siblings was protective for development of rhinitis and allergic rhinitis at age 1.

CONCLUSIONS. ETS exposure is associated with rhinitis and allergic rhinitis in infants. Mold may be a stronger risk factor for URI than ETS.

REVIEWER COMMENTS. This is one of very few studies to have examined the relevance of home exposures in the first year of life and demonstrated that early environmental exposures likely play a significant role in respiratory health outcomes in infancy. One weakness of the study was the fact that ETS exposure and URI and rhinitis symptoms were collected by parental report and may be subject to significant recall bias or erroneous reporting. Nevertheless, these findings support the need for further research in the areas of early environmental-exposure interventions and the role of gene-environment interactions that affect respiratory health outcomes.

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TOBACCO AND AIR POLLUTION

Parental Smoking Increases Exhaled Nitric Oxide in Children

PURPOSE OF THE STUDY. Fraction of exhaled nitric oxide (FeNO) seems to reflect allergic airway inflammation. The authors sought to investigate the association between reported parental smoking and FeNO in young children.

STUDY POPULATION. The study included 78 children (mean age: 51.3 weeks; range: 13–106 weeks) who had FeNO data and had been well for at least 2 weeks before testing. Fifty-six children lived in nonsmoking households, 14 lived with 1 smoking parent, and 8 lived with 2 smoking parents.

METHODS. Children underwent pulmonary-function testing that included measures of lung volumes (forced expiratory volume [FEV] in 0.5 second) and FeNO. The effect of parental smoking on FeNO and FEV was analyzed by using unpaired t tests and analysis of variance. Dose-response relationships between the number of smoking parents and FeNO and FEV were determined by using the χ² test. There were no significant anthropometric differences between children in smoking and nonsmoking households.

RESULTS. Children who had at least 1 smoking parent had a higher FeNO compared with children who lived in nonsmoking households (41.9 vs 33.0 ppb, respectively). Within the smoking group, children who lived with 2 smoking parents had a higher FeNO compared with children who lived with 1 smoking parent (48.3 vs 38.0 ppb, respectively). The difference between these 3 groups was not significant. However, there was a significant dose-response relationship across the 3 groups. Moreover, after controlling for other factors, it was found that parental smoking significantly increased the FeNO. Age, gender, maternal atopy, and doctor-diagnosed eczema and FEV were not associated with FeNO.

CONCLUSIONS. Exposure to environmental tobacco smoke was associated with increased FeNO in young children. There also was evidence of a dose-response relationship between childhood FeNO and the number of smoking parents.

REVIEWER COMMENTS. The role of FeNO as a marker for airway inflammation after exposure to tobacco smoke seems ambiguous. The authors mentioned that although some studies have shown that tobacco smoke has been associated with lower FeNO in smokers, other studies have found a short-term increase in FeNO directly after smoking a cigarette. Moreover, most epidemiologic studies have not found an effect of parental smoking on FeNO in older children. The authors argued that reported parental smoking alone may not be a good indicator for exposure in older children, because they are able to move themselves from the source, compared with younger children who are unable to move from the offending environment. Whether FeNO can prove to be a useful biomarker of allergic sensitization or airway inflammation still remains in question. In addition, even if the association between cigarette-smoke exposure and high FeNO remain after better-powered studies, further investigations are warranted to elucidate what the potential clinical ramifications are for children with high levels of exhaled nitric oxide.

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TNF-308 Modifies the Effect of Second-hand Smoke on Respiratory Illness-Related School Absences

PURPOSE OF THE STUDY. To investigate the role of tumor necrosis factor (TNF) G→308A, a variant in the promoter region of TNF, that has been associated with inflammatory diseases including asthma in the susceptibility of secondhand-smoke–exposed children to respiratory illness.

STUDY POPULATION. A prospective cohort of fourth-grade students (N = 1935) from 27 elementary schools in
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