mice unexposed to smoke. Mice then underwent intratracheal injection of ovalbumin-fluorescein isothiocyanate (FITC) or ovalbumin and were killed at varying time points thereafter depending on the aspect of DC function being studied (migration, maturation, or T-cell activation). Lung cells were isolated, and the numbers of DCs were determined in exposed and unexposed mice by flow cytometry. Migration of lung DCs to thoracic lymph nodes (TLNs) was assessed by measuring CD11c\textsuperscript{+}FITC\textsuperscript{+} cells. Maturation of TLN DCs after ovalbumin exposure was then determined by assessing expression of cell-surface costimulatory molecules (CD80, CD86, MHCII, CD40). T-cell proliferation was assessed by measuring T-cell–derived cytokine production by TLN cells in vitro. The staining profile was assessed to measure T-cell proliferation in vivo.

RESULTS. The number of lung DCs was decreased among mice exposed to cigarette smoke. Exposed mice did not exhibit decreased migration of lung DCs to TLNs, as evidenced by similar numbers of TLN CD11c\textsuperscript{+}FITC\textsuperscript{+} cells as in unexposed mice. Although no differences were observed in migration, smoking suppressed DC maturation in the lymph nodes as demonstrated by decreased cell-surface expression of MHC class II and the costimulatory molecules CD80 and CD86. T-cell proliferation was impaired in smoke-exposed mice, evidenced by reduced interleukin 2 production from CD4 T cells as was impaired in smoke-exposed mice, evidenced by regulatory molecules CD80 and CD86. T-cell proliferation was assessed by measuring T-cell–derived cytokine production by TLN cells in vitro. The staining profile was assessed to measure T-cell proliferation in vivo.

CONCLUSIONS. These findings suggest that cigarette smoke is associated with defects in DC maturation and suppressed thoracic lymph node CD4\textsuperscript{+} T-cell proliferation in mice.

REVIEWER COMMENTS. This study suggests that cigarette-smoke exposure in mice leads to impaired DC maturation and thereby diminished T-cell clonal expansion in the TLNs. The findings imply that, although the T-cell effector program remains intact, fewer cells might be available to perform those duties. The authors asserted that the observed effects on DC function might serve as a potential mechanism for cigarette smoke causing impaired tumor surveillance. In addition, it is suggested that impaired CD4\textsuperscript{+} T-cell function might predispose patients with obstructive lung disease to infections and exacerbations. Additional studies will help to elucidate the specific mechanisms by which cigarette-smoke exposure exerts its harmful immunologic effects.

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Air Quality and Pediatric Asthma-Related Emergencies

PURPOSE OF THE STUDY. To examine the effects of seasonality, outdoor air quality, climatic factors, and presence of outdoor aeroallergens on emergency department (ED) visits for children with asthma.

STUDY POPULATION. Dates with “low” and “high” pediatric ED visits with a primary diagnosis of asthma (International Classification of Diseases, Ninth Revision code 493) were identified for children presenting to Alfred I. du Pont Hospital for Children from January 1, 2000, to December 31, 2003. Dates with zero ED visits for asthma were labeled as low, and dates with ≥7 ED visits for asthma were labeled as high.

METHODS. This was a retrospective review of ED visits for asthma and environmental factors, with 8-hour average ozone, 24-hour average nitrogen dioxide (NO\textsubscript{2}), and particulate matter with an aerodynamic diameter of <2.5 μm (PM\textsubscript{2.5}) levels, as well as tree pollen, weed pollen, grass pollen, and mold levels obtained. Monthly minimum, maximum, and mean temperatures and precipitation levels were obtained. Seasonal and environmental factors associated with high and low ED visits for asthma were compared with independent t tests.

RESULTS. Of 1460 dates reviewed over a 4-year period, 106 were classified as low-visit days and 103 were classified as high-visit days. Of high-visit days, 45.6% were in autumn, 28.2% were in spring, 14.6% were in winter, and 11.7% were in summer. The greatest proportion of high-visit days occurred in September. Mean 8-hour average ozone for low-visit days was significantly higher than on high-visit days (0.047 vs 0.033 ppm). No differences were observed in mean 24-hour average NO\textsubscript{2} levels on high- and low-visit days. The mean 24-hour average PM\textsubscript{2.5} was higher on low-visit days (18.54 μg/m\textsuperscript{3}) versus high-visit days (13.28 μg/m\textsuperscript{3}). Trends were observed with higher weed and tree pollen on high-visit days. Mean temperature and precipitation were both significantly lower on high-visit days.

CONCLUSIONS. Asthma-related ED visits were associated with aeroallergens and climactic factors. Air pollutants seem to play a smaller role in asthma-related ED visits.

REVIEWER COMMENTS. This retrospective analysis has identified significant seasonal variation in high and low ED visits for pediatric asthma. The results suggest that the observed seasonal variation may be related to weed- and tree-pollen levels and climactic factors. However, the study may have been inadequately powered to identify minor differences between the high- and low-visit groups. Air pollutant levels were not associated with asthma-related ED visits in the expected direction (low-visit days had higher pollutant levels). Perhaps there is a
Effect of Exposure to Traffic on Lung Development From 10 to 18 Years of Age: A Cohort Study

PURPOSE OF THE STUDY. To investigate the association between residential exposure to traffic and 8-year lung-function growth in children.

STUDY POPULATION. Two cohorts of 4th-grade children with a mean age of 10 years were recruited from 12 southern California communities and were followed for 8 years. All eligible children were invited, and 3677 (82%) participated; 1445 children were followed for the full 8 years.

METHODS. Yearly pulmonary-function data were obtained for each participant by trained technicians using standard equipment. Indicators of residential exposure to traffic were determined by proximity of the child’s residence to the nearest freeway or major nonfreeway road and by dispersion-model estimates including residence distance to roadways, vehicle counts, vehicle emission rates, and meteorological conditions. Regional air pollution was monitored at a central site within each community. Baseline questionnaires were completed regarding demographic data, doctor-diagnosed asthma, in utero exposure to maternal cigarette smoke, and household exposure to air pollutants. Yearly questionnaires updated information on asthma and personal or environmental tobacco-smoke exposure. Regression models also included adjustment for height, BMI, and recent exercise and respiratory illness.

RESULTS. Children living <500 m from a freeway had reduced 8-year lung-function growth compared with children living >1500 m from a freeway (forced expiratory volume in 1 second deficit: −81 mL [95% confidence interval: −143 to −18]). This effect was slightly greater after adjustment for socioeconomic status and indoor air pollution and after omission of children who changed residence within the study area and continued to participate. Reduced lung-function growth was independently associated with freeway distance and regional air pollutant levels, including nitrogen dioxide, acid vapor, elemental carbon, and particulate matter with aerodynamic diameters of <10 and 2.5 µm. At 18 years of age, lung function was decreased among children who lived <500 m from a freeway (forced expiratory volume in 1 second: 97.0% [95% confidence interval: 94.6 to 99.4]; P = .013).

CONCLUSIONS. The adverse effects of local exposure to freeway traffic on children’s lung development are independent of regional air quality and may result in lung-function deficits later in life.

REVIEWER COMMENTS. This well-designed study suggests a causal association between residential traffic exposure and adverse effects on children’s lung function, especially for children living closest to freeways. A number of other recent cohort studies in children have also suggested associations between traffic-pollution exposure and asthma, respiratory symptoms, and allergic sensitization. Future studies are needed to determine minimum safe distances from major roadways for homes and schools and to continue evaluation of the guidelines restricting levels of airborne pollutants. Concerns regarding the effects of traffic pollutants on children’s respiratory health should continue to be a focus of asthma research.

FOOD ALLERGY
Prevalence and Cumulative Incidence of Food Hypersensitivity in the First 3 Years of Life

PURPOSE OF THE STUDY. To investigate the prevalence and incidence of food hypersensitivity.

STUDY POPULATION. The authors studied a whole population-based birth cohort of 969 children (91% of the target population) born on the Isle of Wight (United Kingdom) between 2001 and 2002.

METHODS. At age 1, 2, and 3 years, all children/parents were invited to attend a clinic for a medical examination and to answer a questionnaire pertaining to food hypersensitivity (FHS), defined as any adverse reaction to food. In addition, all children were asked to participate in skin-prick testing (SPT) to milk, egg, wheat, peanut, sesame, fish, aero-allergens, and other allergens as
# Air Quality and Pediatric Asthma-Related Emergencies

Michelle L. Macy and Harvey L. Leo

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