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Nitrogen Dioxide and Allergic Sensitization in the 2005–2006 National Health and Nutrition Examination Survey

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Abstract

Background—Allergic sensitization is a risk factor for asthma and allergic diseases. The relationship between ambient air pollution and allergic sensitization is unclear.

Objective—To investigate the relationship between ambient air pollution and allergic sensitization in a nationally representative sample of the US population.

Methods—We linked annual average concentrations of nitrogen dioxide (NO_2), particulate matter ≤ 10 μm (PM10), particulate matter ≤ 2.5 μm (PM_{25}), and summer concentrations of ozone (O_3), to allergen-specific immunoglobulin E (IgE) data for participants in the 2005–2006 National Health and Nutrition Examination Survey (NHANES). In addition to the monitor-based air pollution estimates, we used the Community Multiscale Air Quality (CMAQ) model to increase the representation of rural participants in our sample. Logistic regression with population-based sampling weights was used to calculate adjusted prevalence odds ratios per 10 ppb increase in O_3

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and NO_2, per 10 μg/m^3 increase in PM_{10}, and per 5 μg/m^3 increase in $PM_{2.5}$ adjusting for race, gender, age, socioeconomic status, smoking, and urban/rural status.

Results—Using CMAQ data, increased levels of NO_2 were associated with positive IgE to any (OR 1.15, 95% CI 1.04, 1.27), inhalant (OR 1.17, 95% CI 1.02, 1.33), and outdoor (OR 1.16, 95% CI 1.03, 1.31) allergens. Higher $PM_{2.5}$ levels were associated with positivity to indoor allergen-specific IgE (OR 1.24, 95% CI 1.13, 1.36). Effect estimates were similar using monitored data.

Conclusions—Increased ambient NO_2 was consistently associated with increased prevalence of allergic sensitization.

Keywords

air pollution; allergic; sensitization; epidemiology; NHANES; IgE

INTRODUCTION

Both particulate and gaseous air pollutants have been hypothesized to play a role in the development and exacerbation of allergic diseases [1]. Allergic or atopic sensitization is a strong risk factor for childhood and adult asthma and is characterized by increased immunoglobulin E (IgE) production to specific antigens that can be detected by measurements in blood [2, 3].

The evidence for a link between air pollution and allergic sensitization is inconsistent. Experimental studies provide a biologic basis for gaseous and particulate air pollutants as risk factors for allergic sensitization by showing enhanced IgE production after exposure to NO_2, O_3, and particulates [4, 5, 6, 1c]. However, results from epidemiologic studies are equivocal. Positive associations between traffic-related air pollution and allergic sensitization were reported in two birth cohort studies in Germany and Sweden [7, 8]. Nine cross-sectional studies also found positive associations between ambient air pollution and allergic sensitization.9, 10, 11, 12, 13, 14, 15, 16

In contrast, four prospective birth cohort studies conducted in Europe did not find associations between air pollution and allergic sensitization [17, 18,19, 20]. Positive associations in the study by Brauer[17] were limited to sensitization to food allergens and not inhalant allergens. Several cross sectional studies also did not find associations between ambient air pollution and allergic sensitization [21, 22, 23, 24]. To date, most epidemiologic studies of air pollution and allergic sensitization have been conducted in Europe and have focused on air pollution from traffic sources. Diesel emissions represent the largest source of particulate matter from motor vehicles and have been hypothesized to be an adjuvant for allergic sensitization [25]. Diesel vehicles are a much larger percentage of the vehicle fleet in Europe than the US [26]. Recent studies of air pollution and asthma or allergies using nationally representative samples of the US population did not assess allergic sensitization. In addition, these studies relied on monitoring data alone, and as a result, have focused on study subjects mostly in major metropolitan areas [2728]. No population-based studies of air pollution and allergic sensitization representative of the US population have been conducted.

The National Health and Nutrition Examination Survey (NHANES) is a nationally representative survey of adults and children in the United States. The 2005–2006 NHANES survey included measurements of allergen-specific IgE. We linked monitored and modeled air pollution concentrations to the NHANES 2005–2006 dataset to investigate the relationship between ambient air pollution and allergic sensitization. By using an air quality model to assign exposures, we were able to increase the sample size for the investigation by including participants that did not live near air pollution monitors resulting in a sample more representative of the US population.

METHODS

We analyzed data from the NHANES 2005–2006 database. The 2005–2006 survey oversampled Mexican Americans, African Americans, ages 60 and older, adolescents 12–19, and persons with low income to increase the reliability and precision of health status indicator estimates for these groups [29,30, 31]. Our analysis was reviewed and approved by the University of North Carolina Chapel Hill Institutional Review Board. The study participants gave informed consent when they agreed to participate in the NHANES study.

Population and Study Sample

The 2005–2006 NHANES included 10,348 participants. We limited our analysis to participants ages 6 and older that were examined in the mobile exam center (MEC) (n=8086). Among the 8086 participants, 7268 had complete data for all 19 specific IgEs, 686 had no IgE data, and 132 were missing 1 or more specific IgEs. We further limited eligibility to 6917 persons with no missing values for any of the covariates used in our analysis.

Air Pollution Exposure Assignment

At the request of the National Center for Health Statistics (NCHS), the US Department of Housing and Urban Development geocoded the 2005–2006 NHANES (CDC, 2009). The NCHS linked US Environmental Protection Agency Air Quality System (AQS) monitored data and Community Multiscale Air Quality (CMAQ) model data to the 2005–2006 NHANES data by geocoded participant address [2932]. Because the data contains identifiable geographic information, it is not available for public use. We submitted a proposal to NCHS that specified our analysis plan and the variables we required from the public NHANES data file. NCHS approved our proposal and created a data set with AQS and CMAQ data linked to the NHANES data file.

We used AQS monitored data for particulate matter with aerodynamic diameter ≤ 2.5 μm (PM_{25}) and ≤ 10 μm (PM_{10}), ozone (O_3), and nitrogen dioxide (NO_2) to assign exposure estimates to participants within 20 miles (32.2 km) of a monitor. We selected annual calendar year estimates for NO_2, PM_{25}, and PM_{10}. Annual calendar year estimates were the only long term exposure option for monitored data available in NCHS data files. For example, if a participant came into the MEC on June 1, 2005, then the participant received an estimate based on concentrations averaged from 1 Jan 2005 - 31 Dec 2005.

O_3 is monitored at different times throughout the year in different locations. Average 8 hour daily maximum concentrations were calculated from 1 May through 30 September since O_3 is monitored in most locations during this period. For monitored pollutants, inverse distance weighted estimates were calculated using the inverse of the squared distance between the participant residence and monitors within 20 miles (32.2 km) of the residence. Since we included only participants within 20 miles (32.2 km) of a monitor, the sample size differs by pollutant because the location of the monitors varies by pollutant based on regulatory requirements. Of the 6917 participants with a complete panel of allergen-specific IgE and covariates, the number of participants with monitored estimates was 4331 for NO_2, 4492 for PM_{10}, 5201 for O_3, and 5298 for $PM_{2.5}$.

In addition to monitored data, we obtained CMAQ model estimates available from the EPA National Exposure Research Lab Atmospheric Modeling and Analysis Division. Estimates were available for $PM_{2.5}$, O_3 and NO_2, but not for PM_{10}. CMAQ is often used by state air pollution control agencies to assess how proposed air quality management changes might impact air pollution concentrations [33, 34]. CMAQ generates pollutant estimates by simulating the chemistry and physics of the atmosphere using air pollution emissions and meteorological data as inputs.

CMAQ output consisted of hourly surface concentrations for each day of calendar years 2004–2006 for the continental US at a resolution of 36×36 kilometers. Using CMAQ output, NCHS calculated averages for one year prior to the participant medical exam date. Participants received exams throughout the calendar year. By using CMAQ, we increased the number of participants with air pollution estimates in our study sample to 6227 for $PM_{2.5}$, NO_2, and O_3. Participants had missing air pollution concentration data because they did not live within 20 miles (32.2 km) of a monitor, lived outside the domain of the model, or they did not have sufficient address information for data linkage.

Allergic Sensitization

Survey participants ages 6 and older were tested for each of 19 allergen-specific IgE antibodies using the Pharmacia Diagnostics ImmunoCAP 1000 System. The panel included IgE to 15 aeroallergens (*Alternaria alternata*, *Aspergillus fumigates*, Bermuda grass, birch, cat dander, cockroach, dog dander, dust mite [*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*], mouse urine proteins, oak, ragweed, rat urine proteins, Russian thistle, rye grass, and 4 food allergens (egg white, cow's milk, peanut, and shrimp). The lower limit of detection was 0.35 kU/L for each specific IgE. For samples below the detection limit, NHANES reported values equal to the lower limit of detection divided by the square root of 2. The upper limit of detection was 1000 kU/L. Samples that exceeded the upper limit of detection were assigned a value of 1000 kU/L [35].

Variable Definitions

Sensitization was defined as detectable specific IgE (≥0.35 kU/L). We investigated five allergic sensitization outcome variables [36]. These included: 1) any of the IgE antibodies; 2) outdoor allergen-specific IgEs (*Alternaria alternata*, *Aspergillus fumigatus*, Bermuda grass, birch, oak, ragweed, Russian thistle, rye grass); 3) indoor allergen-specific IgEs [cat dander, cockroach, dog dander, dust mite (*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus)*, mouse proteins, rat urine proteins]; 4) inhalant (indoor or outdoor allergen-specific IgEs); and 5) food allergen-specific IgEs (egg white, cow's milk, peanut, shrimp). These five outcomes are not mutually exclusive.

We considered several covariates in our analyses. We obtained data for age, race/ethnicity, gender, and poverty income ratio based on participant responses in the survey questionnaire. Cotinine, a biomarker for smoking and secondhand smoke exposure, was obtained from the medical exam [30]. We used a dichotomous cotinine variable with a cut point of 10 ng/ml to distinguish smokers from non-smokers [37]. We used the NCHS 2005–2006 urban-rural classification scheme to characterize the degree of urbanization where a participant resided [38]. The scheme consists of four metropolitan categories and two non-metropolitan categories. We recoded the six-category NCHS urban-rural variable into five categories to preserve participant confidentiality and eliminate small cell sizes by combining the small and medium metropolitan categories into one category (Table 1). We used poverty income ratio as a surrogate for socioeconomic status. Race/ethnicity was categorized into non-Hispanic white, non-Hispanic black, Hispanic, and other.

Statistical Analyses

We calculated descriptive statistics for NHANES participants both with and without air pollution estimates (Table 2). Descriptive statistics were generated using SAS version 9.2. We used NHANES analytic and reporting guidelines to select the appropriate sub-sample weights (*wtmec2yr*) and design variables for our analysis all analyses except for descriptive statistics of pollutant concentrations since all participants received the medical exam [30].

We calculated crude and adjusted prevalence odds ratios using the SUDAAN R Logistic procedure release 10.0.1 to account for the clustering and stratification in the sample design. We used logistic regression to produce separate odds ratios for 1) monitored air pollution concentrations, 2) CMAQ estimates for participants with monitored and CMAQ estimates, and 3) all participants with CMAQ estimates. Odds ratios are scaled per 10 parts per billion for NO_2 and O_3, per 10 μg/m^3 for PM_{10} and per 5 μg/m^3 for $PM_{2.5}$. We chose scaling factors to have consistency between our modeled air pollution data and our monitored air pollution data. Based on the existing literature we selected age, gender, race/ethnicity, poverty income ratio, cotinine, and level of urbanization as covariates. We included poverty income ratio as a continuous variable and gender, race/ethnicity, age, cotinine, and level of urbanization were included as categorical variables. We also adjusted for indoor air exposures of mold, housing type, and pets; they made no difference in effect estimates, so they were not included in the final models. We conducted interaction testing for age and gender using p value < 0.10 as a criterion for positive interaction.

RESULTS

The total study sample (n=6917) was more white and less urban than the sub-samples created from linking AQS air pollution estimates to NHANES participants (Table 1). As expected, given that CMAQ data are available for subjects living in rural areas far from monitors, the subsample created from linking CMAQ estimates was more similar to the overall sample than the subsamples created from linking monitored data, thus more representative of the overall US population.

Table 2 shows the frequency of sensitization among the total sample and the sub-samples. With the exception of food allergen sensitization, the percentage of sensitization was lower in the subset of participants without linked air pollution estimates compared with the subsample of participants with air pollution estimates. Most of the participants without air pollution estimates live in rural areas. In our sample, the percentage of sensitization is lower in rural areas compared to urban areas (prevalence of sensitization to any allergen = 44.7% for rural subjects and 49.4% for urban subjects). Sensitization was also lower for all subtypes of allergens except food allergens for rural versus urban subjects (data not shown).

Descriptive air pollution statistics are shown in Tables 3 and 4. For participants that had both modeled and monitored estimates, modeled estimates of O_3 and $PM_{2.5}$ based on the year prior to the participant medical exam date were higher than the inverse distance weighted monitored calendar year estimates on average. Model estimates for NO_2 were lower than inverse distance weighted monitored estimates. NO_2 and PM_{10} were most strongly correlated (r=0.48) among monitored pollutants. In contrast, NO_2 was most strongly correlated with $PM_{2.5}$ (r=0.60) among modeled pollutants.

Table 5 displays adjusted prevalence odds ratios and 95% confidence intervals for each air pollutant in relation to each category of allergen-specific IgE based on the following: 1) monitored data, 2) CMAQ data among participants with monitored data and CMAQ data, and 3) the larger sample of all participants with CMAQ estimates. A similar table including crude and adjusted odds ratios is provided as supplemental material (Table S1). The largest percent change in crude odds ratios with adjustment for potential confounders was from the addition of urbanicity and ethnicity. Using an alternative categorization of the current smoking (with 3 categories of cotinine and cut points (< 0.015 ng/ml and < 0.050 ng/ml)) produced similar results.

The results were similar among the three analyses, but a greater number of significant associations were detected using modeled estimates than monitored estimates which might reflect the larger sample size for this analysis. The most frequent associations were observed for NO_2 with most adjusted odds ratios near 1.2. After adjustment for confounders, the only significant association identified using modeled data that was not identified using monitored data was for NO_2 in relation to indoor allergen-specific IgE. Similar effect estimates from CMAQ and monitored data of the same participants provide some confidence that odds ratios produced from our larger CMAQ sample (n=6277) that includes subjects without monitoring data provide reasonable effect estimates in the absence of monitored data. Testing for interaction for age or gender indicated very little evidence of effect modification. Age-stratified analyses with interaction P values are provided as supplemental material (Table S2 and Figure S1. Forest Plot of Age-stratified Analyses). For gender, the relationship between $PM_{2.5}$ and outdoor air pollution was the only relationship with an interaction p value < 0.10.

DISCUSSION

We found associations between increased NO_2 and $PM_{2.5}$ concentrations and allergic sensitization in the US population. NO_2 exposure was significantly associated with three allergic sensitization categories using CMAQ data. Overall, we found similar results using monitored data but with fewer statistically significant results in this smaller subset of the data. $PM_{2.5}$ was consistently associated with sensitization to indoor allergens. This is the first population-based study of air pollution and allergic sensitization that used a nationally representative sample of the US population.

Most previous studies that have identified associations between ambient air pollution and allergic sensitization were studies of traffic-related air pollution in Europe. In contrast, our study was not designed to specifically assess traffic-related air pollution since our exposure metrics do not differentiate near roadway exposures. Additionally, our sample contains both children and adults, whereas most other studies have assessed either children or adults.

Our findings are plausible based on several recent mechanistic studies in mice that provide support for NO_2 exposure as a contributor to the development of allergic sensitization. Ckless [39] provided evidence that NO_2 contributes to allergic sensitization as an exogenous reactive nitrative species and contributes to the production of endogenous reactive oxidative and reactive nitrative species [4039].

Consistent with several recent studies, our most frequent associations involve NO_2. In a Swedish birth cohort, Nordling[8] found an association between traffic-related NO_2 and sensitization to pollens (OR =1.67 95% CI 1.10, 2.53 per 44 μg/m^3, n=2543) at age 4 years. Similarly Kramer [11] identified an association (OR=4.96 95% CI 1.56, 15.74 per 10 μg/m^3) between ambient NO_2 and sensitization to pollens for children 9 years of age residing in urban areas. This cross-sectional study of 317 German children lost significance (OR=1.05 95% CI 0.70, 1.56) when urban and suburban children were analyzed together. In addition, a cross sectional study by Janssen [10] also found a positive association between NO_2 and sensitization to inhalant allergens (OR=1.70 95% CI 1.03, 2.81 per 17.6 μg/m^3) among 1114 Dutch children 7–12 years of age. Overall our effect estimates are generally smaller than reported in these studies. This may be in part because our exposure assessment approach could not resolve within city exposure contrasts or near roadway exposures. Possible reasons for the differences in association are that our sample included children and adults, was larger than the samples of the studies that reported positive associations, and used a different scaling factor. Also, the allergens included in the definition of sensitization are not consistent across studies. In contrast to our findings, several epidemiologic studies did not find positive associations between NO_2 and sensitization to inhalant allergens. Three of these were birth cohort studies [171820] while six were cross sectional studies [9, 22, 23, 19, 41, 24].

Across studies, there are differences in methods of exposure assessment, differences between the interpretation of skin tests and laboratory variability in assays of specific IgE to assess allergic sensitization, as well as differences in ambient pollutant levels that may all contribute to variation in the associations observed [42, 43]. The combination of these factors makes comparisons difficult. For studies where an association was detected, no one pollutant appeared to be most frequently associated with allergic sensitization. This observation raises a question regarding whether our findings for NO_2 represents a pollutant specific finding or if NO_2 is a surrogate for traffic-related pollutants.

Two previous studies reported positive associations between $PM_{2.5}$ and allergic sensitization. A cohort study by Morgenstern [7] found an association between $PM_{2.5}$ and sensitization to inhalant allergens (OR=1.45 95% CI 1.21, 1.74 per 1.5 μg/m^3) but was largely driven by sensitization to outdoor allergens. A cross sectional study by Annesi-Maesano [9] found a positive association between $PM_{2.5}$ and sensitization to indoor allergens (OR=1.29 95% CI 1.11, 1.50 for high versus low pollutant exposure. Low pollutant exposure ranged from 1.6–12.2 μg/m^3. Our finding of an association of $PM_{2.5}$ with IgE of indoor allergens (OR=1.24 95% CI 1.13, 1.36 per 5 μg/m^3) was similar in magnitude to Annesi-Maesano [9]. Our findings were driven largely by dust mite (data not shown), which is the most common antigen to which subjects in this category are sensitized [44]. Other studies identified included four studies that were not consistent with our results for $PM_{2.5}$: two birth cohort studies [1718] and two cross sectional studies [2110].

Our study has several strengths. The NHANES study population is representative of the entire US population. We believe this is particularly important since most studies of air pollution and allergic sensitization have been conducted in Europe, which may have different pollutant mixtures and allergen species. In addition, the study is relatively large and the assessment of sensitization is comprehensive, based on 19 specific allergen IgEs. We also included data on a number of potential confounders, including cotinine to objectively assess smoking and exposure to environmental tobacco smoke.

Another important strength of the current analysis is that we used an air quality model as an alternate method of assigning air pollution exposures to increase inclusion of participants living outside of major metropolitan areas. We found consistent associations using both monitored and modeled air pollution estimates. To our knowledge, this is the first time that both monitoring and air quality modeling exposure assignment methods have been used in an epidemiologic study to assess the US population. Using an air quality model provides air pollution estimates that capture the nonlinear atmospheric chemistry and physics of the atmosphere that linear interpolation methods cannot [33]. Finally, the general concordance of the results using both exposure assignment approaches adds strength to the validity of our findings.

The study has limitations. We adjusted for a number of potential confounders, but we cannot completely rule out unmeasured factors that might be spatially associated with air pollution that biased our effect estimates. Arbes [45] found that the prevalence of atopy differed by census region within the US. We were not able to conduct geographic level stratified analyses with our data. Our primary method of estimating air pollution concentrations relied on US EPA criteria pollutant monitoring. We did not have information on distance to roadway or traffic density to estimate near roadway exposures. The monitoring network has limited coverage of rural areas. However, we used CMAQ to increase the spatial coverage of air pollutant estimates for rural participants. Although we increased the number of rural participants CMAQ has limitations inherent to simulating air pollution concentrations.

Meteorological data, emissions data, and the chemical and physical processes that CMAQ is simulating all introduce uncertainty into estimates of air pollution concentration [33].

Both of our exposure metrics are relatively coarse. We limited our investigation to participants within 20 miles (32.2 km) of a monitor and used a 36 kilometer grid to generate modeled estimates of ambient concentration. Because NHANES is a national sample, we were primarily concerned with exposure contrasts between areas and not within an area. Despite our exposure assignment approach being limited by not being able to capture within area exposure contrasts, we still detected positive associations between air pollution and allergic sensitization. Since our study was aimed at looking at differences between areas, and not within an area, we believe that our estimates of air pollution concentration are suitable for estimating associations under these conditions. Two key factors in how well ambient monitors estimate personal exposure are how close participants are to monitors and how homogeneous the pollutant concentrations are in space. The degree of pollutant spatial homogeneity varies across the study areas selected by NHANES based on the inventory of sources, topography, type of pollutant, atmospheric conditions, locations of monitors relative to study participants, model performance, and size of the area [46]. Ambient concentrations of O_3 and $PM_{2.5}$ are relatively homogeneous over short distances compared to $NO_{2.}$ NO_2 concentrations vary more over short distances as a result of traffic sources. Fourteen of the seventeen studies epidemiologic studies of air pollution and allergic sensitization we referenced estimated exposures from traffic or captured variability in air pollution concentration within an urban area. Since our study cannot, we may miss areas of highest concentration within an urban area that may have attenuated our effect estimates.

We chose to base our estimate of monitored pollutant levels on monitors within 20 miles (32.2 km), based on the work of Parker [47] who linked air pollution estimates for NHIS participants based on an average of 1) all monitors within the county, 2) monitors within a 5 mile radius of the participant census block group, and 3) monitors within 20 miles (32.2 km) of the participant census block group. Parker [47] suggested that these methods gave similar association results but have tradeoffs. Linking air pollution estimates to national survey data sets with finer spatial resolution reduces measurement error but also reduces sample size. On the other hand using air pollution estimates with coarser spatial resolution increases the likelihood of measurement error, increases sample size, and reduces the potential for selection bias.

In summary, our study suggests that ambient air pollution is associated with allergic sensitization. Our main finding of an association with NO_2 and allergic sensitization is seen for both monitored and modeled data and across several categories of allergen-specific IgE. Our study is the first to assess the relationship between air pollution and allergic sensitization in a nationally representative sample of the US population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AQS	Air Quality System
CI	confidence interval
CMAQ	Community Multiscale Air Quality
IgE	Immunogloblin E
MEC	mobile examination center
NCHS	National Center for Health Statistics
NO_2	nitrogen dioxide
NHANES	National Health and Nutrition Examination Survey
$PM_{2.5}$	Particulate matter with aerodynamic diameter ≤ 2.5 μm
PM_{10}	Particulate matter with aerodynamic diameter ≤ 10 μm

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Table 1

Characteristics of the total sample and subsamples with pollutant data[a]

	Total	Monitored Data[b]				CMAQ Data[c]
	n=6917	NO_2 n=4331	O_3 n=5201	PM_{10} n=4492	$PM_{2.5}$ n=5298	n=6227
Race/Ethnicity (%)						
Non Hispanic White	70.4	60.9	65.9	60.9	66.7	68.9
Non Hispanic Black	11.7	14.4	12.9	14.7	12.5	13.0
Mexican American	8.7	13.4	11.0	12.8	10.2	9.1
Other	9.2	11.3	10.2	11.6	10.5	9.1
Age (%)						
6 – 17	17.2	17.4	17.4	17.3	17.5	17.0
≥18	82.8	82.6	82.6	82.7	82.5	83.0
Cotinine (%)						
< 10 ng/ml	75.8	76.8	76.8	77.4	77.3	75.7
≥ 10 ng/ml	24.2	23.2	23.2	22.6	22.7	24.3
Gender						
% Female	51.4	52.3	51.8	52.3	51.8	51.5
Urbanicity (%)						
Large Metropolitan	31.3	53.8	42.9	52.7	41.0	34.9
Large Fringe Metropolitan	18.6	28.5	24.5	25.8	22.2	19.0
Small and Medium Metro	28.7	17.7	21.2	21.7	26.0	23.8
Micropolitan	15.5	0	10.4	0	9.8	15.8
Noncore	6.0	0	0.9	0	0.9	6.5
PIR (mean, Std deviation)	2.5(0.02)	2.5(0.02)	2.5(0.02)	2.5(0.02)	2.5(0.02	) 2.5(0.02)

Abbreviation: PIR=poverty income ratio, CMAQ=Community Multiscale Air Quality Model

[a]All percentages were weighted using NHANES survey weights

[b]Subsamples for monitored NO_2, O_3, $PM_{2.5}$, and PM_{10} linked to NHANES participants

[c]Subsamples for monitored NO_2, O_3, $PM_{2.5}$, and PM_{10} from the CMAQ model linked to NHANES participants

Table 2

Weighted prevalence of sensitization for participants with and without air pollution data[a]

		Monitored Data[b]								CMAQ[c]	
	Total n=6917	NO_2 With n=4331	Without n=2586	O_3 With n=5201	Without n=1716	$PM_{2.5}$ With n=5298	Without n=1619	PM_{10} With n=4492	Without n=2425	With n=6227	Without n=690
Sensitization (%)											
Any[d]	44.8	48.0	41.1	46.2	41.6	45.6	42.7	47.6	41.2	45.3	41.6
Inhalant[e]	42.7	45.9	38.9	43.9	39.8	43.5	40.6	45.5	38.9*	43.1	39.6
Outdoor[f]	30.1	34.4	24.9	32.0	25.7	31.8	25.7	34.4*	24.5*	30.7	26.2
Indoor[g]	30.4	31.1	29.6	30.6	30.0	30.3	30.7	30.7	30.0	30.6	29.3
Food[h]	6.5	5.8	7.3	6.1	7.4	5.9	8.2	5.5	7.8*	6.4	7.4

Abbreviations: CMAQ=Community Multiscale Air Quality Model

*Prevalence differences between with and without are significant at $p<.05$

[a]All percentages were weighted using NHANES survey weights

[b]Subsamples for monitored $PM_{2.5}$, O_3, NO_2, and PM_{10} linked to NHANES participants

[c]Subsamples for $PM_{2.5}$, O_3, and NO_2 linked to NHANES participants based on CMAQ data

[d]Any=Detectable specific IgE to indoor, outdoor, or food allergens

[e]Inhalant= Detectable specific IgE to indoor or outdoor allergens

[f]Outdoor= Detectable specific IgE to *Alternaria alternata, Asperigillus fumigatus*, Bermuda grass, birch, oak, ragweed, Russian thistle, or rye grass

[g]Indoor= Detectable specific IgE to cat dander, cockroach, dog dander, dust mite (*Dermataphagoides farinae* and *Dermatophagoides pteronyssinus*), mouse proteins, rat urine proteins

[h]Food= Detectable specific IgE to egg white, milk, peanut or shrimp

Table 3

Monitored and CMAQ Pollutant Concentrations

Measure	Monitored				CMAQ		
	NO_2 (ppb)	**O_3 (ppb)**	**$PM_{2.5}$ (ug/m^3)**	**PM_{10} (ug/m^3)**	**NO_2 (ppb)**	**O_3 (ppb)**	**$PM_{2.5}$ (ug/m^3)**
5th Percentile	11.4	37.5	8.7	19.4	2.0	45.6	6.9
10th Percentile	11.7	40.4	9.5	20.6	2.5	47.6	7.8
25th Percentile	13.2	48.1	11.4	23.6	3.7	52.4	9.5
Median	17.6	51.5	12.7	27.1	10.6	57.0	13.4
Mean	18.6	52.0	12.7	28	11.6	57.2	12.6
75th Percentile	24.3	55.3	13.9	30.9	15.3	61.2	15.1
95th Percentile	27.0	60.3	16.5	44.1	27.6	70.8	20.0

CMAQ PM10 air pollution concentrations were not available in our data set

Abbreviations: CMAQ=Community Multiscale Air Quality Model

Table 4

Pearson Correlations of Monitored and CMAQ Pollutant Concentrations

	Monitored				CMAQ		
	NO_2 (ppb)	**O_3 (ppb)**	**$PM_{2.5}$ (ug/m^3)**	**PM_{10} (ug/m^3)**	**NO_2 (ppb)**	**O_3 (ppb)**	**$PM_{2.5}$ (ug/m^3)**
Correlation							
NO_2	1	−0.25	−0.01	0.48	0.57	−0.38	−0.1
O_3	−0.25	1	0.08	0.4	−0.22	0.66	−0.29
$PM_{2.5}$	−0.01	0.08	1	0.11	0.45	−0.21	0.57
PM_{10}	0.48	0.4	0.11	1	0.32	0.09	−0.2
CMAQ NO_2	0.57	−0.22	0.45	0.32	1	−0.42	0.48
CMAQ O_3	−0.01	0.66	−0.09	0.09	−0.42	1	−0.21
CMAQ $PM_{2.5}$	−0.1	−0.29	0.57	−0.2	0.48	−0.21	1

Abbreviations: CMAQ=Community Multiscale Air Quality Model

Table 5

Adjusted odds ratios and 95% CIs between air pollution concentrations and allergen-specific IgE for ages ≥ 6[a,b]

Sensitization		Results Based on Monitored Data		CMAQ Results for Subjects with Monitored Data			CMAQ Results Including Subjects without Monitored Data		
Pollutant	n	OR	(95% CI)	**n**	OR	(95% CI)	**n**	OR	(95% CI)
Any[c]									
NO_2	4331	1.24	(1.07, 1.44)	4331	1.13	(1.02, 1.26)	6277	1.15	(1.04, 1.27)
O_3	5201	1.07	(0.94, 1.21)	5151	1.09	(0.91, 1.31)	6277	1.10	(0.93, 1.29)
$PM_{2.5}$	5298	1.13	(0.94, 1.36)	5208	1.02	(0.90, 1.17)	6277	1.04	(0.93, 1.17)
PM_{10}	4492	1.08	(0.95, 1.25)						
Inhalant[d]									
NO_2	4331	1.23	(1.04, 1.46)	4331	1.15	(0.99, 1.34)	6277	1.17	(1.02, 1.33)
O_3	5201	1.06	(0.93, 1.20)	5151	1.09	(0.90, 1.32)	6277	1.11	(0.93, 1.32)
$PM_{2.5}$	5298	1.11	(0.93, 1.32)	5208	1.05	(0.88, 1.24)	6277	1.07	(0.93, 1.24)
PM_{10}	4492	1.07	(0.93, 1.23)						
Outdoor[e]									
NO_2	4331	1.24	(1.00, 1.55)	4331	1.03	(0.83, 1.29)	6277	1.10	(0.89, 1.35)
O_3	5201	1.17	(0.99, 1.38)	5151	1.12	(0.88, 1.42)	6277	1.14	(0.90, 1.43)
$PM_{2.5}$	5298	0.84	(0.62, 1.15)	5208	0.88	(0.68, 1.13)	6277	0.93	(0.74, 1.17)
PM_{10}	4492	1.18	(0.98, 1.42)						
Indoor[f]									
NO_2	4331	1.14	(0.97, 1.35)	4331	1.20	(1.09, 1.33)	6277	1.16	(1.03, 1.31)
O_3	5201	0.91	(0.78, 1.06)	5151	1.03	(0.86, 1.23)	6277	1.02	(0.86, 1.22)
$PM_{2.5}$	5298	1.27	(1.12, 1.45)	5208	1.26	(1.16, 1.38)	6277	1.24	(1.13, 1.36)
PM_{10}	4492	0.93	(0.85, 1.03)						
Food[g]									
NO_2	4331	1.10	(0.77, 1.55)	4331	1.18	(0.91, 1.55)	6277	1.08	(0.82, 1.44)
O_3	5201	0.80	(0.54, 1.19)	5151	1.07	(0.76, 1.51)	6277	1.01	(0.77, 1.32)

		Results Based on Monitored Data		CMAQ Results for Subjects with Monitored Data			CMAQ Results Including Subjects without Monitored Data		
$PM_{2.5}$	5298	1.27	(0.78, 2.08)	5208	1.22	(0.89, 1.69)	6277	1.09	(0.83, 1.44)
PM_{10}	4492	0.78	(0.53, 1.14)						

Abbreviations: OR – odds ratio, CI – confidence interval, CMAQ – Community Multiscale Air Quality Model

[a]Odds ratios are per 10 ppb for NO_2 and O_3, per 5 μg/m for $PM_{2.5}$ and 10 μg/m for PM_{10}

[b]Adjusted for age, ethnicity, poverty income ratio, gender, cotinine, urban/rural status

[c]Any= Detectable specific IgE to indoor, outdoor, or food allergens

[d]Inhalant= Detectable specific IgE to indoor or outdoor allergens

[e]Outdoor= Detectable specific IgE to *Alternaria alternata*, *Aspergillus fumigatus*, Bermuda grass, birch, oak, ragweed, Russian thistle, or rye grass

[f]Indoor= Detectable specific IgE to cat dander, cockroach, dog dander, dust mite (*Dermatophagoides farina and Dematophagoides pteronyssinus*), mouse proteins, rat urine proteins

[g]Food = Detectable specific IgE to egg white, milk, peanut, or shrimp