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Nitrogen dioxide and allergic sensitization in the 2005–2006 National Health and Nutrition Examination Survey



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KEYWORDS Air pollution;	Summary Background: Allergic sensitization is a risk factor for asthma and allergic diseases. The rela-
Allergic;	tionship between ambient air pollution and allergic sensitization is unclear.
Sensitization;	Objective: To investigate the relationship between ambient air pollution and allergic sensiti-
Epidemiology;	zation in a nationally representative sample of the US population.
NHANES;	Methods: We linked annual average concentrations of nitrogen dioxide (NO ₂), particulate mat-
IgE	ter ${\leq}10~\mu\text{m}$ (PM_{10}), particulate matter ${\leq}2.5~\mu\text{m}$ (PM_{2.5}), and summer concentrations of ozone
	(O_3) , to allergen-specific immunoglobulin E (IgE) data for participants in the 2005-2006 Na-
	tional Health and Nutrition Examination Survey (NHANES). In addition to the monitor-based

* Corresponding author. Tel.: +1 (919) 966 9899; fax: +1 (919) 966 2089. *E-mail address:* Karin_Yeatts@unc.edu (K.B. Yeatts).

0954-6111/\$ - see front matter © 2013 Elsevier Ltd. All rights reserved. http://dx.doi.org/10.1016/j.rmed.2013.08.010 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 O₃ and NO₂, per 10 μ g/m³ increase in PM₁₀, and per 5 μ g/m³ increase in PM_{2.5} adjusting for race, gender, age, socioeconomic status, smoking, and urban/rural status.

Results: Using CMAQ data, increased levels of NO₂ 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 indoor (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.

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Abbreviations

AQS	Air Quality System
CI	confidence interval
CMAQ	Community Multiscale Air Quality
lgE	immunoglobulin E
MEC	Mobile Examination Center
NCHS	National Center for Health Statistics
NO ₂	nitrogen dioxide
NHANESNational Health and Nutrition	
	Examination Survey
PM _{2.5}	particulate matter with aerodynamic
	diameter \leq 2.5 μ m
PM ₁₀	particulate matter with aerodynamic
	diameter \leq 10 μ m

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 [9–16].

In contrast, four prospective birth cohort studies conducted in Europe did not find associations between air pollution and allergic sensitization [17-20]. 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 [21-24]. 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 [27,28]. 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 data set 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 [29–31]. Our analysis was reviewed and approved by the University of North Carolina Chapel Hill Institutional

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