

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.

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