Exposure to air pollution has been associated with decrements in lung function (Castillejos et al. 1995; Gauderman et al. 2007; Moshammer et al. 2006; Romieu et al. 1997, 1998; Svendsen et al. 2007) and an increase in respiratory symptoms (Castillejos et al. 1992; Leonardi et al. 2000), effects to which asthmatic children appear more susceptible (Romieu et al. 1996; Ward and Ayres 2004). Experimental and epidemiologic studies have also shown that fossil fuel–combustion products act as an adjuvant in the immune system and may lead to enhancement of allergic reactions (Nel et al. 1998) to airway inflammation in susceptible subjects (Holguin et al. 2007; Leonardi et al. 2000; Yeatts et al. 2007) with potential long-term effects (Calderon-Garciduenas et al. 2006). However, few longitudinal epidemiologic studies have evaluated the impact of fossil fuel combustion on airway inflammatory response (Fischer et al. 2002; Koenig et al. 2003, 2005; Steerenberg et al. 2001) using an integrated approach including markers of airway inflammatory response and lung function changes.

Asthma is a complex disease characterized by inflammation and hyperresponsiveness of the airways. Nitric oxide signaling pathways have been implicated in the regulation of airway hyperresponsiveness and recently of fractional exhaled nitric oxide (FeNO), interleukin-8 (IL-8) in nasal lavage, and pH of exhaled breath condensate every 15 days during follow-up. Data were analyzed using linear mixed-effects models.

RESULTS: An increase of 17.5 pg/m^3 in the 8-hr moving average of PM2.5 levels (interquartile range) was associated with a 1.08-pbb increase in FeNO (95% confidence interval (CI), 1.01–1.16) and a 1.07-pg/mL increase in IL-8 (95% CI 0.98–1.19) in asthmatic children and a 1.16 pg/ml increase in IL-8 (95% CI 1.00–1.36) in nonasthmatic children. The 5-day accumulated average of exposure to particulate matter < 2.5 µm in aerodynamic diameter (PM2.5) was significantly inversely associated with forced expiratory volume in 1 sec (FEV1) (p = 0.048) and forced vital capacity (FVC) (p = 0.012) in asthmatic children and with FVC (p = 0.021) in nonasthmatic children. FeNO and FEV1 were inversely associated (p = 0.005) in asthmatic children.

CONCLUSIONS: Exposure to PM2.5 resulted in acute airway inflammation and decrease in lung function in both asthmatic and nonasthmatic children.

KEY WORDS: air pollution, airway inflammation, asthma, epidemiology, lung function, schoolchildren. Environ Health Perspect 116:832–838 (2008). doi:10.1289/ehp.10926 available via http://dx.doi.org/ [Online 8 February 2008]
Collection of health outcomes. We collected data using a general-purpose questionnaire (adapted from existing survey instruments) on sociodemographic variables, past health history, and potential indoor environmental exposures (tobacco smoke and pets in the home). Information on allergy test results, medication, and medical visits in the preceding 2 years was obtained from the medical record. At baseline and every 15 days during follow-up, a respiratory symptoms questionnaire was applied, anthropometric measurements were taken, spirometric tests performed, FeNO levels measured, and samples of nasal lavage and EBC obtained. Spirometric tests were performed on 158 asthmatic children (1,503 measurements) and 50 nonasthmatic children (591 measurements). Measurements were repeated an average of 11 times (range, 5–21) per subject during the study period. We were unable to take all samples for inflammatory markers from all the children, for logistic reasons. We obtained 702 measurements of FeNO from 126 asthmatic children and 302 from 50 nonasthmatic children. 759 measurements of IL-8 in nasal lavage in the laboratory of D. Diaz–Sanchez, using commercially available ELISA kits according to the manufacturer’s instructions. However, except for IL-8, the levels in most of the samples were below the detection limit and we report only the IL-8 results. For logistic reasons, we did not determine cellular composition.

EBC collection. EBC was collected using an R-tube, and the breath was cooled by placing an aluminum cooling sleeve over the disposable polypropylene tube (Hunt 2002). Samples were obtained following the ATS/ERS (European Respiratory Society) Task Force recommendations (Hovath et al. 2005; Hunt 2002). Participants were asked to breathe tidally through the mouthpiece connected to the R-tube for 10 min to collect approximately 2 mL of exhaled breath fluid, which was aliquoted and frozen to −70°C within 15 min of collection.

Exposure assessment. Exposure was estimated from outdoor PM$_{2.5}$ (particulate matter < 2.5 μm in aerodynamic diameter), NO$_2$ and O$_3$ concentrations recorded by the Mexico City government at four fixed-site central monitoring [Red Automática de Monitoreo Atmosférico (RAMA)] locations within the study area (Cerro de la Estrella and Hangares; Merced; Universidad Autonoma Metropolitana, Iztapalapa; and La Perla). Daily average, maximum moving average and 8-hr maximum ozone, nitrogen dioxide, and PM$_{2.5}$ concentrations, and meteorologic data (temperature and humidity) were obtained for all (505) days of the study period. The home of each participating child was georeferenced using a geographic information system (GIS), and the closest monitoring station was assigned to the child. All children attended public schools located close to their home, and no fixed-site monitoring station was > 5 km from a child’s home or school. We also conducted monitoring at each school for three 15-day periods during the follow-up to validate data obtained from the fixed-site monitoring stations (RAMA). Local daily 24-hr average PM$_{2.5}$ was determined using Mini-Vol portable air samplers (version 4.2;
Airmetrics, Eugene, Oregon, USA) with 47-mm Teflon filters (R2P)047; Pall Gelman, Ann Arbor, MI, USA) and flows set at 5 L/min, and 7-day integrated data for NO₂ and O₃ concentrations were obtained using Ogawa passive samplers (Ogawa USA, Pompano Beach, FL, USA) (Watson et al. 1999). Samplers were located outside the 37 schools, generally on the roof, at a height of up to 4 m and far from any objects (e.g., trees, buildings) that would prevent air flow. Gravimetric analysis of the 47-mm Teflon filters was performed at the air laboratory of the National Center for Environmental Research and Training (CENICA) in Mexico City. The NO₂ and O₃ filters were assembled at the Mexico City laboratory. After exposure, all badges were placed in sealed bags and sent to the Harvard School of Public Health for chemical analysis (Levy et al. 1998).

**Statistical analysis.** The basic characteristics of the two groups of children were compared by bivariate analysis using the t-test, the Fisher exact test, or the chi-square test, depending on variable type. The short-term association of traffic-related pollutants (PM₂.₅, O₃, and NO₂ concentrations at the fixed-site monitoring stations) with the health outcomes was studied using linear mixed-effects models, considering models for continuous and binary response. This enabled us to appreciate the variability within and between subjects. We ran models with both random intercept and random slope, and with random intercept only. Because the coefficients were similar in both types of model, we present results from the linear mixed model with random intercept only. The model is as follows:

\[ Y_i = X_i \beta + Z_i \delta + \epsilon_i \]  

where \( X_i = Z_i X_i \) is the appropriate (n x p) matrix of known covariates with fixed effects \( \beta \) and subject-specific effects \( \delta \) and \( \epsilon \) in an n-dimensional vector of residual components. Another advantage of the models used is that they do not discard subjects with incomplete data. We determined the goodness of fit of each model using residual diagnosis and the Hausman specification test (Hausman 1978).

Based on our previous work and international studies conducted among asthmatic children (Delfino et al. 2004, 2006; Romieu et al. 1996, 2002, 2004; Schildcrout et al. 2006), we hypothesized a priori that the acute effect of air pollutants on pulmonary functions would occur with lags of 1–2 days and that a larger effect would be observed when considering cumulative exposure over several (up to 5) days, whereas for inflammatory markers (FeNO levels, IL-8 levels, pH of EBC) we hypothesized a shorter response with 0- to 1-day lag.

We modeled several pollution exposure indices (8-hr maximum moving average, 24-hr average, 24-hr maximum average, accumulated days). Pearson correlations were determined between air pollutant levels and various climatic variables. FeNO and IL-8 levels were not normally distributed and were log transformed. Models were adjusted for potential confounding factors including sex, body mass index, previous day minimum temperature, corticoid use, and chronological time. Other variables such as age, socioeconomic index (considering mother education and school type), outdoor activities, atopic status, exposure to environmental tobacco smoke, use of antiallergy medicine, and season were not significant (\( p > 0.10 \)) and did not alter the results by >1%. Analyses were conducted using STATA (version 9.2; StataCorp., College Station, TX, USA).

**Results**

Table 1 presents the characteristics of the study population. The median age of participants was 9.6 years (quartile (Q) 25: 7.9; Q75: 11.0) for the asthmatic and 9.3 (Q25: 7.9; Q75: 11.5) for the nonasthmatic children. Of the asthmatic children, 55% were classified as having mild intermittent, 26.9% as having mild persistent, and 17.5% as having moderate

---

Table 1. Basic characteristics and main outcomes of the study population.

| Variable                              | Asthmatic (n = 158) | Nonasthmatic (n = 50) | p-Value |
|---------------------------------------|---------------------|----------------------|---------|
| Sex (% male)                          | 61.9                | 40.0                 | 0.005   |
| Age (years)                           | 9.6 (7.9–11.0)      | 9.3 (7.9–11.5)       | 0.986   |
| Weight (kg)                           | 36.0 (27.0–46.0)    | 32.0 (26.0–45.0)     | 0.307   |
| Height (cm)                           | 137.0 (124.5–147.0) | 134.0 (127.0–147.0)  | 0.842   |
| Paternal smoking at home (%)          | 54.8                | 45.0                 | 0.424   |
| Maternal smoking at home (%)          | 41.1                | 28.6                 | 0.281   |
| Pets at home (%)                      | 56.6                | 72.7                 | 0.035   |
| Carpet at home (%)                    | 14.4                | 34.6                 | 0.001   |
| Humidity at home (%)                  | 42.5                | 41.5                 | 0.899   |
| Prick test positivity (%)             | 89.0                | 72.0                 | 0.129   |
| Moderate persistent asthma (%)        | 17.5                |                      |         |
| Mild persistent asthma (%)            | 26.9                |                      |         |
| Mild intermittent asthma (%)          | 55.0                |                      |         |
| Exhaled NO levels (ppb)               | 23.2 (11.2–46.7)    | 11.2 (6.0–20.1)      | 0.000   |
| IL-8 levels (pg/mL) in nasal lavage   | 157.2 (78.2–295.1)  | 202.2 (113.0–333.6)  | 0.002   |
| pH of EBC                             | 7.43 (7.1–7.6)      | 7.56 (7.3–7.8)       | 0.632   |
| FEV₁ [L/sec (mean ± SD)]              | 1.89 ± 0.66         | 1.95 ± 0.59          | 0.595   |
| FVC [% (mean ± SD)]                   | 2.30 ± 0.79         | 2.25 ± 0.88          | 0.678   |
| FEV₂-% (mean ± SD)                    | 1.89 ± 0.89         | 2.15 ± 0.88          | 0.718   |

Q_1, quartile.  
*Mann-Whitney test (median) (Q25–Q75).*

---

Figure 2. Eight-hour moving average concentrations of PM₂.₅ (µg/m³) during the study period, Mexico City, 2003–2006.
persistent asthma, according to the GINA guidelines. Of the asthmatic children, 6% used an inhaled corticosteroid and 10% were prescribed antibiotics on at least one occasion during follow-up. Eighty-nine percent of the asthmatic children and 72% of the non-asthmatic children had positive skin prick tests. The most common sensitivities were to house dust mite (Dermatophagoides pteronyssinus), cat (Fel d 1), and cockroach (Blatella americana) allergens.

Participants with asthma had higher average levels of FeNO (p = 0.001) and lower average levels of IL-8 (p = 0.002). The pH measurements in EBC were similar in both groups (p = 0.632).

Environmental exposure data. The 8-hr moving average PM$_{2.5}$ ranged from 4.24 to 102.8 µg/m$^3$ during the study period, with a mean of 28.9 µg/m$^3$. It exceeded 30 µg/m$^3$ for PM$_{2.5}$, 13.4 ppb for NO$_2$, and 22 ppb for O$_3$. Lung function models: n = 158 and 1,503 measurements. Inflammatory marker models: n = 119 and 551 measurements of pH in EBC.

| Variable | Mean ± SD | IQR* | Min–max |
|---------|----------|------|---------|
| FeNO$_a$ (ppb) | 1.08 (1.01 to 1.16)* | 1.05 (0.98 to 1.12) | 1.06 (1.02 to 1.09)* |
| IL-8$_b$ (pg/mL) | 1.16 (0.98 to 1.36)** | 1.10 (0.99 to 1.23)** | 1.11 (0.92 to 1.33)* |
| pH$_{EBC}$ | -0.05 to -0.09 | 0.001 to 0.007 | -0.07 to 0.02 |
| FEV$_1$ | -21.0 (–31.0 to –0.13)* | -11.0 to -12.0 to 9.80 | -13.5 (–50 to 19.0) |
| FEV$_{25–75}$ | -11.0 to -20.2 | -0.04 to 8.86 to 8.79 | -6.4 to 29.0 |

*Coefficient was calculated for an IQR of pollutants: 17.5 µg/m$^3$ for PM$_{2.5}$, 13.4 ppb for NO$_2$, and 22 ppb for O$_3$. Lung function models: n = 158 and 1,503 measurements. Inflammatory marker models: n = 119 and 551 measurements of pH in EBC.

Table 4. Association [coefficients per increase in IQR (95% CI)] between exhaled NO, IL-8, pH of EBC, and lung function and air pollutants in asthmatic children living in Mexico City, 2003–2005.

| Variable | PM$_{2.5}$ (µg/m$^3$) | NO$_2$ (ppb) | O$_3$ (ppb) |
|---------|----------------------|-------------|-------------|
| FeNO$_a$ (ppb) | 0.89 (0.78 to 1.01)* | 1.10 (0.99 to 1.23)** | 1.11 (0.92 to 1.33)* |
| IL-8$_b$ (pg/mL) | 1.16 (1.00 to 1.36)** | 1.15 (1.01 to 1.32)** | 1.19 (1.00 to 1.45)* |
| pH$_{EBC}$ | -0.05 (–0.14 to 0.04) | -0.07 (–0.07 to 0.09) | -0.07 (–0.02 to 0.05) |
| FEV$_1$ | -21.0 (–42.3 to 0.38) | -6.73 (–22.0 to 9.52) | -21.3 (–66.5 to 23.9) |
| FEV$_{25–75}$ | -29.0 (–52.8 to –4.36)** | -9.51 (–27.0 to 7.91) | -23.6 (–75.0 to 28.1) |

*Coefficient was calculated for an IQR of pollutants: 17.5 µg/m$^3$ for PM$_{2.5}$, 13.4 ppb for NO$_2$, and 22 ppb for O$_3$. Lung function models: n = 158 and 1,503 measurements. Inflammatory marker models: n = 119 and 551 measurements of pH in EBC.

*Same day exposure: 8-hr moving averages for PM$_{2.5}$ (µg/m$^3$), NO$_2$ (ppb), and O$_3$ (ppb). Inflammatory marker models were adjusted for sex, body mass index, previous day minimum temperature, corticoid use, and chronological time. Five-day accumulated average (maximum) PM$_{2.5}$ (µg/m$^3$), 4-day accumulated average (maximum) NO$_2$ (ppb), and 5-day accumulated moving average O$_3$ (ppb). Lung function models were adjusted for sex, body mass index, previous day minimum temperature and chronological time.

*p < 0.05.
pollutants. An increase in PM$_{2.5}$ of 17.5 µg/m$^3$ was associated with an 11% increase in cough (odds ratio (OR) = 1.11; 95% CI, 1.06–1.17) and a 6% increase in wheezing (OR = 1.06; 95% CI, 0.99–1.13). An increase in NO$_2$ of 34 ppb (1-hr maximum) was associated with a 10% increase in cough (OR = 1.10; 95% CI, 1.04–1.16) and a 10% increase in wheezing (OR = 1.10; 95% CI, 1.03–1.18). And an increase in O$_3$ of 48 ppb (1-hr maximum) with a 9% in cough (OR = 1.09; 95% CI, 1.03–1.15) but no significant increase in wheezing. Among nonasthmatic children, only an increase in cough related to cumulative NO$_2$ exposure (OR = 1.22; 95% CI, 1.03–1.45 for an increase of 24.5 ppb in 2-day cumulative exposure) was observed.

In multipollutant models including O$_3$ as well as PM$_{2.5}$, we noted that among asthmatic children O$_3$ remained significantly related to inflammatory markers (FeNO, IL-8, and pH of EBC) whereas PM$_{2.5}$ remained inversely related to pulmonary functions (FEV$_1$ and FVC) but lost its significant effect on FeNO. Among nonasthmatic children, the inverse relation between PM$_{2.5}$ and FEV$_1$ and FVC persisted, whereas PM$_{2.5}$ and O$_3$ had a lesser effect on inflammatory markers (data not shown).

A large proportion (72%) of the nonasthmatic children were atopic, so we repeated the analysis including only atopic children with our asthma. In this subgroup we observed a significant increase of FeNO related to PM$_{2.5}$ exposure, suggesting that atopy increases the FeNO response (data not shown).

**Discussion**

The results of the present cohort study of asthmatic and nonasthmatic children show that FeNO levels, IL-8 levels in nasal lavage, pH of EBC, and changes in lung function are associated with acute exposure to traffic-related air pollutants. Changes in FeNO were inversely associated with FEV$_1$ in asthmatic children, suggesting that the inflammatory response of airways most likely influences the decrease in lung function. The effects on inflammatory markers were higher for PM$_{2.5}$ and O$_3$ concentrations than for NO$_2$. The effect appears on the same day as the exposure and can cumulate over several days, resulting in lung function decrement after 4 or 5 days of cumulative exposure.

Although exposure to outdoor ambient levels of PM$_{2.5}$, NO$_2$, and O$_3$ has been associated with increased asthma and respiratory symptoms in children, there are few previous longitudinal studies combining inflammatory response and change in lung function in response to air pollution, particularly in nonasthmatic children. Epidemiologic panel studies report that exposure to air pollutants increases the FeNO levels in susceptible children. Delfino et al. (2006), showed that an IQR increase of 73 µg/m$^3$ in 1-hr maximum personal PM$_{2.5}$ was associated with a 0.60-ppb increase in FeNO (95% CI, 0.14–1.05) in asthmatic children who live in an urban area. A panel study of asthmatic children in Seattle, Washington, reported that ambient PM$_{2.5}$ concentrations were also associated with increased levels of exhaled NO (Mar et al. 2005). We recently reported an increase in FeNO in asthmatic children living close to roads (50-m buffer) whereas no effect was observed in nonasthmatic children; but no data on lung function or other inflammatory markers were available (Fischer et al. 2002; Holguin et al. 2007; Steerenberg et al. 1999). The present study provides evidence of the adverse effect of traffic-related pollution not only in asthmatic but also in nonasthmatic children.

The effect of PM$_{2.5}$ on IL-8 was stronger in nonasthmatic than in asthmatic children. This was contrary to our expectation. However this has already been reported in another population, in which exposure to diesel exhaust provoked airway inflammation with airway neutrophilia and an increase of IL-8 in healthy subjects shortly after exposure but did not induce neutrophilic response in asthmatic subjects (Stenfors et al. 2004).

A potential explanation is that, in asthmatics, IL-10 might inhibit the synthesis of many inflammatory cytokines including IL-8, which has low baseline levels in asthmatic subjects (Barnes and Lim 1998; Stenfors et al. 2004). A large proportion (72%) of the nonasthmatic children were atopic, so we repeated the analysis including only atopic children with asthma. In this subgroup we observed a significant increase of FeNO related to PM$_{2.5}$ exposure, suggesting that atopy increases the FeNO response (data not shown).

Another contribution of our study is the evaluation of pH of EBC related to air pollutant exposure. We hypothesized that asthmatic children would have a lower exhaled breath pH at baseline than nonasthmatic children and that exposure to air pollutants would lead to a decrease in the pH of exhaled condensate as a marker of lower airway inflammation. The exhaled breath condensate of asthmatics contains a high concentration of reactive oxygen species (ROS) and reactive nitrogen species (RNS), reflecting changes in the lower respiratory tract during inflammation and affecting local pH. This acidification might contribute to asthma pathophysiology (Hunt et al. 2000). Exposure to air pollutants, particularly O$_3$, which has a strong oxidative potential and acts in the lower airway, is likely to result in an increase in ROS and RNS. This would affect the pH of the lower respiratory tract, reflecting local inflammation. We found that the pH of EBC decreased in both nonasthmatic and asthmatic children with air pollutant exposure, and significantly so with O$_3$ exposure in asthmatic children. Brunetti et al. (2006) observed that the pH of EBC in patients with acute asthma was lower than that of patients with stable asthma, and the amplitude of the effect we observed among asthmatic children in response to O$_3$ (16-ppb increase) was similar to that described by these authors. Our results therefore add a plausible...
biological mechanism to the association of traffic-related emissions and adverse respiratory health effects. An important issue is the interrelation between inflammatory response and change in lung function in relation to air pollution exposure. Among asthmatic children, FEV₁ was inversely related to FeNO, IL-8, and PM_{2.5} exposure, but the coefficient of PM_{2.5} decreased when the inflammatory markers were introduced in the models. In contrast, among nonasthmatic children this adjustment strengthened the inverse association between FEV₁ and PM_{2.5}. This suggests that, in asthmatic children, the inflammatory response may partly explain the effect of PM_{2.5} on FEV₁. We also observed a significant inverse association between decrement in FEV₁ and increase in FeNO in asthmatic children. However, given the complexity of inflammatory responses to air pollutants, these results need to be interpreted with caution.

Some limitations must be taken into account when interpreting the results of this study. Daily variations in air pollutant exposure were evaluated through the daily records of the fixed central monitoring locations (RAMA). The temporal variations in each child’s exposure were assumed to follow those at the central monitoring site. To strengthen the validity of this assumption, each child was assigned to the monitoring site closest to his or her home by means of a spatial GIS, providing greater variability in the data. We also monitored PM_{2.5}, NO₂, and O₃ at the schools and correlated the data from the two sets of monitors. The correlations between the fixed central monitors and the local monitors were 0.77, 0.21, and 0.60 respectively. The lowest correlation was for NO₂, as in other studies (Kodama et al. 2002), whereas the highest was for PM_{2.5}, which might explain why significant changes in lung function were observed only for this pollutant, possibly because of better estimation of exposure. However, specific components of PM_{2.5} also appear to play a role in the adverse effect of this pollutant. When we analyzed data on elemental carbon levels measured in a subsample of PM_{2.5} filters (n = 37) from the school-based monitors, we observed that elemental carbon levels were inversely associated with FEV₁ and FVC in a subgroup of 30 asthmatic children (data not shown) (ρ = 0.005), highlighting the role of heavy vehicle (diesel) emissions as a source of exposure (Janssen et al. 2001) and strengthening the causal relationship with PM_{2.5}. Finally, although some degree of exposure may be confounded by other unmeasured factors such as variations in socioeconomic status, this is unlikely in the present study because all our participants came from the same study area and attended the same public school system; and further adjustment by socioeconomic status did not modify our results.

Most of the asthmatic children in this study were classified as having mild intermittent asthma (55%) and only 17.5% as having moderate persistent asthma. This limited our ability to study the impact of air pollution on asthma severity. In previous studies conducted in Mexico City, we had observed that children with moderate to severe asthma were more susceptible to air pollution (Romieu et al. 2004). Our results might therefore underestimate the impact of air pollution on children with more severe asthma.

**Conclusion**

Our data show that FeNO levels, IL-8 levels in nasal lavage, pH of EBC, and changes in lung function are associated with acute exposure to traffic-related air pollutants. These adverse effects were observed in a longitudinal setting in a free-living population, more specifically in a cohort of schoolchildren including nonasthmatic children. Among the asthmatic children, air pollution exposure was related to an increase in FeNO levels and a decrease in lung function, whereas neutrophil airway inflammation as well as a decrease in lung function was observed in the nonasthmatic children. These results could have significant public health policy implications, because a large proportion of schools in Mexico City and other countries are located very close to roads with heavy traffic.

**References**

ATS (American Thoracic Society). 1995. Standardization of spirometry. Am J Respir Crit Care Med 152:1107–1130.

ATS (American Thoracic Society). 1999. Recommendations for standardized procedures for the online and offline measurement of exhaled lower respiratory nitric oxide and nasal nitric oxide in adults and children—1999. Am J Respir Crit Care Med 160:2044–2017.

Barnes PJ. 1996. NO or NO in asthma? Thorax 51:218–220.

Barnes PJ, Lim S. 1998. Inhibitory cytokines in asthma. Mol Med Today 4:452–458.

Behneidig AF, Mudway IS, Brown JL, Stenfors N, Hellelly D, Duggan ST, et al. 2006. Airway antioxidant and inflammatory responses to diesel exhaust exposure in non-asthmatic humans. Eur Respir J 27:359–365.

Brunetti L, Francavilla R, Tesei B, Strippoli A, Pelizzoni L, Loforese A, et al. 2006. Exhaled breath condensate pH measurement in children with allergic rhinitis and atopic dermatitis. Pediatr Allergy Immunol 17:422–427.

Castillejos M, Gold DR, Dockery D, Tosteson T, Baum T, Speizer FE. 1992. Effects of ambient ozone on respiratory function. Am J Respir Crit Care Med 146:1025–1032.

Castillejos M, Gold DR, Dockery D, Tosteson T, Baum T, Speizer FE. 1992. Effects of ambient ozone on respiratory function. Am J Respir Crit Care Med 146:1025–1032.

Castillejos M, Gold DR, Dockery D, Tosteson T, Baum T, Speizer FE. 1992. Effects of ambient ozone on respiratory function. Am J Respir Crit Care Med 146:1025–1032.

Castillejos M, Gold DR, Dockery D, Tosteson T, Baum T, Speizer FE. 1992. Effects of ambient ozone on respiratory function. Am J Respir Crit Care Med 146:1025–1032.
Narayanan PK, LaRue KE, Goodwin EH, Lehnert BE. 1999. Alpha particles induce the production of interleukin-8 by human cells. Radiat Res 152:57–63.

Nel AE, Diaz-Sanchez D, Ng D, Hiura T, Saxon A. 1998. Enhancement of allergic inflammation by the interaction between diesel exhaust particles and the immune system. J Allergy Clin Immunol 102:539–544.

Romieu I, Barraza-Villarreal A, Escamilla-Nuñez C, Almstrand AC, Diaz-Sanchez D, Sley et al. 2008a. Exhaled breath malondialdehyde as a marker of effect of exposure to air pollution in children with asthma. J Allergy Clin Immunol 121(4):903–909.

Romieu I, Castro-Giner F, Kunzli N, Sunyer J. 2008b. Air pollution, oxidative stress and dietary supplementation: a review. Eur Respir J 31:179–197.

Rosias PP, Dompeling E, Dientener MA, Penning MA, Hendriks HJ, Van Iersel MP, et al. 2000. Childhood asthma: exhaled markers of airway inflammation, asthma control score, and lung function test. Pediatr Pulmonol 30:107–114.

Stamler JS, Lamas S, Fang FC. 2001. Nitrosylation: the prototypic redox-based signaling mechanism. Cell 106:675–683.

Steerenberg PA, Nierkens S, Fischer PH, van Loveren H, van Amsterdam JG. 2000. Increased exhaled nitric oxide on days with high outdoor air pollution is of endogenous origin. Eur Respir J 13:334–337.

Stenfors N, Nordhelm C, Salvi SS, Mudway I, Söderberg M, Blomberg A, et al. 2004. Different airway inflammatory responses in asthmatic and healthy humans exposed to diesel. Eur Respir J 23:82–86.

VOLUME 116 | NUMBER 6 | June 2008 • Environmental Health Perspectives