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Racial Disparities in Allergic Outcomes in African Americans Emerge as Early as Age 2 Years

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Abstract

BACKGROUND—Racial disparities in allergic disease outcomes have been reported with African Americans suffering disproportionately compared to White individuals.

OBJECTIVE—Examine whether racial disparities are present as early as age 2 years in a racially diverse birth cohort in the Detroit metropolitan area.

METHODS—All children who were participants in a birth cohort study in the Detroit metropolitan area were invited for a standardized physician exam with skin prick testing and parental interview at age 2 years. Physicians made inquiries regarding wheezing and allergy symptoms and inspected for and graded any atopic dermatitis (AD). Skin testing was performed for *Alternaria*, cat, cockroach, dog, *Dermatophagoides farinae* (Der F), Short Ragweed, Timothy grass, egg, milk and peanut. Specific IgE was measured for these same allergens and total IgE was determined.

RESULTS—African American children (n=466) were more likely than White children (n=223) to have experienced any of the outcomes examined: at least 1 positive skin prick test from the panel of 10 allergens (21.7% versus 11.0%, p=0.001); at least one specific IgE ≥ 0.35 IU/mL (out of a panel of 10 allergens) (54.0% versus 42.9%, p=0.02); had AD (27.0% versus 13.5%, Chi-square p<0.001); and to ever have wheezed (44.9% versus 36.0%, p=0.03). African American children also tended to have higher total IgE (geometric means 23.4 IU/mL (95%CI 20.8, 27.6) versus 16.7 IU/mL (95%CI 13.6, 20.6 IU/mL), Wilcoxon Rank Sum p=0.004). With the exception of wheezing, the associations did not vary after adjusting for common social economic status variables (e.g.; household income), environmental variables (endotoxin; dog, cat and cockroach allergen in house dust) or variables that differed between the racial groups (e.g.; breastfeeding). After adjustment, the wheeze difference was ameliorated.

CONCLUSIONS—With disparities emerging as early as age 2 years, investigations into sources of the disparities should include the prenatal period and early life.

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Conflict of Interest

No author has any financial relationships with a biotechnology and/or pharmaceutical manufacturer that has an interest in the subject matter or materials discussed in the submitted manuscript.

Keywords

allergic disease; racial disparities; IgE; atopy

INTRODUCTION

Childhood atopic diseases, including asthma, cause significant morbidity and mortality and are common in the westernized world.[1] In particular, asthma is the most common chronic disease in childhood and has increased in frequency over the last several decades with half of the current cases of asthma in the United States being attributable to atopy.[2] It is also estimated that the majority (54.3%) of the United States population is atopic (sensitized to 1 allergen) – a prevalence that is 2.1–5.5 times higher than the prevalence from a decade earlier.[3] Therefore prevention of atopy has been considered a leading strategy to decrease asthma.[2,4]

Racial disparities in allergic disease outcomes have been reported with African Americans suffering disproportionately compared to White individuals.[3,5–11] Sources of racial differences in allergic disease may not be fully explained by social and economic status (SES). For example, our research group has shown that even among middle class children, African Americans may be more susceptible to asthma.[12,13] Based on census and health interview survey data in Rhode Island, Black children were more likely to have asthma than either their White or Hispanic counterparts even after adjusting for measures of family and neighborhood socioeconomic position.[14]

Our goal was to examine whether racial disparities are present as early as age 2 years in a racially diverse birth cohort established in the Detroit metropolitan area. Additionally, we sought to determine whether any observed racial differences could be explained, either fully or in part, by various components of SES.

METHODS

Study Population

“WHEALS” is an NIH and institutionally funded cohort study that enrolled pregnant women receiving care at Henry Ford Health System (HFHS) obstetrics clinics in urban and suburban Detroit, Michigan, for longitudinal study of their offspring through early childhood with the goal of examining early life exposures related to childhood allergic diseases. Recruitment began in September 2003. These children serve as the source population for these analyses. The study provides interview data, a clinic visit with a study physician and results from biological and environmental samples that are incorporated into these analyses. Details of the cohort’s creation have been published.[15,16]

The last “WHEALS” birth was in December 2007 after enrolling 1258 pregnant women. Since enrollment, 241 (19.2% of total enrolled) children have been dropped from the study. The majority were removed for noncompliance with the study protocol (no child blood collected or interviews completed) (n=108); some families have moved from the area (n=62), six children died and one infant has been in child protective services custody. The remaining children were lost to follow up (n=64).

The children included in the present analyses have information on at least one outcome at age 2 years (information on wheeze, or results from skin prick testing (SPT), total IgE, allergen specific IgE (sIgE), or evaluation of atopic dermatitis (AD)). The child’s race was based on maternal report for her child at age 2 years. We are including only those children

who are African American and White/Non-Hispanic/Non-Middle Eastern to examine racial disparities. The subgroups of children who are White/Hispanic or White/Middle Eastern may have different risks for the outcomes and are too few in number to analyze as subgroups.[3] Participants who were included and not included in the analyses are compared to assess differences (Table 1).

Data Collection

The baseline interview at recruitment during pregnancy included questions about maternal educational status, household income, and whether or not the mother paid for housing (rent or a mortgage). The mother's address at recruitment was used to determine whether the residence was in an urban (Detroit, Hamtramck, Ypsilanti) or suburban (all other cities) setting. We also asked the mother about her marital status. As part of the study protocol, additional interviews were collected with the mothers at 1, 6 and 12 month home visits about the health of their children, breastfeeding and exposure to animals and tobacco smoke. Dust samples were collected from the children's bedroom floor in the first year of life. Samples were analyzed for cat allergen (Fel d 1), dog allergen (Can f 1), cockroach allergen (Bla g 2) and endotoxin using methods we have previously published (DRO Ped Allergy Immunol 2010).

All children were invited for a standardized physician exam with skin prick testing and parental interview at age 2 years with a physician trained in the study protocol. The physicians received a history of wheezing and allergy symptoms and inspected for and graded any atopic dermatitis (AD). Children were evaluated by a physician using history, physical examination and SCORAD (a system to quantify symptoms and signs of atopic dermatitis). The physician was then asked to record a response to the following question: "By your clinical evaluation do you believe that this child has or has had atopic dermatitis or eczema?". The average age at completion of the 2 year clinic visit was 2.2 years (standard deviation = SD = 0.2 years).

If the child was free of wheezing and there were no other contraindications, skin testing was performed by the puncture method on the volar surfaces of the forearms using a Duotip-test® device (Lincoln Diagnostics, Inc., Decatur, IL) for *Alternaria*, cat, cockroach, dog, *Dermatophagoides farinae* (Der F), Short Ragweed, Timothy grass, egg, milk and peanut. Tests were spaced at least 6 cm apart to prevent interference. The order of the allergens was randomly assigned in four different patterns to eliminate any bias of size that might have resulted from the position of the tests on the arm. A negative saline control and a positive control of histamine, 1 mg/ml, were included with all tests. The extracts for skin testing were commercial extracts at the following concentrations: 1:20 w/v for dog, short ragweed and *Alternaria* (Bayer Corp, Spokane, WA), 10,000 AU/ml concentrations of the dust mite and grass and 5000 BAU of standardized cat extract. Fifteen minutes after the skin tests were placed the resulting wheal and flare responses were measured at the longest diameter. All measurements were recorded in the 2 year clinic visit form. A positive skin test was defined as one producing a wheal with the longest diameter at least 3 mm greater than the negative control.

Blood was collected to determine levels of total and allergen-specific IgE [sIgE: dust mite (*Der f*), dog, cat, Timothy grass, ragweed, *Alternaria*, egg, peanut, milk and cockroach (*Blatella germanica*)]. Measurement of total IgE and sIgE was performed according to the manufacturer's standard protocols using the Pharmacia UniCAP system (Pharmacia-Upjohn Diagnostic Division, Kalamazoo, Michigan, USA). A positive sIgE was defined as ≥ 0.35 kU/l.

Statistical Analyses

Chi square tests and t tests were used to compare characteristics of participants who were included and excluded from the analyses. Percentages (binary outcomes) and geometric means (continuous outcomes) were used to describe the outcomes and Chi square and Wilcoxon Rank Sum tests were used to compare the outcomes between African American and White children. Logistic regression models were used to investigate binary outcomes (at least 1 positive SPT, at least 1 positive sIgE, AD, any wheeze) and linear regression was used to model continuous data (log transformed total IgE) to determine whether SES and other factors could explain the observed differences. Data were transformed as needed to meet model assumptions.

No single definition of SES has proven to explain racial disparities in allergic diseases. We used numerous factors to consider SES in our analyses including: household income, maternal education and whether the child's family paid for housing (made a rent or mortgage payment).

We also considered additional factors not necessarily considered SES qualities in the analyses including whether the mother was exposed to indoor pets during pregnancy, whether the child was ever breastfed and maternal marital status (was she married/living as married or not). Based on maternal report, we also considered whether the child lived with a smoker in early childhood, as well as the reported parental history of allergy. We also considered the child's birth weight (from delivery record), season of birth, maternal age at study enrollment and the child's gender. Since not all children had dust samples for analyses, we included additional models for the children who did have dust samples with individual adjustments for the samples (endotoxin, dog allergen, cat allergen, cockroach allergen) and for simultaneous adjustment for all variables including results from the dust analyses.

Our analyses considered how the association between race and the outcome varied in models with the addition of the SES and other factors. We could not adjust for whether the family home was in an urban or suburban setting as 122 (26.2%) African American children lived in a suburban setting and 90% of White children lived in an suburban setting; thus race was highly correlated with residence setting.

RESULTS

There were 223 White children and 466 African American children who participated in the clinic visit and thus the proportion of cohort subjects included in the analyses were 66% and 58%, respectively. Among White children, those who participated in the clinic visit at age 2 years tended to have higher family income and maternal education. They also tended to have greater birthweight and be born in the winter and spring and had a slightly higher maternal age at enrollment (Table 1). Among African American children, those included tended to have higher maternal education, were more likely to have mothers who kept indoor pets during pregnancy, were more likely to have mothers who were married or living as married, be less likely to live with a smoker and had slightly older mothers at enrollment (Table 1). Table 1 indicates that while there are overall differences in the distributions of income and maternal education between White and African American households, all income and maternal education levels are represented in the included study samples of White and African American children.

Skin Prick Testing (SPT)

African American children were more likely than White children in our cohort study to have had at least 1 positive skin prick test from a panel of 10 allergens at age 2 years (21.7%

versus 11.0%, $p=0.001$; odds ratio=OR = 2.26, 95% confidence interval=CI= 1.37, 3.73) (Table 2). After adjusting for each SES and other factor individually, the adjusted ORs did not differ from the crude OR (Table 2). When the association between race and SPT was adjusted for all the SES and other factors simultaneously, the OR did not change appreciably (adjusted OR=2.52, 95% CI= 1.32, 4.82) (Table 2).

Total IgE and sIgE

African American children were more likely to have had at least one positive specific IgE 0.35 IU/mL (out of a panel of 10 allergens) at age 2 years compared to White children (54.0% versus 42.9%, $p=0.02$) (Table 2). Compared with White children, African American children in WHEALS had higher total IgE at age two years (geometric means 23.4 IU/mL (95% CI 20.8, 27.6) versus 16.7 IU/mL (95% CI 13.6, 20.6 IU/mL), Wilcoxon Rank Sum $p=0.004$). Based on a linear regression model, the unadjusted beta for race in the model of log transformed total IgE was 0.36 (95% CI=0.11, 0.61). This suggests African American children have a total IgE level that is approximately 43% higher than their White counterparts. Adjusting for any individual factor or all factors simultaneously did not appreciably alter this association (Table 3).

After adjusting for each SES and other factor individually, the adjusted ORs did not differ from the crude OR for sIgE (1.57, 95% CI=1.07, 2.29) (Table 2). When the association between race and having at least one elevated allergen-specific IgE was adjusted for all the SES and other factors simultaneously, the OR did not change substantially (adjusted OR=1.99, 95% CI= 1.19, 3.31) (Table 2).

Atopic Dermatitis

In WHEALS, African American children were more likely than White children to have had AD (27.0% versus 13.5%, Chi-square $p<0.001$) (Table 2). After adjusting for each SES and other factor individually, the adjusted ORs did not differ from the crude OR for AD (2.36, 95% CI=1.50, 3.73) (Table 2). When the association between race and having AD was adjusted for all the SES and other factors simultaneously, the OR did not change appreciably (adjusted OR=1.95, 95% CI= 1.09, 3.47) (Table 2).

Wheeze

In WHEALS, African American children were more likely than White children to have ever wheezed in the first two years of life (44.9% versus 36.0%, $p=0.03$) (Table 2). After adjusting for each SES and other factor individually, the adjusted ORs did not differ from the crude OR for wheeze (1.45, 95% CI=1.03, 2.04) (Table 2). When the association between race and ever having wheezed was adjusted for all the SES and other factors simultaneously, the weak crude OR changed by approximately 30% (adjusted OR=1.11, 95% CI= 0.71, 1.75) and the association was no longer statistically significant (Table 2).

Analyses for Children with Dust Samples

The results indicate that the associations were not changed significantly with adjustment for endotoxin, dog allergen, cat allergen or cockroach allergen levels individually or simultaneously for all variables (Table 2 and Table 3).

DISCUSSION

Racial disparities in each of the allergic disease outcomes examined, with the exception of wheeze, were apparent in our study population at age 2 years with African American children experiencing higher rates/levels of each of the outcomes examined compared to White children. These findings indicate that potential sources of disparities may occur, at

least in part, in very early life including the prenatal period and the first 2 years of life. A major implication of this finding is that the focus of future investigations into sources of racial disparities and allergic disease causation must begin during pregnancy and infancy.

There is some prior documentation of racial differences in the outcomes investigated; however, our data provide the earliest indication of the disparities. In an analysis of NHANES II data, African American children ages 6–16 years were more likely to be SPT+ for German cockroach, dust mite and the mold *Alternaria* compared with White children.[8] NHANES III data indicated that Black individuals ages 6–59 years were more likely than White individuals to have had a positive skin prick test to at least one of ten allergens (adjusted OR=1.6, 95% CI 1.4, 1.9).[3] In their study of 882 pregnant women (169 were Black and 577 were White), Litonjua et al. reported higher total IgE for Black women compared with White women.[9] Black women were also approximately 2.5 times more likely than White women to be sensitized to 3 allergens. Results from the 2003 National Survey of Children's Health reported the odds of having had a doctor's diagnosis of AD was almost double among African American children under 18 years of age compared to White children.[10] Black individuals were twice as likely as White individuals to seek treatment for AD (54.4 versus 25.7 visits per 10,000 population) in the United States based on the 1990–1998 National Ambulatory Medical Care Survey (not limited to any age group).[11]

In the CDC's May 3, 2011 Morbidity and Mortality Weekly Report, it was reported that asthma was most common in non-Hispanic Black children and had increased from 2001 to 2009 (17.0% from 11.4% in 2001).[6] The prevalence among White children was reported as 8.5%.[6] Based on data from numerous national surveys and health statistic reports (1980–2007), Akinbami et al. reported that 1 in 11 children in the United States has asthma and that Black children were 1.6 times as likely as White children to have asthma.[5] The disproportionately higher prevalence of severe childhood asthma and its medical consequences (emergency department and acute care visits) among African Americans have been well documented.[7]

With or without adjustment for SES and other factors, the rates of wheeze in this study did not differ tremendously between African American and White children at this early age. We did not collect information on repeated wheeze and this was a limitation. We only asked whether the child ever wheezed. This lack of specificity about wheezing could have contributed to its moderate to weak observed associations.

Our analyses also found that SES factors and other key variables that sometimes differ by race used in our analyses could not fully explain the racial differences in allergic outcomes – associations between race and the outcomes persisted even after adjusting for these common SES and other variables. However, while the sources of well-documented racial disparities have not been thoroughly studied, racial disparities in management of allergic diseases, especially asthma, have garnered much attention from researchers.[18–21] Our work focused on racial disparities in disease incidence rather than racial disparities in disease management. Factors related to causation are poorly understood and novel hypotheses are needed to address this problem. As our research group has proposed, prevention is as important as disease management if racial disparities in asthma are to be eliminated.[22]

Our results further suggest that other potential sources of these racial disparities, beyond frequently collected SES data, should be investigated. Genetic variations by race have been an exciting research area [23,24]; additional avenues should be pursued. For example, vitamin D represents a clear opportunity for research as it has received much recent attention in the area of allergic disease causation.[25–34] There is an epidemic of vitamin D deficiency that may have been caused by changes in lifestyles in westernized cultures,

especially the United States, over the last several decades.[35,36] The epidemic has also disproportionately affected African Americans compared to lighter skinned individuals. In NHANES 2001–2004, Black children ages 1–21 years were more than 20 times as likely as White children to have 25(OH)D deficiency (defined as <15 ng/ml) (age and gender adjusted odds ratio (OR)=24.2, 95% confidence interval (CI) 15.9, 36.9).[35] Lack of exercise, nutrition, and inner city air pollution, especially diesel exhaust, are also possible causes of increased asthma prevalence among African Americans living in cities.

We do not have data on all environmental exposures including airborne particulates. The racial disparities observed could be caused by environmental differences since they would be part of the child's early exposures. However, in this study, adjustments for endotoxin and some allergen (dog, cat, cockroach) levels in the home did not explain the observed racial disparities. Further, our prior research on racial disparities in children in suburban Detroit would suggest that the environment does not fully explain observed racial differences.[12]

There were additional limitations to our study. Ideally, we would have examined the association with race separately for urban and suburban residents; however, this was not possible. Participation rates for the clinic visit at age 2 years were not as high as we had hoped for; however, the factors that differed between included and excluded children were considered in our analyses. Following diverse cohorts is important, but a challenge; our participation rates at 2 years of age are similar to those of other NIH-funded birth cohort studies in the United States comprised of urban and suburban women not selected by allergic status.[37] Further, those included in the analyses tended to be more educated and have higher income. These and other observed differences should be considered when comparing our results to other populations.

In summary, racial disparities in allergic disease related outcomes were seen as early as age 2 years in our cohort study with African American children being more likely to have: a positive skin prick test, an elevated allergen-specific IgE and atopic dermatitis. African American children also tended to have higher total IgE. The associations between race and these outcomes could not be explained by family income, maternal education or marital status, family housing payment, prenatal exposure to indoor pets, living with a smoker, breastfeeding or a host of other factors. A less marked association between race and wheeze dissipated when adjusted for all the SES and other factors. Novel hypotheses beyond those related to SES (or better ways of measuring SES) are needed to explain the causes of these racial disparities and investigations of the causation of disparities must include information from very early life.

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Table 1

Comparison of women from WHEALS whose children were included or excluded from the current analyses.*

	White Women with a Child Included in the Analyses	White Women with a Child Excluded from the Analyses	African American Women with a Child Included in the Analyses	African American Women with a Child Excluded from the Analyses
N	223	114	466	338
Number of older siblings (mean (SD))	1.0 (1.0)	1.1 (1.3)	1.1 (1.2)	1.2 (1.4)
P (included versus excluded)	0.44		0.40	
Reported Household income *				
<\$20K	10 (4.9%)	16 (15.4%)	67 (17.1%)	65 (21.6%)
\$20K-<\$40K	27 (13.2%)	23 (22.1%)	120 (30.5%)	99 (33.0%)
\$40K-<\$80K	73 (35.6%)	36 (34.6%)	115 (29.3%)	90 (30.0%)
\$80K-<\$100K	42 (20.5%)	13 (12.5%)	48 (12.2%)	23 (7.7%)
\$100K	53 (25.8%)	16 (15.4%)	43 (10.9%)	23 (7.7%)
P (included versus excluded)	0.001		0.11	
Maternal education				
Less than high school graduate	5 (2.2%)	9 (7.9%)	24 (5.2%)	28 (8.3%)
High school graduate	24 (10.8%)	27 (23.7%)	84 (18.0%)	77 (22.8%)
Some college	77 (34.5%)	42 (36.8%)	253 (54.3%)	185 (54.7%)
At least a Bachelor's degree	117 (52.5%)	36 (31.6%)	105 (22.5%)	48 (14.2%)
P (included versus excluded)	<0.001		0.006	
Makes a monthly mortgage or rental payment (n%) yes	193 (86.6%)	95 (83.3%)	341 (73.2%)	248 (73.4%)
P (included versus excluded)	0.04		0.36	
Indoor pet(s) at predelivery (n%) yes	136 (61.0%)	73 (64.0%)	134 (28.8%)	67 (19.8%)
P (included versus excluded)	0.59		0.004	
Ever breastfed the child (n%) yes	150 (67.3%)	48 (56.5%)	227 (48.7%)	111 (49.1%)
P (included versus excluded)	0.08		0.92	
Mother was married or living as married during pregnancy	193 (86.8%)	90 (79.0%)	245 (52.6%)	145 (42.9%)
P (included versus excluded)	0.07		0.007	
Child lived with a smoker	51 (22.9%)	37 (32.5%)	122 (26.2%)	117 (34.6%)
P (included versus excluded)	0.06		0.010	
Home is in an urban rather than suburban setting	22 (9.9%)	15 (13.2%)	344 (73.8%)	263 (77.8%)
P (included versus excluded)	0.36		0.19	
Parental History of Allergy	94 (45.6%)	46 (41.4%)	146 (35.0%)	85 (28.4%)
P (included versus excluded)	0.47		0.06	
Birthweight Mean (SD)	3558 (546)	3278 (531)	3260 (579)	3206 (571)
P (included versus excluded)	<0.001		0.21	
Season of birth				
Winter	49 (22.0%)	15 (13.2%)	90 (19.3%)	79 (23.4%)
Spring	58 (26.0%)	25 (21.9%)	92 (19.7%)	78 (23.1%)
Summer	65 (29.1%)	31 (27.2%)	126 (27.1%)	90 (26.6%)

	White Women with a Child Included in the Analyses	White Women with a Child Excluded from the Analyses	African American Women with a Child Included in the Analyses	African American Women with a Child Excluded from the Analyses
Fall	51 (22.9%)	43 (37.7%)	158 (33.9%)	91 (26.9%)
P (included versus excluded)	0.022		0.13	
Maternal age at enrollment Mean (SD)	31.0 (4.5)	30.0 (5.2)	29.3 (5.4)	28.5 (5.2)
P (included versus excluded)	0.08		0.027	
Child's gender				
Male	114 (51.1%)	48 (42.1%)	243 (52.2%)	157 (46.7%)
Female	109 (48.9%)	66 (57.9%)	223 (47.8%)	180 (53.4%)
P (included versus excluded)	0.12		0.12	

* There are some missing data. P-values are for a comparison of those included and those excluded within the racial group for that factor.

Table 2

The crude and adjusted odds ratios (95% confidence intervals) describing the effect of SES and selected variables on the relationship between race (African American versus White) and outcomes

	N=597	N=513	N=607	N=621
	1 positive skin prick test n (%)	1 positive specific IgE n (%)	Atopic dermatitis n (%)	Any wheeze first 2 years n (%)
African American	86 (21.7%)	194 (54.0%)	108 (27.0%)	184 (44.9%)
White	22 (11.0%)	66 (42.9%)	28 (13.5%)	76 (36.0%)
p-value	0.001	0.02	<0.001	0.03
Crude OR (95% CI)	2.26 (1.37–3.73)	1.57 (1.07–2.29)	2.36 (1.50–3.73)	1.45 (1.03–2.04)
OR (95% CI) for each outcome comparing African American to White children adjusting for each of the following individually:				
Number of older siblings	2.32 (1.40–3.85)	1.59 (1.09–2.33)	2.39 (1.51–3.77)	1.44 (1.02–2.02)
Report of Household income	2.49 (1.47–4.24)	1.78 (1.18–2.69)	2.27 (1.41–3.66)	1.34 (0.93–1.93)
Maternal education	2.15 (1.27–3.62)	1.71 (1.13–2.57)	2.30 (1.43–3.69)	1.29 (0.90–1.84)
Make monthly mortgage or rental payments	2.23 (1.34–3.71)	1.53 (1.04–2.25)	2.38 (1.50–3.76)	1.47 (1.04–2.08)
Prenatal indoor pet(s)	1.98 (1.18–3.35)	1.56 (1.06–2.31)	2.16 (1.34–3.48)	1.36 (0.95–1.94)
Ever breastfed the child	2.31 (1.39–3.85)	1.67 (1.13–2.47)	2.40 (1.51–3.81)	1.34 (0.94–1.90)
Mother was married or living as married during pregnancy	2.29 (1.35–3.87)	1.69 (1.12–2.54)	2.20 (1.37–3.54)	1.30 (0.90–1.87)
Child lived with a smoker	2.24 (1.36–3.71)	1.57 (1.07–2.30)	2.40 (1.52–3.79)	1.44 (1.02–2.03)
Parental History of Allergy	2.40 (1.42–4.08)	1.77 (1.18–2.65)	2.19 (1.37–3.51)	1.54 (1.08–2.22)
Birthweight	2.56 (1.48–4.41)	1.72 (1.15–2.58)	2.63 (1.60–4.31)	1.32 (0.92–1.90)
Season of birth	2.29 (1.38–3.80)	1.55 (1.05–2.28)	2.47 (1.56–3.91)	1.43 (1.01–2.01)
Maternal age at enrollment	2.28 (1.37–3.78)	1.61 (1.10–2.37)	2.36 (1.49–3.73)	1.39 (0.98–1.96)
Child's gender	2.26 (1.37–3.74)	1.56 (1.07–2.29)	2.37 (1.50–3.74)	1.44 (1.02–2.03)
Adjusted for all factors but dust *	2.52 (1.32–4.82)	1.99 (1.19–3.31)	1.95 (1.09–3.47)	1.11 (0.71–1.75)
For children who had dust samples:				
Endotoxin	2.32 (1.38–3.91) n=543	1.57 (1.05–2.33) n=461	2.61 (1.61–4.24) n=550	1.46 (1.02–2.09) n=563
Dog allergen	2.21 (1.30–3.73) n=556	1.52 (1.01–2.29) n=473	2.49 (1.51–4.11) n=564	1.50 (1.04–2.16) n=578
Cat allergen	2.29 (1.33–3.93) n=557	1.67 (1.10–2.53) n=473	2.66 (1.59–4.44) n=565	1.62 (1.11–2.36) n=579
Cockroach allergen	2.33 (1.39–3.90) n=541	1.49 (1.00–2.22) n=458	2.77 (1.69–4.54) n=549	1.50 (1.05–2.14) n=563
Adjusted for all factors including dust †	2.36 (1.28–4.37) n=523	1.87 (1.14–3.08) n=443	2.27 (1.27–4.07) n=530	1.28 (0.82–1.98) n=543

* Variables are: Number of older siblings, income, education, rental/mortgage payments, pets in the home during pregnancy, ever breastfed, whether the mother was married or living as married during pregnancy, child lived with a smoker, parental history, birthweight, season of birth, maternal age at enrollment and gender.

† Variables are: Number of older siblings, income, education, rental/mortgage payments, pets in the home during pregnancy, ever breastfed, whether the mother was married or living as married during pregnancy, child lived with a smoker, parental history, birthweight, season of birth, maternal age at enrollment and gender. Models also adjusted for levels of endotoxin and dog, cat and cockroach allergen from home samples.

Table 3

The regression coefficient (beta) with 95% confidence intervals in the linear regression model for race (African American versus White) and log transformed total IgE. The crude estimate is included as are estimates adjusted for various SES and other selected factors.*

	Log transformed total IgE	% Increase in total IgE (for African American children compared to White children)
N	561	
Regression coefficient (beta) (95% CI) for Race (comparing African American with White children)	0.36 (0.11, 0.61)	43%
Regression coefficient (beta) (95% CI) for race (comparing African American with White children) adjusting for each of the following individually:		
Number of older siblings	0.36 (0.12, 0.61)	43%
Report of Household income	0.45 (0.18, 0.71)	57%
Maternal education	0.41 (0.15, 0.67)	51%
Make monthly mortgage or rental payments	0.35 (0.09, 0.60)	42%
Prenatal indoor pet(s)	0.34 (0.08, 0.60)	40%
Ever breastfed the child	0.42 (0.17, 0.67)	52%
Mother was married or living as married during pregnancy	0.42 (0.16, 0.69)	52%
Child lived with a smoker	0.37 (0.12, 0.62)	45%
Parental History of Allergy	0.37 (0.11, 0.64)	45%
Birthweight	0.40 (0.13, 0.66)	49%
Season of birth	0.33 (0.08, 0.58)	39%
Maternal age at enrollment	0.38 (0.13, 0.63)	46%
Child's Gender	0.36 (0.11, 0.61)	43%
Regression coefficient (beta) (95% CI) for Race (comparing African American with White children) adjusted for all variables except dust [†]	0.47 (0.16, 0.78)	60%
For children who had dust samples:		
Endotoxin	0.33 (0.07, 0.59) n=505	39%
Dog allergen	0.28 (0.01, 0.55) n=518	32%
Cat allergen	0.27 (0.01, 0.55) n=518	31%
Cockroach allergen	0.37 (0.11, 0.63) n=501	45%
Regression coefficient (beta) (95% CI) for Race (comparing African American with White children) adjusted for all variables including dust [‡]	0.37 (0.06, 0.68) n=485	45%

* Total IgE was log transformed to meet model assumptions. The regression coefficient (beta) should be interpreted as the ($e^{\text{beta}} - 1$) percent increase in the mean total IgE for African American children compared to White children.

[†] Variables are: Number of older siblings, income, education, rental/mortgage payments, pets in the home during pregnancy, ever breastfed, whether the mother was married or living as married during pregnancy, child lived with a smoker, parental history, birthweight, season of birth, maternal age at enrollment and gender.

[‡] Variables are: Number of older siblings, income, education, rental/mortgage payments, pets in the home during pregnancy, ever breastfed, whether the mother was married or living as married during pregnancy, child lived with a smoker, parental history, birthweight, season of birth, maternal age at enrollment and gender. Models also adjusted for levels of endotoxin and dog, cat and cockroach allergen from home samples.