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.


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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 wheezing-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 salivary cotinine levels. Of those children with reported tobacco exposure, 87.6% had detectable levels of serum cotinine and 97.7% had detectable levels of salivary 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 salivary (86.8%) cotinine. Detection of passive smoke exposure by using biomarkers was inversely proportional to household 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 salivary cotinine than for serum cotinine, which is consistent with previous studies showing increased sensitivity of salivary cotinine levels. Obtaining a salivary 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).

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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.
STUDY POPULATION. This population-based cohort included 5619 seven-year-old children in grades 1 and 2 recruited from a random sample of 283 public schools in the greater Toronto area.

METHODS. Cross-sectional data were collected on demographic characteristics, family history of atopy, smoke exposure, and outcome information until age 6 or 7 years by using validated questionnaires completed by a parent or guardian for all 5619 children. Further detailed longitudinal exposure data were obtained via telephone survey on a randomly selected case-control subset of 1497 children, one-half of whom had a reported history of asthma or wheezing. Statistical methods used to analyze associations included Cox proportional and discrete-time hazard survival analyses.

RESULTS. Increased risk of asthma development was associated with maternal smoking or home secondhand smoke exposure during pregnancy and first year of life, male gender, preterm birth, and maternal asthma. Breastfeeding at least 6 months conferred a protective effect. When adjusting for the aforementioned factors, maternal smoking or secondhand smoke exposure during pregnancy was associated with a 30% increase in adjusted hazard of childhood asthma development. This association persisted for secondhand smoke exposure alone during pregnancy as well as after adjusting for secondhand smoke exposure during the first year of life and for exposure from birth to 7 years.

CONCLUSIONS. The results of this study suggest that there is an increased risk of developing childhood asthma with maternal secondhand smoke exposure during pregnancy, regardless of the mother’s active smoking status during that time.

REVIEWER COMMENTS. This study is the first to evaluate the association between maternal secondhand smoke exposure during pregnancy and childhood asthma development. Previous studies evaluated maternal smoking status during pregnancy, and others assessed early-life exposures after birth. Such findings highlight the need for smoking cessation education not only for pregnant mothers but for all smokers in the home.


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Persistent Effects of Maternal Smoking During Pregnancy on Lung Function and Asthma in Adolescents


PURPOSE OF THE STUDY. The study was conducted to determine if the negative effects of maternal smoking during pregnancy on respiratory health persist into adolescence and, if so, to identify a mechanism.

STUDY POPULATION. The study population included 1129 Australian children, age 14 years, seen for one of multiple scheduled follow-up visits as part of a birth cohort study.

METHODS. Clinical data were collected on current asthma status; serum was collected for total and allergen-specific IgE measurements and for cytokine measurements; and urine was collected for prostaglandin F2α and eosinophil protein X measurements. Prenatal maternal smoking was determined by using an antenatal questionnaire for all participants and with urine cotinine measurements in some participants.

RESULTS. Prenatal exposure to maternal smoking was reported in 21%, and current smoke exposure was reported in 8%. Maternal smoking in pregnancy resulted in a significantly increased risk for current asthma, current wheeze, exercise-induced wheeze, and forced expiratory volume in 1 second/vital capacity <80%. However, there was no increased risk for atopy, current asthma medication use, or bronchial hyperresponsiveness. These associations were not altered when adjustments were made for various factors, including current lung function, specific IgE level, and cytokine and inflammatory markers.

CONCLUSIONS. Maternal smoking during pregnancy resulted in increased risk of asthma and wheezing at age 14 years. This increased risk was not due to increased atopic sensitization or reduced lung function at this age.

REVIEWER COMMENTS. This study is novel in that it assesses the effects of maternal smoking during the prenatal period on asthma and wheezing during adolescence. The findings suggest that prenatal counseling regarding smoking can be used to assist in the primary prevention of asthma.


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Relationship of Secondhand Smoke and Infant Lower Respiratory Tract Infection Severity by Familial Atopy Status


PURPOSE OF THE STUDY. The goal of this study was to establish atopic predisposition as a predictive factor to lower respiratory tract infection severity in infants with secondhand smoke (SHS) exposure.

STUDY POPULATION. Study patients were 451 mother–infant pairs enrolled in TCRI (Tennessee Children’s Respiratory
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