Exposures to Molds in School Classrooms of Children With Asthma


PURPOSE OF THE STUDY. The goal of this study was to examine the diversity and concentrations of molds in inner-city schools and to describe differences between classrooms within the same school.

STUDY POPULATION. Four hundred students with physician-diagnosed asthma attending inner-city elementary schools are currently being recruited from screening surveys collected during the spring and phenotypically characterized at baseline in the summer in an ongoing longitudinal study (SICAS [School Inner-City Asthma Study]). The primary purpose of the study is to evaluate the role of indoor allergens specific to the inner-city classroom environment and asthma morbidity.

METHODS. Classroom airborne mold spores, collected over a 2-day period, were measured twice during the school year by using direct microscopy. Only classrooms of asthmatic children who were part of this study were sampled.

RESULTS. There were 180 classroom air samples collected from 12 schools. Mold was present in all classrooms analyzed, and there was a high degree of variability in the quantity and diversity of molds between classrooms, even within the same school. The classroom accounted for the majority of variance (62%) in the total mold count and for the majority of variance (56%) for the mold diversity score versus the school. The most prevalent spores were Cladosporium, basidiospores, Penicillium/Aspergillus, and smut spores, which are species generally known to cause symptoms in sensitized individuals. The study also found that visualized mildew was a predictor of increased mold spore levels, which was more commonly reported in classrooms compared with homes. Finally, there was a seasonal relationship as mold spore concentrations were higher earlier in the academic school year during the fall.

CONCLUSIONS. This study found that the school is a source of mold exposure, but the classroom microenvironment particularly varies in quantity of spores and species among classrooms within the same school. In addition, the study also confirms that the presence of visible mold may be a predictor for high mold spore counts.

REVIEWER COMMENTS. Many studies have suggested that mold exposure is associated with asthma development and morbidity, but most studies on mold have focused on home environments. This study demonstrates the role of classrooms and schools as being an important source of mold allergen exposure, especially because children spend the majority of their day in schools/classrooms when not at home. Further studies are needed to determine the clinical significance of mold exposure relative to asthma morbidity in sensitized and nonsensitized asthmatic children.

Indoor Pollutant Exposures Modify the Effect of Airborne Endotoxin on Asthma in Urban Children


PURPOSE OF THE STUDY. This study aimed to examine the effects of airborne endotoxin on persistent asthma in an urban population by evaluating whether indoor pollutant exposure modifies the relationship between indoor airborne endotoxin and asthma morbidity.

STUDY POPULATION. One hundred forty-six urban Baltimore children and adolescents, aged 5 to 17 years, with persistent asthma were followed up for 1 year. Median age of participants was 11 years, 57% were male, and >90% were African American. The majority of participants were of low socioeconomic status.

METHODS. Participants underwent clinical assessments at 0, 3, 6, 9, and 12 months. Skin prick testing for 14 allergens and total immunoglobulin E levels were obtained at the baseline visit. Spirometry and fractional exhaled nitric oxide measurements were performed at all study visits, as well as medical questionnaires. Airborne nicotine, endotoxin, and nitrogen dioxide (NO2) levels were measured at home visits. The effects of pollutant exposures on the association between endotoxin exposure and asthma outcomes were assessed via stratified and interaction analyses.

RESULTS. Both air nicotine and NO2 levels modified the relationship between home endotoxin exposure and asthma outcomes. For children with no exposure to nicotine at home, higher endotoxin exposure was associated with fewer acute visits and oral corticosteroid bursts. In contrast, for children in whose homes nicotine was detected, endotoxin exposure was associated with higher rates of acute asthma visits and oral corticosteroid bursts. Higher endotoxin level was positively associated with acute asthma visits for children living in homes with lower NO2 levels (<20 ppb). Fewer acute asthma visits were noted for children living in homes with higher NO2 levels. A similar interaction between endotoxin exposure and NO2 was also noted for asthma-related symptoms.
CONCLUSIONS. This study demonstrates that, for urban children with asthma, the effect of endotoxin exposure on disease morbidity is influenced by levels of exposure to indoor nicotine and NO₂. Airborne endotoxin seems to be protective against acute asthma visits and oral corticosteroid bursts in the setting of very low or no air nicotine exposure, whereas it is associated with worse asthma morbidity in the setting of high indoor air nicotine exposure. In contrast, airborne endotoxin is associated with increased asthma morbidity in the setting of low NO₂ exposure and seems to be protective against asthma-related morbidity in the setting of high NO₂ exposure.

REVIEWER COMMENTS. This study strengthens the idea that multiple components of the air we breathe can have complementing and/or opposing effects on asthma morbidity. This study reiterates the concept that airway hyperreactivity is multifactorial. Due to the high prevalence of concomitant indoor endotoxin exposure and secondhand smoke exposure, the association noted between airborne nicotine and endotoxin exposure may help explain the disproportionate asthma-related morbidity observed in urban populations. The findings support the importance of the interplay of indoor exposures and their effects on asthma. Effective approaches to environmental control, including smoking cessation or home smoking bans, may mitigate the harmful effects of endotoxin on asthma. Further studies are needed to support these conclusions.


Jennilee Mumm, PA-C
Todd A. Mahr, MD, FAAP
La Crosse, WI

Cotinine in Children Admitted for Asthma and Readmission


PURPOSE OF THE STUDY. The goal of this study was to explore the relationship between tobacco smoke exposure (caregiver reported versus serum or salivary biomarkers) and rates of readmission for children hospitalized for asthma.

STUDY POPULATION. This prospective cohort included 774 children aged 1 to 16 years admitted to the hospital for asthma or bronchodilator-responsive wheezing. Of these, 619 children had complete tobacco exposure information.

METHODS. The primary outcome was ≥1 asthma or wheeze-related readmission within 1 year of enrollment in the study. Those results were then stratified based on reported tobacco exposure and measurement of serum and salivary cotinine levels.

RESULTS. Overall, 17% of children were readmitted to the hospital for asthma within 1 year. Tobacco exposure rates were 35.1%, 56.1%, and 79.6% according to report, serum, and saliva measures, respectively. Caregiver report of tobacco exposure was not associated with increased odds of readmission (odds ratio: 1.18 [confidence interval: 0.79–1.89]). In contrast, detectable serum or salivary cotinine was associated with increased odds of readmission (odds ratios of 1.59 [confidence interval: 1.02–2.48] and 2.35 [confidence interval: 1.22–4.55], respectively). Among children in whom caregivers reported no tobacco exposure, 39.1% had detectable serum cotinine levels and 69.9% had detectable saliva cotinine levels. Of those children with reported tobacco exposure, 87.6% had detectable levels of serum cotinine and 97.7% had detectable levels of saliva cotinine. In this study, passive smoke exposure was common and varied significantly with sociodemographic status. African-American children had the highest rates of serum (61.1%) and saliva (86.8%) cotinine. Detection of passive smoke exposure by using biomarkers was inversely proportional to house income: 71.9% of children in households reporting annual income of less than $15 000 had detectable serum cotinine levels versus 11.4% of children with household income of greater than $90 000.

CONCLUSIONS. In this cohort, detectable serum and salivary cotinine levels were common among children admitted for asthma and were associated with readmission, whereas caregiver-reported tobacco exposure was not.

REVIEWER COMMENTS. The results of this study suggest that cotinine levels could be used when an asthmatic child is seen in the emergency department or hospital to help predict risk for future hospitalizations. As it turns out, the level of detection was lower for saliva cotinine than for serum cotinine, which is consistent with previous studies showing increased sensitivity of saliva cotinine levels. Obtaining a saliva cotinine measurement as a proxy for tobacco smoke exposure could be used to target specific interventions (eg, parental counseling and contacting the primary care physician before the asthmatic child is discharged from the emergency department or hospital).


Frank S. Virant, MD
Seattle, WA

Maternal Second-Hand Smoke Exposure in Pregnancy Is Associated With Childhood Asthma Development


PURPOSE OF THE STUDY. The goal of this study was to determine longitudinal associations between maternal secondhand smoke exposure during pregnancy and the development of childhood asthma.
Indoor Pollutant Exposures Modify the Effect of Airborne Endotoxin on Asthma in Urban Children

Jennilee Mumm and Todd A. Mahr

*Pediatrics* 2014;134;S144
DOI: 10.1542/peds.2014-1817U

Updated Information & Services
including high resolution figures, can be found at:
/content/134/Supplement_3/S144.2.full.html

Permissions & Licensing
Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
/site/misc/Permissions.xhtml

Reprints
Information about ordering reprints can be found online:
/site/misc/reprints.xhtml
Indoor Pollutant Exposures Modify the Effect of Airborne Endotoxin on Asthma in Urban Children
Jennilee Mumm and Todd A. Mahr
Pediatrics 2014;134;S144
DOI: 10.1542/peds.2014-1817U

The online version of this article, along with updated information and services, is located on the World Wide Web at:
/content/134/Supplement_3/S144.2.full.html