Health Effects of Acid Aerosols on North American Children: Respiratory Symptoms

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We examined the respiratory health effects of exposure to acidic air pollution among 13,369 white children 8 to 12 years old from 24 communities in the United States and Canada between 1988 and 1991. Each child’s parent or guardian completed a questionnaire. Air quality and meteorology were measured in each community for a 1-year period. We used a two-stage logistic regression model to analyze the data, adjusting for the potential confounding effects of sex, history of allergies, parental asthma, parental education, and current smoking in the home. Children living in the community with the highest levels of particle strong acidity were significantly more likely [odds ratio (OR) = 1.66; 95% confidence interval (CI) 1.11–2.48] to report at least one episode of bronchitis in the past year compared to children living in the least-polluted community. Fine particulate sulfate was also associated with higher reporting of bronchitis (OR = 1.65; 95% CI 1.12–2.42). No other respiratory symptoms were significantly higher in association with any of the air pollutants of interest. No sensitive subgroups were identified. Reported bronchitis, but neither asthma, wheezing, cough, nor phlegm, were associated with levels of particle strong acidity for these children living in a nonurban environment. Key words: acid aerosols, air pollution, bronchitis, children, respiratory symptoms. Environ Health Perspect 104:500–505 (1996)

Although there has been much public and scientific debate about acid rain and its precursors, the focus in the past was largely on ecological effects and secondary health effects related to mobilization of toxic metals. Concern about the possible adverse human health effects resulting from exposure to acid aerosols has been more recent (1,2).

Experimental studies of sulfur dioxide and total suspended particulates have shown adverse health effects in both animals (3) and human subjects (4). In epidemiologic studies, particulate pollution has been associated with decrements in lung function (5), higher prevalence of respiratory symptoms and illness (6–8), and higher rates of hospitalizations (9,10) and mortality (11), even at the levels currently found in many urban areas.

Fine particles, which penetrate deeply into the lungs, are produced by direct emissions from combustion sources or atmospheric reactions of gaseous pollutants. These fine particles are often very acidic. The observed health effects associated with particulate pollution may be attributable at least in part to the acidity of the fine particles (12,13). Previous population-based studies lacked direct measures of ambient aerosol acidity, but recent improvements in atmospheric monitoring techniques have made it possible to directly measure particle strong acidity (14).

In children from the Six Cities Study, reported bronchitis and chronic cough were associated with total suspended particles (7) and with particulate matter less than 15 μm in aerodynamic diameter (8). In a later analysis of children from the same six communities plus Kanawha County, West Virginia (15), the city-specific summer-time mean of particle strong acidity was associated with significantly higher risk of bronchitis and chronic cough.

This study was specifically designed to examine the relationship between long-term, intermittent exposure to particulate matter with particle strong acidity and the respiratory health of children (16). In this article, we describe the associations between particle strong acidity and respiratory symptoms.

Methods

Air monitoring. We selected 24 communities on the basis of previously measured sulfate and ozone concentrations and demographic characteristics (17). The study included 18 sites in the United States and 6 sites in Canada to provide a wide range of expected particle strong acidity and ozone levels. Communities were predominantly suburban or rural with homogeneous, relatively stable populations and no major local sources of air pollution.

Air pollution measurement methods and calibration procedures are described in an accompanying paper (17). Air pollution monitoring began at each site before the summer high acid–ozone exposure period and continued for at least 11 months. Particulate pollutants were sampled for 24 hr every other day. Inhalable particulate matter with an aerodynamic diameter >10 μm (PM_{10}) was sampled using a Harvard Impactor (18). Fine particulate matter with an aerodynamic diameter >2.1 μm (PM_{2.1}) was sampled using a glass impactor/filter pack system. Fine particle strong acidity, fine particle sulfate, and gaseous acids (nitrous and nitric acids) were sampled using the Harvard EPA Annular Denuder System (HEADS) in 21 communities (19–21). In three communities with expected low levels of gas-phase acidity, the Harvard Impactors with ammonia denuders were used. Strong acidity was measured by pH analysis (20) and sulfate was measured by ion chromatography of the extracted particles. Ozone was monitored continuously.

We grouped the 24 communities by the sources of particulate matter and acidity (17). The Sulfate Belt was defined as the 11 sites with high sulfate and acidity concentrations lying generally along the Appalachian Plateau and the Allegheny Mountains. The Transport Region included communities north and east of the Sulfate Belt, which are generally downwind of the principal sulfur emissions in the United States. The remaining communities were designated as the Southern Region.

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nities were grouped as West Coast (three sites) and Background (four sites).

**Respiratory health questionnaire.** The study protocol and methods were approved by the Institutional Review Board at the Harvard School of Public Health and also by the local health department and school board(s) in each community. We contacted principals of selected schools directly and invited them to participate. In communities with fewer than 750 children in the fourth and fifth grades, all schools with at least 10 students in each grade were approached. In larger communities, selection of schools was based either on proximity to the air monitoring station or on random selection, depending on the geography of the school district(s). We distributed standard respiratory health questionnaires to the children at participating schools in September and October of 1988 (eight communities), 1989 (nine communities), and 1990 (seven communities). Children were instructed to have the questionnaire completed by a parent or guardian and to return it to school the following day. Classroom (but not individual) incentives were used to encourage participation, and questionnaires were distributed multiple times over several weeks. The overall response rate (returned questionnaires plus written referrals) was 91%, with a range of 85–99% for the individual communities (Table 1).

Later in the school year, each school was visited again and the pulmonary function of children who had their parents' permission to participate was measured. The results of the analysis of pulmonary function are reported in an accompanying article (23).

**Definition of respiratory symptoms.** Children's respiratory symptoms in the previous year were determined from questionnaire responses defined as follows: 1) asthma: ever diagnosed with asthma by a physician plus asthmatic symptoms present in the past year; 2) attacks of wheezing: episodes of shortness of breath with wheezing and/or episodes of wheezing after playing hard or exercising; 3) persistent wheeze: wheeze apart from colds and/or wheeze for 3 more days out of the week for a month or longer; 4) chronic cough: cough first thing in the morning for as long as 3 months in a row and/or cough at other times during the day or night for as long as 3 months in a row; 5) chronic phlegm: congestion in the chest or bringing up phlegm other than with colds for as long as 3 months in a row; 6) bronchitis: a chest illness diagnosed as bronchitis; 7) asthmatic symptoms: asthma, attacks of wheezing, and/or persistent wheeze; and 8) bronchitic symptoms: chronic cough, chronic phlegm, and/or bronchitis. Only reports of symptoms in the past 12 months were considered.

**Statistical methods.** Previously reported analyses of city-specific health outcomes have demonstrated a larger between-city variation than would be predicted by interindividual variation (7,8). In the present study, we used a two-step analysis to correct for any excess between-city variability. In the first step, city-specific–adjusted logits of symptom prevalence were calculated from a logistic regression model for the respiratory symptom of interest controlling for sex, child's history of diagnosed allergies, parental history of diagnosed asthma (for either biological parent), parental post-high school education (for either parent), and current smoking in the home. In the second step, these city-specific–adjusted logits were regressed against the city-specific annual mean air pollution concentrations using weights inversely proportional to the sum of the between-city and within-city variances of the adjusted logits. Analyses were done using the SAS software (SAS Institute, Inc., Cary, North Carolina). We scaled the regression coefficients from the second step to the range of the city-specific means for the air pollutant of interest (highest to lowest; Table 1) and expressed them as odds ratios (OR) and 95% confidence intervals (95% CI).

To assess the consistency of the evidence and to search for sensitive subgroups, the analyses were repeated stratifying for sex; history of a severe chest illness before the age of 2; use of a humidifier in the home; lung function in the lowest quartile of forced vital capacity (FVC); current exposure to environmental tobacco smoke at home; exposure to maternal smoking during pregnancy; year of study; country; and eastern versus western cities.

**Results**

The city-specific ranges in the annual pollutant means (Table 1) were 51.9 nmol/m³ for particle strong acidity, 6.8 µg/m³ for sulfate particles, 14.9 µg/m³ for respirable particulate matter (PM₂.₅), and 17.3 µg/m³ for inhalable particulate matter (PM₁₀). The gaseous acids (HONO and HNO₃) were measured in all but three communities and together had a range of 111 nmol/m³. City-specific mean particle strong acidity was moderately correlated with PM₁₀ (Pearson's correlation coefficient (r) = 0.47), strongly correlated with sulfate (r = 0.90) and PM₂.₅ (r = 0.82), and essentially uncorrelated with gaseous acids (r = 0.07). Three exposure parameters were considered for the annual mean ozone concentration: the average maximum 1-hr mean (range 46 ppb), the average daytime 8-hr (1000–1800 hr local time) mean (range 39 ppb), and the average daily 24-hr mean (range 19 ppb). All three ozone parameters were highly correlated across the 24 communities with Pearson correlation coefficients ranging from 0.74 to 0.98. By design, city-specific mean particle strong acidity was only weakly correlated with the three ozone parameters; the strongest correlation was with the average 1-hr maximum ozone concentration (r = 0.37). A total of 17,008 children were eligible to participate in the study, of whom 15,523 children (91%) returned a questionnaire. Children who were reported to have cystic fibrosis, a history of a chest

| Table 1. Summary of city-specific annual mean air pollution concentrations: 24 communities, United States and Canada, 1988–1991 |
| Pollutant | Grand mean | SD | Minimum | Maximum | Range |
|-----------|------------|----|---------|---------|-------|
| Particulate matter |           |    |         |         |       |
| Inhalable (µg/m³) | 23.8 | 5.0 | 15.4 | 32.7 | 17.3 |
| Respirable (µg/m³) | 14.5 | 4.2 | 5.8 | 20.7 | 14.9 |
| Sulfate particles (µg/m³) | 4.7 | 2.2 | 0.7 | 7.4 | 6.8 |
| Particle strong acidity (nmol/m³) | 27.5 | 16.2 | 0.0 | 51.9 | 51.9 |
| Gaseous pollutants | | | | | |
| Ozone, 1-hr max. (ppb) | 46.5 | 8.5 | 26.9 | 72.5 | 45.6 |
| Ozone, 8-hr daytime avg. (ppb) | 38.4 | 7.5 | 21.0 | 60.4 | 39.4 |
| Ozone, 24-hr avg. (ppb) | 27.8 | 5.1 | 18.3 | 34.8 | 18.5 |
| Sulfur dioxide (ppb) | 4.8 | 3.5 | 2.0 | 12.9 | 12.7 |
| Ammonia (ppb) | 1.3 | 1.4 | 0.1 | 5.8 | 5.7 |
| Nitric acid (ppb) | 0.6 | 0.4 | 0.1 | 1.4 | 1.3 |
| Nitric acid (ppb) | 0.9 | 0.4 | 0.3 | 2.1 | 2.7 |
| Acidity | | | | | |
| Particle strong acidity (nmol/m³) | 31.1 | 14.0 | 8.5 | 51.9 | 43.4 |
| Gaseous acids (nmol/m³) | 59.4 | 28.4 | 22.7 | 134.0 | 111.3 |
| Total (nmol/m³) | 90.6 | 33.9 | 38.0 | 150.5 | 112.5 |

*Excluding Eggert, Ontario; Yorkton, Saskatchewan; and Aberdeen, South Dakota.

*ppb = 40.9 nmol/m³.

Gaseous acids = nitric + nitric acid.
Table 2. Distribution of covariates used in the respiratory symptoms model by community: 13,369 children, 24 communities, United States and Canada, 1988–1991

| Site code       | Participation (%) | No. of children | Females (%) | Allergy history (%) | Parental asthma (%) | Parental post-high school education (%) | Current smoking in the home (%) |
|-----------------|-------------------|-----------------|-------------|--------------------|---------------------|----------------------------------------|-------------------------------|
|                 |                   |                 | Excluded    | Included           |                     |                                        |                               |
| Sulfate Belt    | Hendersonville, TN| HEN 85          | 59          | 504                | 56                  | 40                       | 13                           | 68                           | 55                           |
|                 | Oak Ridge, TN     | OAK 96          | 119         | 540                | 46                  | 41                       | 15                           | 83                           | 42                           |
|                 | Morehead, KY      | MOR 93          | 48          | 393                | 50                  | 30                       | 14                           | 52                           | 61                           |
|                 | Blacksburg, VA    | BLB 94          | 124         | 666                | 48                  | 37                       | 16                           | 74                           | 37                           |
|                 | Charlottesville, VA| CHV 91          | 171         | 522                | 46                  | 24                       | 12                           | 45                           | 53                           |
|                 | Zanesville, OH    | ZAN 90          | 122         | 647                | 51                  | 26                       | 16                           | 50                           | 61                           |
|                 | Athens, OH        | ATH 88          | 82          | 609                | 53                  | 23                       | 13                           | 69                           | 52                           |
|                 | Parsons, WV       | PAR 91          | 50          | 580                | 51                  | 28                       | 13                           | 43                           | 56                           |
|                 | Uniontown, PA     | UNT 94          | 63          | 584                | 48                  | 24                       | 10                           | 46                           | 62                           |
|                 | Penn Hill, PA     | PEN 90          | 119         | 566                | 49                  | 32                       | 11                           | 80                           | 44                           |
|                 | State College, PA | STC 91          | 97          | 601                | 51                  | 31                       | 16                           | 89                           | 30                           |
| Transport Region| Leamington, ON    | LEM 93          | 111         | 586                | 51                  | 30                       | 10                           | 54                           | 55                           |
|                 | Newtown, CT       | NEW 92          | 50          | 458                | 52                  | 37                       | 16                           | 89                           | 38                           |
|                 | Egbert, ON        | EGB 90          | 76          | 618                | 49                  | 37                       | 13                           | 63                           | 62                           |
|                 | Pembroke, ON      | PMB 96          | 55          | 530                | 49                  | 33                       | 9                            | 53                           | 64                           |
|                 | Dunnville, ON     | DUN 89          | 37          | 442                | 50                  | 29                       | 15                           | 51                           | 58                           |
|                 | South Brunswick, NJ| SBK 88         | 131         | 329                | 48                  | 42                       | 11                           | 79                           | 46                           |
| West Coast      | Simi Valley, CA   | SIM 88          | 129         | 539                | 49                  | 31                       | 17                           | 78                           | 40                           |
|                 | Livermore, CA     | LIV 84          | 102         | 582                | 52                  | 46                       | 18                           | 87                           | 36                           |
|                 | Monterey, CA      | MTK 90          | 92          | 642                | 52                  | 32                       | 17                           | 91                           | 29                           |
| Background      | Springdale, AK    | SPG 91          | 58          | 506                | 53                  | 38                       | 13                           | 50                           | 61                           |
|                 | Aberdeen, SD      | ABD 96          | 71          | 676                | 51                  | 26                       | 11                           | 77                           | 49                           |
|                 | Yorkton, SK       | YOR 95          | 62          | 628                | 47                  | 39                       | 10                           | 58                           | 58                           |
|                 | Penticton, BC     | PCT 95          | 106         | 621                | 47                  | 33                       | 13                           | 65                           | 52                           |
|                 | All 24 communities| 91              | 2154        | 13369              | 50                  | 33                       | 13                           | 67                           | 50                           |

Table 3. Adjusted prevalence of respiratory symptoms by community and symptom: 13,369 children, 24 communities, United States and Canada, 1988–1991*  

| Asthma symptoms (%) | Bronchitic symptoms (%) |
|---------------------|-------------------------|
| Asthma              | Bronchitis              |
| Attacks of wheeze   | Chronic cough           |
| Persistent wheeze   | Chronic phlegm          |
| Any asthmatic       | Any bronchitic          |

*SAdjusted for sex, history of allergies, parental asthma, parental education, and current smoking in the home. See text for definition of symptoms.
operation, serious chest injury, or heart disease, or who were reported to have received oxygen for more than 2 weeks as newborns were excluded (n = 790). Other exclusions were based on age (not 8–12 years; n = 14), race (not white; n = 1013), and missing information on covariates of interest (n = 337). A total of 13,369 children in 24 communities were included in this analysis.

Overall, half (50%) of the subjects were girls, 33% had a history of diagnosed allergies, 13% had at least one parent with a history of asthma, 67% had at least one parent with some post-high school education, and 50% were currently exposed to environmental tobacco smoke at home. The prevalence of these factors varied by community (Table 2). After adjusting for these factors, the community-specific prevalence of asthma was 3–11%; attacks of wheeze, 5–11%; persistent wheeze, 4–12%; any asthmatic symptoms, 7–16%; bronchitis, 3–10%; chronic cough, 3–9%; chronic phlegm, 1–5%; and any bronchitic symptoms, 8–15% (Table 3).

Particle strong acidity was associated with significantly higher reporting of bronchitis in the past year (Fig. 1 and Table 4; OR = 1.66, 95% CI, 1.11–2.48 for the range of 52 nmol/m³), as was annual mean sulfate (OR = 1.65, 95% CI, 1.12–2.42 for the range of 7 µg/m³).

None of the particulate pollutants was associated with significantly higher reporting of asthma, attacks of wheeze, persistent wheeze, chronic cough, or chronic phlegm (Table 4). There was a significant inverse association between chronic cough and PM₁₀. Gaseous acids were associated with a significantly higher risk of asthma (OR = 2.00; 95% CI, 1.14–3.53) and showed a positive association with higher reporting of attacks of wheezing, persistent wheeze, and any asthmatic symptoms (Table 4). Ozone was not associated with a significantly higher risk of any of the respiratory symptoms of interest, regardless of the averaging time used (Table 4). There was a suggestion of a higher risk of chronic cough associated with 24-hr average ozone (OR = 1.29; 95% CI, 0.87–1.91). Ozone for all three averaging times showed some association with higher reporting of bronchitis.

The association between particle strong acidity and bronchitis was similar when the sample was stratified by sex (Fig. 2). The association was sensitive to the inclusion of the Canadian sites, which tended to have low bronchitis prevalence and low to moderate particle strong acidity. Thus, much weaker associations were found when the sample was restricted to U.S. cities or cities studied in the second year, which were predominantly U.S. cities (Fig. 2). No subgroups appeared to be markedly more sensitive to particle strong acidity than the overall sample of children with respect to their risk of bronchitis (Fig. 3). The risk of bronchitis among children who were in the lowest quartile of FVC was not associated with level of particle strong acidity.

**Discussion**

This study was designed to determine whether long-term exposure to acidic aerosols, estimated by annual mean concentrations, was associated with differences in reported respiratory symptoms in children. The data from this study indicate that adjusted prevalence of bronchitis in children aged 8–12 years was significantly associated with particle strong acidity concentrations. Asthma, persistent wheeze, chronic cough, and chronic phlegm were not significantly associated with higher levels of acid aerosols. No sensitive subgroups were identified.

These results are consistent with earlier reports from the Six Cities Study (7,8,15), which noted higher rates of reported bronchitis, but not asthma or wheeze, in association with total suspended particles (7), with particulate matter < 15 µm in aerodynamic diameter (8), or with levels of particle strong acidity (15). In these earlier studies, reporting of chronic cough was higher, but not significantly so, in association with these pollutants.

Prior controlled exposure studies of animals and humans provide support for a relationship between acid aerosols and bronchitis. Short-term exposures to acid aerosols have been shown to result in alterations of mucociliary clearance in rabbits, mice, donkeys, and humans (24). Thus it is reasonable to suspect that chronic exposure to moderate levels of acidic air pollution may affect children's nonspecific defense mechanisms adversely, increasing their risk of respiratory illness.
Table 4. Association of air pollutants with respiratory symptoms by symptom and pollutant: 13,369 children, 24 communities, United States and Canada, 1988–1991

| Pollutant                        | Asthma | Attacks of wheeze | Persistent wheeze | Any asthmatic symptoms | Bronchitis | Chronic cough | Chronic phlegm | Any bronchitic symptoms |
|----------------------------------|--------|-------------------|-------------------|------------------------|------------|--------------|-----------------|------------------------|
| **Particle strong acidity**      | OR     | 0.71              | 0.79              | 0.83                   | 0.81       | 1.66         | 0.92            | 0.95                   |
| (52 nmol/m³)                     | 95% CI | 0.43–1.16         | 0.59–1.06         | 0.60–1.15              | 0.60–1.10  | 1.11–2.48    | 0.64–1.31        | 0.60–1.49              | 0.92–1.53              |
| **Sulfate particles**            | OR     | 0.80              | 0.93              | 0.96                   | 0.91       | 1.65         | 1.04            | 1.29                   |
| (6.8 µg/m³)                      | 95% CI | 0.50–1.29         | 0.70–1.24         | 0.70–1.31              | 0.67–1.24  | 1.12–2.42    | 0.74–1.47        | 0.84–1.96              | 1.01–1.61              |
| **Respirable particulate matter**| OR     | 0.72              | 0.82              | 0.80                   | 0.79       | 1.50         | 0.82            | 1.02                   |
| (14.9 µg/m³)                     | 95% CI | 0.40–1.28         | 0.58–1.15         | 0.55–1.16              | 0.55–1.12  | 0.91–2.47    | 0.54–1.23        | 0.61–1.73              | 0.90–1.46              |
| **Inhalable particulate matter** | OR     | 0.77              | 0.79              | 0.75                   | 0.75       | 1.50         | 0.69            | 1.05                   |
| (17.3 µg/m³)                     | 95% CI | 0.44–1.35         | 0.57–1.08         | 0.53–1.07              | 0.54–1.05  | 0.93–2.43    | 0.46–0.99        | 0.54–1.72              | 0.90–1.43              |
| **Gaseous acids**                | OR     | 2.00              | 1.40              | 1.34                   | 1.34       | 1.18         | 1.02            | 1.11                   |
| (2.7 ppb)                        | 95% CI | 1.14–3.53         | 0.89–1.99         | 0.87–2.05              | 0.91–1.96  | 0.67–2.08    | 0.64–1.61        | 0.90–2.43              | 0.80–1.53              |
| **Total acid**                   | OR     | 1.64              | 1.26              | 1.24                   | 1.24       | 1.43         | 0.98            | 1.40                   |
| (114 nmol/m³)                    | 95% CI | 0.98–2.71         | 0.92–1.72         | 0.86–1.79              | 0.89–1.77  | 0.89–2.20    | 0.66–1.45        | 0.91–2.15              | 0.90–1.55              |
| **Max. ozone, 1-hr avg. (45.6 ppb)** | OR | 1.02 | 0.99 | 0.90 | 0.89 | 1.49 | 1.13 | 1.12 | 1.17 |
| **Sulfur dioxide**               | OR     | 1.05              | 1.07              | 1.19                   | 1.16       | 1.56         | 1.02            | 1.55                   |
| (12.7 ppb)                       | 95% CI | 0.57–1.93         | 0.75–1.55         | 0.80–1.73              | 0.80–1.68  | 0.95–2.56    | 0.66–1.58        | 1.01–2.37              | 0.98–1.71              |

*Symptoms adjusted for sex, history of allergies, parental asthma, parental education, and current smoking in home. Odds ratio (OR) represents relative risk over the range of city-specific air pollution; CI, confidence interval.

The higher reporting of bronchitis, in contrast to asthma, persistent wheeze, or chronic cough, is consistent with an increased susceptibility to illness in association with particle strong acidity. Alternatively, there may be more misclassification of relatively subjective symptoms like cough and wheeze in contrast to diagnosed bronchitis. Such misclassification may have limited our ability to observe associations of symptoms with air pollution.

Although these data indicate an association between particulate air pollution, specifically particle strong acidity, and bronchitic symptoms, there is no evidence for higher reporting of asthmatic symptoms associated with these pollutants. Similar results in which particulate air pollution was associated with bronchitic symptoms but not with any indicator of asthma have been reported in the Six Cities Study (7, 8) for the United States and in German studies (25). Children in Leipzig with high sulfur dioxide and particle exposures were compared to children in Munich with much lower exposures to these pollutants. Bronchitic symptoms were elevated in the high sulfur dioxide/particle exposures, but asthmatic symptoms were decreased in Leipzig, the community with higher particle and sulfur dioxide pollution. The authors suggested that asthmatic symptoms were associated with higher ozone concentrations in Munich. However, in the current data the association between asthmatic symptoms and measures of chronic ozone exposure were very slight. No association was found between ozone and respiratory symptoms in the Six Cities Study (7,8).

The observed association of asthma and asthmatic symptoms with gaseous acids must be interpreted with caution because this association appears to be driven by only one community. The annual mean for gaseous acid in Simi Valley was one-third higher than the mean for the next highest community (17), and Simi Valley also had the second highest rate of reported asthma. If Simi Valley is excluded from consideration, the association between gaseous acid levels and asthma is no longer significant.

There appears to be no evidence that the prevalence of asthma or asthmatic symptoms in children is associated with chronic exposure to particulate, sulfur oxide, or ozone air pollution. This does not imply that acute air pollution episodes do not trigger or exacerbate asthmatic attacks, as has been shown in multiple studies. However, air pollution does not appear to contribute to the increased prevalence of new cases of asthma, as is often claimed in the popular press.
In this study, at least 11 months of air monitoring data were used to calculate annual means of pollutants as a surrogate for lifetime exposure. The validity of such a monitoring scheme depends on the representativeness of the selected year's aerometrics. Brook and Spengler (26) have examined historical aerometric data and concluded that community air pollution levels have been relatively stable over these children's lifetimes and that the relative rankings of the communities with respect to particle strong acidity, ozone, and PM$_{10}$ were not likely to have changed. Although it is possible that the children differed in their personal activity and exposure patterns between communities, these differences were not likely to be related to annual means of acid air pollution.

Particle strong acidity, sulfate, and fine particulate matter are all formed in secondary reactions of the emissions from fossil fuel combustion. They have similar regional and temporal distributions. The strong correlations of several pollutants in this study, especially particle strong acidity with sulfate ($r = 0.90$) and PM$_{2.5}$ ($r = 0.82$), make it difficult to distinguish the agent of interest. This study was designed to have a low correlation of particle strong acidity with ozone, and although high ozone was positively associated with reporting of bronchitis, the association was not statistically significant.

The sampled children in each community were selected to have a narrow range of age and race. Communities also were selected to have similar demographic characteristics. We adjusted for differences among communities in the distributions of sex, child's allergy history, parental education, parental asthma, and current smoking in the home in the first stage of the two-stage model. No evidence of effect modification was observed among these children. The association of bronchitis with particle strong acidity was similar regardless of sex, year of study, country, home characteristics, or child's health history. The only exception was for children in the lowest quartile of FVC, who had no association between a higher risk of bronchitis and particle strong acidity.

These data indicate that chronic exposure to acid aerosol pollution may have observable negative consequences for the health of children. Although the long-term consequences of bronchitis in these children remain unclear, respiratory illnesses in childhood may be a risk factor for chronic obstructive disease. These children also may be at a higher risk from other environmental and occupational exposures later in life.

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