predict disease severity in asthma. These results indicate that for most indoor allergens, allergen-specific IgE levels might be a marker of allergen exposure and disease burden.

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TOBACCO AND AIR POLLUTION

Smoke-Free Air Laws and Asthma Prevalence, Symptoms, and Severity Among Nonsmoking Youth

Dove MS, Dockery DW, Connolly GN. *Pediatrics*. 2011; 127(1):102–109

PURPOSE OF THE STUDY. To investigate the relationship between smoke-free laws and asthma prevalence, symptoms, and severity among nonsmoking children aged 3 to 15 years.

STUDY POPULATION. The data were obtained from the National Health and Nutrition Examination Survey 1999–2006 (NHANES).

METHODS. Survey sites were designated as having or not having at least 1 smoke-free work location, restaurant, or bar law at the county or state level that encompassed the entire county population. Asthma prevalence was assessed as self-reported current asthma and as ever having asthma with current symptoms. Asthmatic symptoms included persistent wheeze, chronic night cough, and wheeze-medication use. The authors also examined asthma severity defined by asthma episode or emergency department visit for asthma.

RESULTS. Smoke-free laws were significantly related with lower odds of asthma symptoms (odds ratio [OR]: 0.67 [95% confidence interval (CI): 0.48–0.93]) among nonsmoking youth. The relationship between smoke-free laws and ever having asthma with current symptoms trended to significance (OR: 0.74 [95% CI: 0.53–1.03]). Smoke-free laws were associated with lower odds of asthma episodes (OR: 0.66 [95% CI: 0.28–1.56]) and emergency department visits for asthma (OR: 0.55 [95% CI: 0.27–1.13]), but these outcomes were not statistically significant.

CONCLUSIONS. Smoke-free laws decrease asthma symptoms, including persistent wheeze, chronic nocturnal cough, and wheeze-medication use in youthful nonsmoking populations.

REVIEWER COMMENTS. This study was limited by the county-limited definition of smoke-free laws, which is only an estimate of individual exposure to secondhand tobacco smoke outside the home. Misclassification of county smoke-free laws might not reflect individual exposure, and misclassification of current asthma is possible because self-reports were not validated by objective measures or clinical assessment. However, the findings of this study are consistent with those of other studies of secondhand smoke. In summary, the take-home message and conclusion of this important study is that smoke-free laws are associated with decreased exposure to secondhand smoke but equally with decreased respiratory symptoms as well.

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A Strong Synergism of Low Birth Weight and Prenatal Smoking on Asthma in Schoolchildren

Bjerg A, Hedman L, Perzanowski M, Lundbäck B, Rönmark E. *Pediatrics*. 2011;127(4). Available at: www.pediatrics.org/cgi/content/full/127/4/e905

PURPOSE OF THE STUDY. To study the independent and joint effects of prenatal smoking and low birth weight (LBW) on childhood asthma.

STUDY POPULATION. The study included asthmatic 11- to 12-year-old children in Sweden ($N = 3389$).

METHODS. Children were studied by questionnaire survey as part of the International Study of Asthma and Allergy in Childhood (ISAAC). A subset of 2121 children also underwent skin-prick testing.

RESULTS. Mean birth weight was 3360 g in children exposed to prenatal smoking and 3571 g in nonexposed children ($P < .001$). The association of prenatal smoking with physician-diagnosed asthma was stronger in LBW children (risk ratio: 8.8 [95% confidence interval: 2.1–38]) than in normal birth weight children (risk ratio: 1.3 [95% confidence interval: 1.0–1.8]). LBW alone was not an independent predictor of asthma.

CONCLUSIONS. There is a strong interaction of LBW and prenatal smoking on the risk of physician-diagnosed asthma, which is observed even after adjusting for known risk factors including allergic sensitization.

REVIEWER COMMENTS. This report highlights the observation that the combination of LBW and prenatal smoking increases the risk of physician-diagnosed asthma sixfold versus either LBW (no effect) or prenatal smoking (weak effect) alone. The authors speculated that smoke-induced oxidative stress in underdeveloped airways (caused by impaired fetal growth) might lead to increased asthma risk. In this regard, it has been shown that smoke exposure interacts with *ADAM33* polymor-

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, Breysse 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 PM_{10} (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 NO_2 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-NO_2 compared with lower-NO_2 communities.

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Frank S. Virant

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