.

In contrast to the total IgE correlation, all measures involving children's allergen-specific sensitization yielded stronger associations with mothers' than with fathers' measures. Although we examined specific IgE levels to 4 allergens of clinical importance in this population,[21,22] the sum of the specific IgE levels generally represented a small fraction of the total IgE level for each seroatopic subject (range, 0.2% to 46%; median, 5%). That these 2 measures showed distinct patterns of association between parent and child might reflect differences in the relative importance of genetics and environment as determinants of total and specific IgE levels. The heritability of total IgE levels has been estimated from twin studies to be approximately 65%, with only 35% of

TABLE V. Relative odds of children's sensitization* to specific allergens given parents' sensitization to specific allergen†

		Children's sensitization to:											
		Dust mite (n = 37)			Cockroach (n = 146)			Mouse (n = 133)			Cat (n = 130)		
Allergen	Parents' status	n	OR	95% CI	n	OR	95% CI	n	OR	95% CI	n	OR	95% CI
Dust mite													
	M–F–	97	1.0	Referent	104	1.0	Referent	96	1.0	Referent	95	1.0	Referent
	M+F–	13	11.0	2.5-45.0‖	14	6.4	1.9-22.0‖	12	13.0	2.8-60.0‖	12	16.0	3.2-84.0‖
	M–F+	18	3.0	0.6-15.0	19	1.9	0.5-7.2	17	2.6	0.4-16.0	16	1.2	0.1-13.0
	M+F+	9	24.0	3.0-184.0‖	9	12.0	1.8-78.0§	8	11.0	1.2-109.0§	7	6.9	0.6-79.0
Cockroach													
	M–F–	56	1.0	Referent	54	1.0	Referent	52	1.0	Referent	50	1.0	Referent
	M+F–	26	1.4	0.2-12.0	32	6.2	1.4-29.0§	28	6.5	0.8-49.0	30	—	
	M–F+	41	1.7	0.5-5.9	42	2.7	0.7-9.9	37	2.1	0.3-15.0	37	—	
	M+F+	14	3.8	0.7-19.0	18	10.0	2.5-44.6‖	16	18.0	2.6-134.0‖	13	—	
Mouse													
	M–F–	126	1.0	Referent	134	1.0	Referent	123	1.0	Referent	121	1.0	Referent
	M+F–	5	1.6	0.1-27.0	5	2.0	0.1-31.0	5	6.5	0.3-144.0	5	6.5	0.3-144.0
	M–F+	5	3.5	0.4-28.0	7	6.7	1.3-35.0§	5	9.0	1.0-83.0	4	3.7	0.2-56.0
	M+F+	1	—‡		0	—		0	—		0	—	
Cat													
	M–F–	109	1.0	Referent	115	1.0	Referent	103	1.0	Referent	103	1.0	Referent
	M+F–	9	0.6	0.1-7.0	9	0.7	0.1-6.4	9	2.4	0.2-26.0	10	4.5	0.7-30.0
	M–F+	14	0.5	0.1-4.5	17	1.3	0.4-4.5	17	3.0	0.8-12.0	13	—	
	M+F+	5	7.7	0.6-106.0	5	12.0	0.7-213.0	4	—		4	—	

OR, Odds ratio; *M*, male; *F*, female; *M-F-*, neither parent seroatopic; *M+F−*, mother alone seroatopic; *M-F+*, father alone seroatopic; *M+F+*, both parents seroatopic.
*Children who have specific IgE levels of 0.35 IU/mL or greater to the designated allergen compared with those with no detectable IgE to any allergen.
†All regressions adjusted for national origin.
‡Indeterminate.
§$P < .05$.
‖$P < .005$.

variance in total IgE levels attributable to environmental factors.[30] In contrast, the development of allergen-specific IgE levels has an absolute requirement for environmental exposure. Given this difference, associations between parents' and children's specific IgE levels are likely to be more dependent on the shared environment than associations of total IgE levels. In this regard more than 95% of the children in this sample were raised in homes where both parents reside, and in 94.5% of households, the mother was reported to be the main caretaker, whereas in 90% the fathers were employed outside the home. Therefore with respect to measures of specific IgE, the stronger association of child with mother than with father might reflect a disproportionate amount of time spent by mothers and children in a shared environment.

In a larger subset of the Head Start study cohort, we have previously demonstrated an association between cockroach allergen in vacuumed dust from the homes of these children and cockroach-specific IgE levels at age 4 years.[26] With the current study, we were interested in testing for potential confounding of the parent-child IgE associations by allergen exposure in the home. Inclusion of exposure to cockroach, mouse, and cat allergen in the association models, however, did not substantially alter the regression results for either total or allergen-specific IgE levels. This was the case whether allergen levels were handled as dichotomous (see Tables E1 and E3) or continuous variables (data not shown). The lack of change in the parameter estimates for parents' IgE levels predicting children's IgE levels, after adjusting for the allergen measurements, indicates that the associations were not confounded by allergen exposure, as reflected in vacuumed dust samples. Dust mite allergen levels in New York City multiple-dwelling units are typically very low[31,32] and therefore were not assessed in this study.

The allergen-specific associations between parent and child also differed from the total IgE associations with respect to the factors that proved to be significant effect modifiers. Child age, for example, had little effect on the allergen-specific associations. Similarly, stratification by sex did not reveal sex differences in the parent-child correlations of summed specific IgE levels or number of sensitizations, although sex did affect the predictive value of maternal seroatopy (Table IV). Stratification by ETS exposure or by birth order did not reveal substantial effects on either total or specific IgE level correlations.

Including national origin in the regression models modestly strengthened the correlation of parents' and children's total IgE levels (see Table E1) and improved the predictive value of mothers' and both parents' seroatopy (see Table E3). ETS exposure was examined as a possible confounder (as well as an effect modifier) because of its potential to affect both parents' and children's IgE levels. Inclusion of ETS exposure also produced a slight improvement in the parent-child total IgE level correlation and the association of maternal with child seroatopy. Inclusion of both national origin and ETS exposure further strengthened parent-child associations.

Among the limitations of this study, participants were specifically recruited from New York City neighborhoods with high pediatric asthma hospitalization rates.[23] Our sample might therefore have included a disproportionate number of atopic children, and findings in our sample might be limited in generalizability to larger US populations. The overall Head Start study sample might also have been enriched for atopic children because of selection bias reflecting parental interest in participation. Among the study families, however, there was no difference between the M-F-C triad subgroup (n = 161) and the overall Head Start home visit

sample (n = 517) with respect to age, sex, IgE levels, or rates of sensitization or with respect to prevalence of asthma and allergic rhinitis symptoms (data not shown).

One factor potentially influencing both children's and parents' atopy rates is socioeconomic status. All study families had income levels below the federal poverty threshold, a requirement for eligibility in the Head Start preschool program. To evaluate socioeconomic status within this low-income population, we categorized families according to the mother's years of education completed. The triad families were nearly equally distributed among 4 categories: fewer than 9 years, 9 to 11 years, 12 years, and greater than 12 years of education. Inclusion of this index in the regression models for total IgE level, seroatopy, and allergen-specific IgE level did not affect the associations (data not shown).

The degree to which we were able to perform stratified analyses was limited by sample number. Although IgE analyses were performed on 501 Head Start children, the analyses reported here were restricted to the 161 families for which triad data were available to be able to examine the relative predictive value of mother's and father's IgE levels. Because multiple statistical tests were performed in this analysis, P values are indicated for each test to facilitate interpretation of the level of significance. The vast majority of the statistical tests supporting the major findings yielded highly significant P values ($P \leq .01$).

Taken together, the findings presented here confirm the correlation of total IgE levels between parents and their children at age 4 years and indicate that specific sensitizations among parents might provide an additional means of predicting sensitization in children independent of total IgE. Although the presence of specific IgE to environmental antigens does not equate with clinical allergic disease, sensitization is an absolute requirement for atopic conditions, including allergic asthma. The relevant allergens will likely vary among communities examined, but our results provide justification for further studies of the associations between parents' and children's IgE levels.

Clinical implications: Preventive interventions might be targeted to children at high risk of sensitization on the basis of parents' sensitization patterns.

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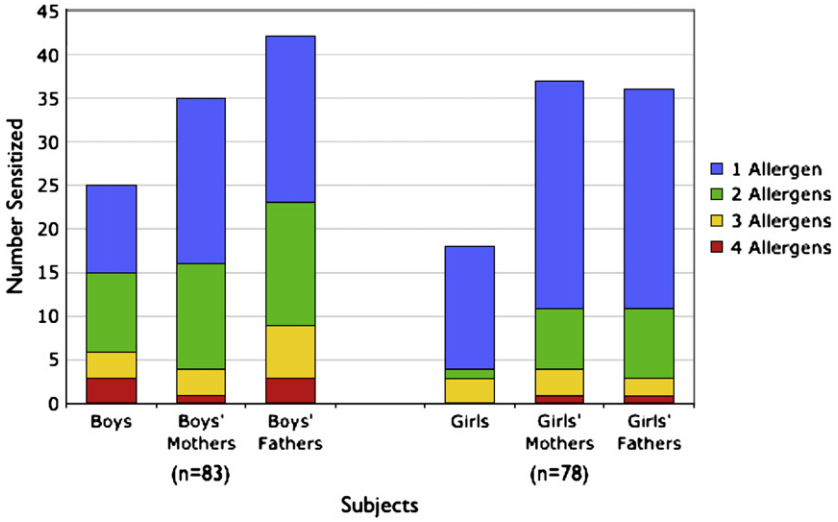

FIG E1. Number of participants sensitized among M-F-C triads to any of 4 allergens (dust mite, cockroach, mouse, cat). Each *bar* is divided into 4 sections. The height of each section represents the number sensitized to 1 *(blue)*, 2 *(green)*, 3 *(yellow)*, or 4 *(red)* allergens. The full height of each *bar* represents the number sensitized to any allergen.

TABLE E1. Linear regression models of the associations of children's Ln(total IgE) with mothers' and fathers' Ln(total IgE) adjusted for national origin, tobacco smoke exposure, and home allergen levels

		Mothers		Fathers	
	r	β	*P* value	β	*P* value
M-F-C triads (n = 161)					
Unadjusted	0.361	.247	.001	.245	001
Adjusted models					
National origin	0.383	.244	.002	.230	.003
ETS exposure*	0.378	.256	.001	.253	.001
National origin and ETS exposure	0.410	.259	.001	.241	.002
Allergen subset† (n = 110)					
Unadjusted	0.353	.283	.002	.192	.037
Adjusted models					
Cat >8 μg/g	0.364	.281	.003	.195	.034
Mouse >1.6 μg/g	0.369	.290	.002	.187	.041
Cockroach >2 U/g	0.367	.282	.002	.185	.044
All 3 allergens	0.398	.285	.002	.185	.044

*ETS exposure defined as report of 1 or more tobacco smokers living in the residence.
†Triad subgroup for which 3 allergen levels were assayed. Vacuumed dust was collected from the kitchen (cockroach and mouse) and child's bed (cat).

TABLE E2. Correlations of children's numbers of sensitizations (among 4 allergen-specific IgEs examined) with mothers' and fathers' numbers of sensitizations*

			Mothers		Fathers	
Stratified on:	**Stratum**	**n**	**Spearman rho**	***P* value**	**Spearmen rho**	***P* value**
—	All	161	0.339	.000	0.116	.1
Age of child (mo)						
	<48	85	0.288	.007	0.159	.1
	≥48	76	0.391	.000	0.074	.5
Sex of child						
	Girls	78	0.323	.004	0.108	.3
	Boys	83	0.363	.001	0.116	.3
No. of smokers in residence						
	0	123	0.360	.000	0.189	.037
	≥1	38	0.281	.1	−0.086	.6
No. of older siblings						
	0	64	0.325	.009	0.245	.1
	1	51	0.412	.003	−0.177	.2
	≥2	46	0.303	.040	0.278	.1

*Number of sensitizations (specific IgE ≥0.35 kU/L) to any of 4 allergens ranked as 0, 1, 2, and 3 or more.

TABLE E3. Relative odds of children's seroatopic status* given parents' seroatopic status: Regression models adjusted for national origin, ETS exposure, and home allergen levels

M-F-C triads		OR (95% CI) Adjusted models		
Parental seroatopy status	Unadjusted (n = 161)	National origin	Smoker in residence (ETS)	National origin + ETS
M–F–	Referent	Referent	Referent	Referent
M+F–	3.5 (1.2-9.9)‡	5.7 (1.6-20)§	4.2 (1.4-13)‡	6.8 (1.8-26)§
M–F+	1.0 (0.3-3.3)	1.2 (0.3-4.0)	1.0 (0.3-3.3)	1.2 (0.3-3.9)
M+F+	5.0 (1.8-14)§	6.5 (2.1-20)§	5.0 (1.8-14)§	6.7 (2.1-21)§

Allergen subset†		OR (95% CI) Adjusted models			
Parental seroatopy status	Unadjusted (n = 110)	Cockroach allergen >2 U/g	Cat allergen >8 μg/g	Mouse allergen>1.8 μg/g	All 3 allergens included
M–F–	Referent	Referent	Referent	Referent	Referent
M+F–	2.9 (0.9-9.4)	2.9 (0.9-9.3)	2.9 (0.9-9.4)	3.0 (0.9-9.4)	3.0 (0.9-9.5)
M–F+	1.0 (0.2-3.7)	1.0 (0.3-3.6)	1.0 (0.2-3.8)	1.0 (0.2-3.6)	0.9 (0.2-3.7)
M+F+	5.8 (1.7-19)§	6.0 (1.8-20)§	6.3 (1.8-22)§	5.4 (1.6-18.0)§	6.1 (1.7-22)§

M, Male; *F*, female.

*Children were grouped by parental seroatopy (any specific IgE level ≥0.35 kU/L): neither parent seroatopic *(M–F–)*, mother alone or father alone seroatopic (*M+F−* and *M−F+*, respectively), or both parents seroatopic *(M+F+)*.

†Triad subgroup for which 3 allergen levels were assayed. Vacuumed dust was collected from the kitchen (cockroach and mouse) and child's bed (cat).

‡$P < .05$.

§$P < .01$.