

phisms in a way that adversely affects lung function and hyperresponsiveness.

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Indoor Particulate Matter Increases Asthma Morbidity in Children With Non-Atopic and Atopic Asthma

McCormack MC, Breyse PN, Matsui EC, et al; Center for Childhood Asthma in the Urban Environment.

Ann Allergy Asthma Immunol. 2011;106(4):308-315

PURPOSE OF THE STUDY. Environmental control is an accepted component of asthma management in children with atopic asthma, but it is not usually a part of management in nonatopic asthma. Air pollutants, particularly particulate matter, might have a stronger effect on nonatopic asthma and might have significant indoor sources. This study examined the effect of indoor particulate matter in children with asthma.

STUDY POPULATION. Studied were 150 predominantly black children from the east Baltimore, Maryland, area aged 2 to 6 years with physician-diagnosed asthma and symptoms or medication use in the previous 6 months. Most of the children were from lower-income households.

METHODS. Integrated air sampling in the child's bedroom was performed over 3 days at baseline, 3 months, and 6 months, using PM₁₀ (particulate matter that is <10 μm in diameter) and PM_{2.5} (particulate matter that is <2.5 μm in diameter) samples collected with personal environmental monitors. Ambient particulate matter for the study was monitored at a central site within the study area. Each child underwent baseline skin testing to a mix of 14 aeroallergens. Atopy was defined as at least 1 positive skin-test result. At baseline, 3 months, and 6 months, caregivers completed questionnaires adapted from the International Study of Asthma and Allergies in Childhood and the Children's Health Survey for Asthma Questions. Participants completed a daily activity diary during each 3-day monitoring period, including an account of the time spent in the room where monitoring was performed.

RESULTS. Subjects were classified as nonatopic (31%) or atopic (69%). Nonatopic children were slightly younger. Indoor PM_{2.5-10} concentrations were similar in atopic and nonatopic children's homes, although PM_{2.5} exposure was significantly higher in the homes of children with nonatopic asthma ($P = .04$). Concentrations of PM_{2.5} exceeded Environmental Protection Agency standards in 75% of the homes. There were statistically significant interactions found between both coarse and fine particulate matter levels and asthma symptoms in both atopic and nonatopic asthmatic children.

CONCLUSIONS. In-home particle concentrations are associated with asthma morbidity, including symptoms and use of rescue medications, among atopic and nonatopic children with asthma. Strategies for reducing and eliminating sources of indoor particulate matter pollution should be considered a priority in the management of nonatopic asthma.

REVIEWER COMMENTS. This study is one of few to note that the effect of indoor air pollution is at least as important in nonatopic children with asthma. As clinicians, we often discuss secondhand smoke, which is a component of indoor particulate matter, but we also should consider other sources including cooking and cleaning products.

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Microsomal Epoxide Hydroxylase Genotypes/ Diplotypes, Traffic Air Pollution, and Childhood Asthma

Tung KY, Tsai CH, Lee YL. *Chest.* 2011;139(4):839-848

PURPOSE OF THE STUDY. The gene that encodes microsomal epoxide hydroxylase, (*EPHX1*), is responsible for detoxification of reactive epoxides to generate reactive oxygen species. The different polymorphisms influence *EPHX1* activity. The associations of *EPHX1* Tyr113His and His139Arg genotypes and diplotypes with asthma and wheezing outcomes were examined with a focus on the functional genetic change in glutathione S-transferase m1 (*GSTM1*) genotypes.

STUDY POPULATION. The study included 3741 7th-grade schoolchildren from 14 communities enrolled in the Taiwan Children Health Study.

METHODS. Asthma and wheeze status was determined by a baseline questionnaire. Children were classified as having lifetime asthma (physician-diagnosed asthma) or early-onset asthma (onset at <5 years old). Air pollution data (average hourly NO₂ level) were available from monitoring stations for the Taiwan Environmental Protection Agency. DNA was collected from oral mucosa, and genomic DNA was isolated.

RESULTS. Having the *EPHX1* Arg/His or Arg/Arg genotypes at codon 139 was significantly associated with increased risks of lifetime asthma (adjusted odds ratio [aOR]: 1.3 [95% confidence interval (CI): 1.1-1.7] and 1.5 [95% CI: 1.1-2.1], respectively). The *EPHX1* diplotypes showed significant associations with lifetime asthma (global P value = .01) and early-onset asthma (global P value = .01). The risk of *EPHX1* 139Arg allele and 113Tyr139Arg diplotype was of greater magnitude in higher-NO₂ compared with lower-NO₂ communities.

The increase of the effect from the *EPHX1* 139Arg allele with higher NO₂ exposure was most marked in the GSTP1 Val allele and GSTM1-present genotype.

CONCLUSIONS. Children with high *EPHX1* activity have an increased risk of asthma and wheezing outcomes. The risk is higher with high NO₂ exposure and a GSTP1 105Val allele or GSTM1-present genotype, which suggests that these common genetic polymorphisms and diplotypes play important roles in asthma pathogenesis among children, depending on airway oxidative stress.

REVIEWER COMMENTS. This article, although technical, sheds light on the scientific background of a basic premise in asthma: the association of air pollution on asthma risk. The results of previous studies have suggested that exposure to air pollution carries an increased risk of asthma. This study examined the genetic basis of this principle with a focus on the epoxide hydroxylase enzyme activity. An increased risk of asthma was seen in children with certain genotypes, and the risk was of higher magnitude depending on environmental NO₂ levels. These results add to the complex pathogenesis of asthma in regards to both genetic and environmental influences.

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FOOD ALLERGY

National Prevalence and Risk Factors for Food Allergy and Relationship to Asthma: Results From the National Health and Nutrition Examination Survey 2005–2006

Liu AH, Jaramillo R, Sicherer SH, et al. *J Allergy Clin Immunol.* 2010;126(4):798–806

PURPOSE OF THE STUDY. To investigate the prevalence and demographic risk factors of food allergy (FA) and its association with other atopic diseases in a population sample.

STUDY POPULATION. Data were collected from 10 348 adult and children older than 1 year, who represented the national population from 30 sites across the continental United States. Blood was collected and specific immunoglobulin E (IgE) panels were run for 79.3% of the subjects.

METHODS. Specific IgE levels to peanut, cow's milk, egg white, and shrimp were collected from subjects aged 6 years and older. Shrimp-specific IgE was not tested for subjects younger than 6 years. Food sensitization was defined as having at least 1 food-specific serum IgE level at ≥ 0.35 kU/L. FA risk categories included unlikely FA

(between ≥ 0.35 and 2 kU/L), likely FA (egg white: ≥ 7 kU/L, or ≥ 2 kU/L if ≤ 2 years old; milk: ≥ 15 kU/L, or ≥ 5 kU/L if ≤ 2 years old; peanut: ≥ 14 kU/L; and shrimp: ≥ 5 kU/L), and possible FA (between 2 kU/L and the likely FA threshold level for each food). Clinical FA rates were based on the sum of 50% of possible FA and 95% of likely FA.

RESULTS. Overall food sensitization was 16.8%. Milk and egg sensitization were highest (22% and 13.9%, respectively) in children aged 1 to 5 years. Peanut sensitization was highest in older children aged 6 to 19 years (10.7%) and young adults aged 20 to 39 years (8.7%). Shrimp sensitization did not vary with age. Overall prevalence of multiple sensitizations was 4.7%. The overall estimated clinical FA rate was 2.5% ([3.1% possible FA \times 0.5] + 1.0% likely FA). The highest prevalence of clinical FA was in children aged 1 to 5 years (4.2%) and lowest in adults aged 60 years or older (1.3%). Clinical FA was 1.8% in children aged 1 to 5 years for milk, egg, and peanut. Peanut (2.7%) was the most common clinical FA in older children aged 6 to 19 years. Peanut and shrimp (range: 0.9%–1.2%) were the most common clinical FA in adults aged 20 to 59, and shrimp (0.7%) was the most common clinical FA in adults aged 60 years or older. Overall prevalence of multiple clinical FA was 1.3%. Clinical FA was more prevalent in younger subjects ($P < .001$), male subjects ($P < .001$), and non-Hispanic black subjects ($P < .001$). Household income and education level were not significantly associated with clinical FA. Subjects with doctor-diagnosed asthma were at a higher risk for likely FA; this risk increased with increased asthma persistence and severity and with an emergency department visit for asthma in the previous year. The odds of doctor-diagnosed hay fever were increased for those with possible FA. Eczema was not significantly increased for any FA risk group.

CONCLUSIONS. The estimated population prevalence of clinical FA was 2.5% and was associated with childhood, male gender, and non-Hispanic black race/ethnicity. Asthma and emergency department visits for asthma were associated with likely FA.

REVIEWER COMMENTS. This is an important study that investigated the prevalence of clinical FA in children and adults in the same large population sample; it confirmed early observations of association of FA with childhood, non-Hispanic black race, and asthma. Use of objective data eliminated some limitations of previous survey studies. However, these data most likely underestimate clinical FA, because the study only accounted for 4 common allergenic foods, no clinical history is included (a small but significant percentage of patients with undetectable specific IgE might have clinical FA), and confirmation with oral food challenges was not performed. The next step will be to expand the number of foods

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