Particulate Air Pollution and Respiratory Disease in Anchorage, Alaska

Mary Ellen Gordian,[1] Halûk Özkaynak,[2] Jianping Xue,[2] Stephen S. Morris,[1] and John D. Spengler[2]

[1]Department of Health and Human Services, Municipality of Anchorage, Anchorage, AK 99519–6650 USA; [2]Department of Environmental Health, Harvard School of Public Health, Boston, MA 02115 USA

This paper examines the associations between average daily particulate matter less than 10 µm in diameter (PM_{10}) and temperature with daily outpatient visits for respiratory disease including asthma, bronchitis, and upper respiratory illness in Anchorage, Alaska, where there are few industrial sources of air pollution. In Anchorage, PM_{10} is composed primarily of earth crustal material and volcanic ash. Carbon monoxide is measured only during the winter months. The number of outpatients visits for respiratory diagnoses during the period 1 May 1992 to 1 March 1994 were derived from medical insurance claims for state and municipal employees and their dependents covered by Aetna insurance. The data were filtered to reduce seasonal trends and serial autocorrelation and adjusted for day of the week. The results show that an increase of 10 µg/m^3 in PM_{10} resulted in a 3–6% increase in visits for asthma and a 1–3% increase in visits for upper respiratory diseases. Winter CO concentrations were significantly associated with bronchitis and upper respiratory illness, but not with asthma. Winter CO was highly correlated with automobile exhaust emissions. These findings are consistent with the results of previous studies of particulate pollution in other urban areas and provide evidence that the coarse fraction of PM_{10} may affect the health of working people. *Key words:* asthma, carbon monoxide, morbidity, outpatient visits, particulate pollution, PM_{10}. *Environ Health Perspect* 104:290–297 (1996)

Recent studies have reported that particulate pollution in ambient air is associated with increased mortality (*1–3*) and morbidity (*4,5*). Studies have been done in cities where the primary source of particulate pollution is combustion products. Studies of areas with high industrial particulate pollution show increases in asthma symptoms and hospital admissions. The present study examined the association between particulate pollution and the incidence of acute respiratory diseases as measured by outpatient visits for specific respiratory diagnoses in an area without significant industrial pollution, Anchorage, Alaska. Anchorage is a city of 240,000 people located in a "bowl" surrounded by mountains and sea coast. Wood smoke is not a major contributor to particulate pollution in this area because wood is not commonly used as fuel due to its high cost. Electric power plants are fueled by natural gas. The main sources of particulate pollution are unpaved roads, road sanding, vehicular traffic, and ashfall from volcanic eruptions.

On 18 August 1992, during the period of this study, Mt. Spurr, 60 miles west of Anchorage, erupted and rained ash on the city. Hourly measurements of particulate matter with aerodynamic diameter less than 10 µm (PM_{10}) reached a maximum level of 3000 µg/m^3. The 24-hr average concentration was 565 µg/m^3 on the day after the eruption. Computer-controlled scanning electron microscopy (CCSEM) was used to determine the composition of particles from 10 random samples taken before and after the volcano erupted (*6*). Over 80% of the particle mass was between 2.5 and 10 µm. The composition was mainly silica and silica–aluminum. CCSEM showed that less than 5% by weight of the filter mass was carbonaceous particles. This is consistent with source apportionment studies by chemical mass balance done 7 years previously (*7*), which concluded that more than 85% of the total suspended particulates (TSP) in Anchorage was earth crustal material. Size-fractionated mass measurements below 15 µm were made in Anchorage by the U.S. EPA in the early 1980s using dichotomous samplers. Historic data collected by EPA during 1983 also suggest a high coarse-particle mass fraction. Average fine [aerodynamic diameter (d_a) < 2.5 µm] to coarse (2.5 µm < d_a < 15 µm) particle mass ratios in the summer of 1983 were 0.14 ± 0.05. The overall median ratio of fine to coarse PM_{15} was calculated to be 0.26. This is in distinct contrast with the 0.4 to 0.7 $PM_{2.5}$/ PM_{15} ratios reported for 6 communities in the lower 48 states by Spengler and Thurston in 1983 (*8*). We investigated the relationship between respiratory illness treated on an outpatient basis and ambient particulate PM_{10} pollution using a health insurance database. Working people and their dependents, generally considered a healthy group, are the sample population used in this analysis. The sample size was approximately 6% of the population of the city of Anchorage.

Methods

Database

Particulates are measured daily as 24-hr PM_{10} samples using an Anderson head sampler at a central location in Anchorage, the Gambell site. Measurements are made intermittently at two other sites within the city. The Pearson correlation coefficient between sites ranges from 0.76 to 0.81. The Gambell site is located close to a major highway, and PM_{10} concentrations are 43–76% higher than concentrations measured at other sites. Other than PM_{10}, few pollutants are routinely monitored in Anchorage. Carbon monoxide monitoring is conducted hourly and daily between October and March. CO is routinely monitored at five locations in the Anchorage area. Only two sites exceeded the 9 ppm CO standard during the period of this study: the Seward Highway site, located four blocks from the Gambell PM_{10} monitoring site, and the Garden site, a residential area about 2 miles from the Gambell PM_{10} monitoring site. Measurement of criteria pollutants, sulfur dioxide, nitrogen dioxide, and ozone are only done occasionally as they remain quite low.

Available 8-hr maximum CO concentrations measured at each of the five CO monitoring sites in Anchorage between 1 October 1992 and 31 March 1993 and between 1 October 1993 and 31 March 1994 were obtained from the Municipality of Anchorage Environmental Services Division. These data were processed to generate the average 8-hr maximum CO concentration for each day, which was then used in the analysis.

Daily claims made for outpatient visits for respiratory illness were obtained from

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Address correspondence to M. E. Gordian, PRO-West, 721 Sesame Street, Suite 1A, Anchorage, AK 99503 USA.

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Aetna Insurance Company, which processes the health insurance claims for both employees of the State of Alaska and employees of the Municipality of Anchorage. Both groups have comprehensive health insurance with low deductibles for employees and dependents. We analyzed data from a 22-month period from 1 May 1992 to 1 March 1994. All outpatient visits that were submitted to insurance, whether they occurred in doctors' offices or in emergency rooms, were captured by this method. The diagnosis code recorded for the visit was based on the International Classification of Diseases 9th Revision (ICD–9) coding. ICD–9 codes were grouped to identify upper respiratory problems such as sore throat, earaches, sinusitis, rhinitis, and other nonspecific upper airway problems. This whole group of illnesses is referred to as upper respiratory illness (URI). The second group, referred to as bronchitis, includes lower airway diseases such as bronchitis, tracheitis, and nonspecific cough. Pneumonia was not included, as it is frequently treated on an inpatient basis. The third respiratory category, referred to as asthma, included all reactive airway disease, bronchospasm, and asthma ICD–9 codes. Diarrhea, a common diagnosis presumably unrelated to air pollution, was recorded as a control diagnosis. The ICD–9 codes used were: for asthma, 519.1, 493.9, 493.0, 495; for bronchitis, 466.0, 490, 490.0, 491.0, 491.1, 786.2; for chronic obstructive pulmonary disease (COPD), 491.2, 491.9, 492.0, 492.8, 496, 506.4; for congestive heart failure (CHF), 428.0, 428.1, 402.01, 402.11, 402.91, 440.9, 398.91, 429.1, 429.4, 429.9; for diarrhea, 558.9; and for upper respiratory illness (URI), 077.2, 460, 461, 461.0, 461.1, 461.2, 461.3, 461.8, 461.9, 462, 465, 465.0, 465.9, 472, 472.0, 472.1, 472.2, 473.0, 473.1, 473.2, 473.3, 477, 477.1, 477.9, 478.2, 478.8.

Reiterations of the insurance data collection were done until we were confident of a stable claims report. Only visits where both patient and provider had an Anchorage zip code were included in the analysis. There were approximately 11,000 State of Alaska and 3000 municipal employees and dependents eligible for health insurance in Anchorage during the time of the study.

Analytical Methods

Daily outpatient visits, temperature, and PM_{10} series exhibit seasonal cycles, some of which are common. Unless adjusted for long-term cycles, shared seasonal or monthly cycles among outpatient visits and environmental variables could confound results. Adopting the technique used in Kinney and Özkaynak (*8*), a weighted 19-day moving average filter developed by Shumway (*9*) was used to detrend the pollution and meteorological series. The method involves subtracting the weighted moving average of each variable (X_t) from itself on each observation. In other words, the X_t on day $t = i$ is filtered as:

$$X\_F_i = X_i - \sum_{i=-9}^{9} X_i w_i \qquad (1)$$

where w_i is the filter weights shown in Shumway (*9*). This process of filtering removes the long-term cycles but not the short-term cycles (i.e., high frequencies). When a linear filter such as this is applied to both the predictor and predicted variables before regression analysis, linear regression relationships among variables are preserved and can be estimated without bias. In addition, this filter efficiently removes the autocorrelation in the pollution and the outpatient visit series. Autocorrelation functions were examined to detect any remaining temporal structure in the filtered data, and none was found.

We computed descriptive statistics for all the filtered and unfiltered data. We used a generalized linear model procedure to test statistical differences in the daily outpatient visits by day of the week. Cross-correlations between filtered outpatient visits (e.g., for asthma) and filtered PM_{10} were calculated to determine the importance of the relationship between doctors visits and same-day (or lag 0), previous-day (or lag 1) and 2-days prior (or lag 2) PM_{10} measurements. We analyzed the daily outpatient visit (OV) counts and pollution data using time–series and regression modeling techniques implemented with SAS software (SAS Institute Inc., Cary, North Carolina).

Because of low daily counts for some categories of doctors visits (e.g., asthma, bronchitis), we examined two different methods of modeling the pollution–health effect relationships. Both ordinary and Poisson regression models were fitted to filtered outpatient visit, temperature, and pollution data. Consistency of results and normality of model residuals were examined. In all cases, results from Poisson and multiple regression models were almost identical. Moreover, residuals from the multiple regression models were very nearly normally distributed. Consequently, for technical and practical reasons, we chose multiple regression modeling framework in the analysis. Basic analysis involved fitting multiple regression models to four filtered morbidity variables (i.e., doctors visits diagnosed as asthma, bronchitis, diarrhea, and upper respiratory infections) using filtered same-day or previous day PM_{10} and temperature as explanatory variables. The diarrhea category was selected for analysis as a control category. The form of the basic regression model (model I) was:

$$OV\_F = \beta_o + \sum \beta_i X\_F_i + E \qquad (2)$$

where OV_F is the filtered daily outpatient visits, X_F_i is the filtered same-day or previous-day daily temperature and PM_{10} measurements, and E is the error term. Other models were also done. Model 2 added a weekend/weekday indicator variable (W_D) as an additional explanatory variable to Equation 2. Model 3 was a regression specification using as the dependent variable outpatient visits that were both filtered and weekend/weekday adjusted. Specifically, model 3 was written as:

$$OV\_R = \sum \beta_o + \sum \beta_i X\_F_i + E \qquad (3)$$

where:

$$OV\_R = OV\_F - \left(\alpha_o + \alpha_1 W\_D\right) \qquad (4)$$

In models 1–3, same-day temperature and same-day PM_{10} were included. We also ran models with different lags of temperature and PM_{10}. We present results from one of these, Model 4, where previous day's PM_{10} (or lag 1 PM_{10}) instead of same-day PM_{10} is included in the specification. The models were run for all ages combined and separately for three age groups (<10 years, 11–45 years, and 46+). Due to sample size limitations, male and female outpatient visits were combined.

Finally, we also examined potential statistical confounding of results due to other pollutants of health concern and the influence of variations in the PM_{10} composition over time. We included the available wintertime CO measurements independently, as well as jointly with PM_{10} data, in the regression models tested. Potential changes in the seasonal composition of PM_{10} and the influence of the volcanic eruption that occurred on 18 August 1992 were also modeled using nested regression modeling methods. In this case, we estimated separate PM_{10} slopes for winter versus summer seasons and periods strongly influenced by volcanic eruption (18 August 1992–31 December 1992) versus the remaining period less influenced by volcano ash (i.e., 1 May 1992–17 August 17 1992; 1 January 1993–1 March 1994).

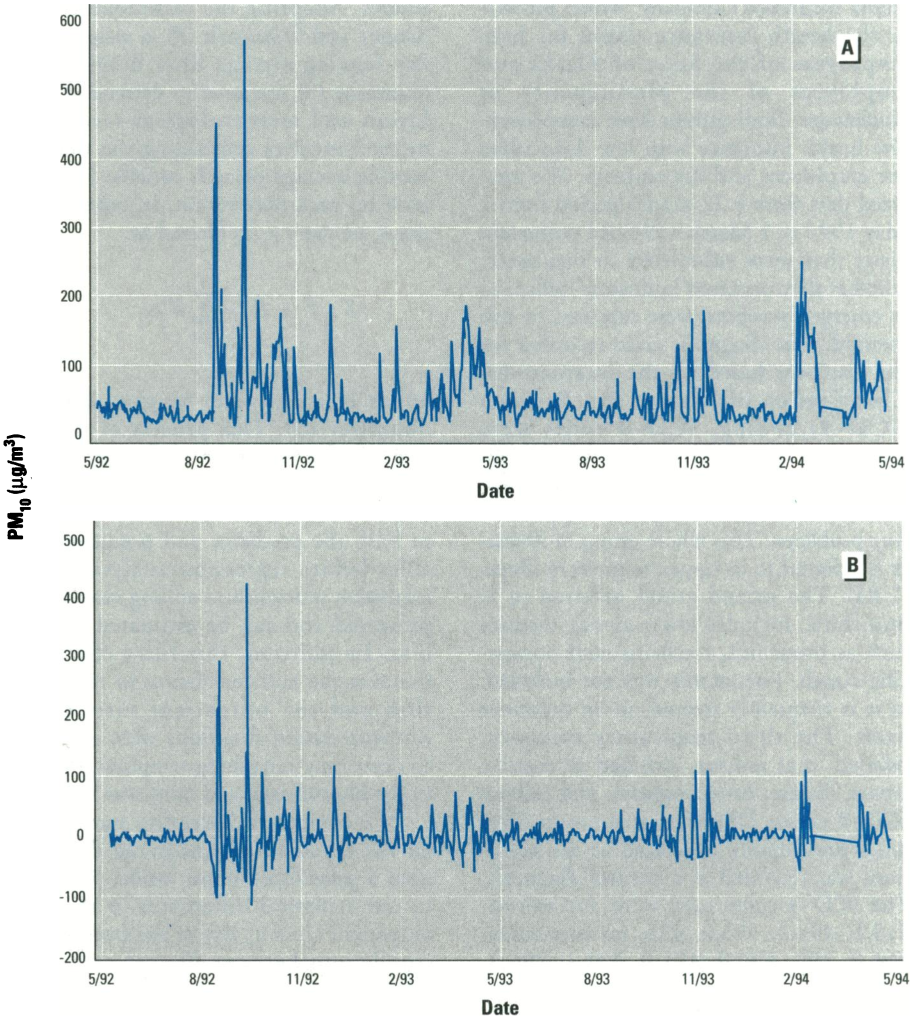

Figure 1. Measurements of PM_{10} (particulate matter <10 μm in diameter) in Anchorage, Alaska (Gambell site). (A) Original data; (B) filtered data.

Results

Figure 1 displays the daily PM_{10} measurements collected at the Gambell site in Anchorage. Both the original and filtered series are shown. The influence of volcanic eruptions on PM_{10} levels during the fall of 1992 are clear. After detrending, long-term cycles and seasonal patterns are no longer apparent. Daily counts for outpatient visits for asthma, bronchitis, and URI are shown in Figures 2–4. Again, the 19-day moving average filter detrends the observations for respiratory illness visits. Because of the relatively young age of the sample population, CHF and COPD visits were infrequent, and no analysis of these was done. Table 1 presents the summary statistics for the analysis variables: temperature, PM_{10}, CO, visits for illnesses of asthma, bronchitis, diarrhea, and URI. Correlation of 8-hr maximum CO measurements among the five different sites was quite high ($\rho \approx 0.8$). Consequently, using the data from all monitors, we calculated the 8-hr maximum CO value in the Anchorage area for each day data were collected. The correlation between daily PM_{10} and daily average maximum CO was found to be small ($\rho \approx 0.15$). Table 1 also provides a breakdown of the statistics by different age categories. Clearly, most of the visits are recorded in the largest age category, 11–45 year olds. Because only active employee insurance records were analyzed, most of the population at risk were under 65 years of age.

Ordinary regression models were run for all outpatient visit categories. Table 2 presents the results for the basic model (model 1) for the three respiratory illness categories: asthma, bronchitis, and URI. All of the estimated PM_{10} regression coefficients were significant for these illness categories. However, a generalized linear model analysis indicated substantial weekend/ weekday differences in the recorded outpatient visits. Because most doctors' offices are closed on the weekends, typical weekend visit counts for all causes of illness were five times lower than during the weekdays. Moreover, there was also a slight difference, though not statistically significant, in the PM_{10} concentrations during weekdays versus weekends. Average PM_{10} concentration on Saturdays and Sundays was about 37 μg/m^3, whereas during weekdays it was around 48 μg/m^3. We suspected that less traffic over the weekend results in slightly lower PM_{10} levels in the city. Consequently, we attempted to control for the differences in the outpatient visits during weekends in two different ways. One way was to add a weekend-versus-weekday dummy or indicator variable to the basic regression model (Eq. 2), which we called model 2. The other way was to adjust for day-of-the-week effect on outpatient visits first, and then run the regression on the residuals with either same-day PM_{10} (model 3) or previous-day PM_{10} value (model 4). The results from these alternative model fits are shown in Table 2. Model 2 results with the weekend/weekday dummy variable do not indicate a significant association between PM_{10} and doctors visits. The reason for nonsignificant findings under model 2 is the induced statistical collinearity between the estimated PM_{10} coefficients and the weekend/weekday (*W_D*) coefficients. Because the underlying reason for reduced outpatient visits over weekend days are different and much more pronounced than those affecting the differences in weekend/weekday PM_{10} concentrations, a separate adjustment of outpatient visits was considered more appropriate. Therefore, a two-stage regression analysis was considered to be the most reliable method with this data set. Models 3 and 4 were both developed as two-stage regression analyses using filtered weekday/weekend adjusted outpatient visits as the dependent variable.

Statistically significant associations were found between both same-day and previous-day PM_{10} (lag 0, lag 1) and asthma visits and between same-day PM_{10} and URI diagnosed outpatient visits based on model 3 and 4 specifications. The statistical association found between lag 1 PM_{10} and visits for asthma was stronger and more significant than the association found between same-day PM_{10} and visits for asthma (Tables 2 and 4). Other lags (i.e., lag 2,3) of PM_{10} were also studied but not found to be significant in the models tested. Using the coefficients from model 3 and model 4, the magnitude of the projected PM_{10} effect on outpatient visits for each 10 μg/m^3 increase in PM_{10} is 2.5–3.5% excess outpatient visits for asthma and 1.2% excess outpatient visits for URI (Table 2).

Next we examined the age dependence of the results by repeating the model 3 analysis for daily visits recorded separately

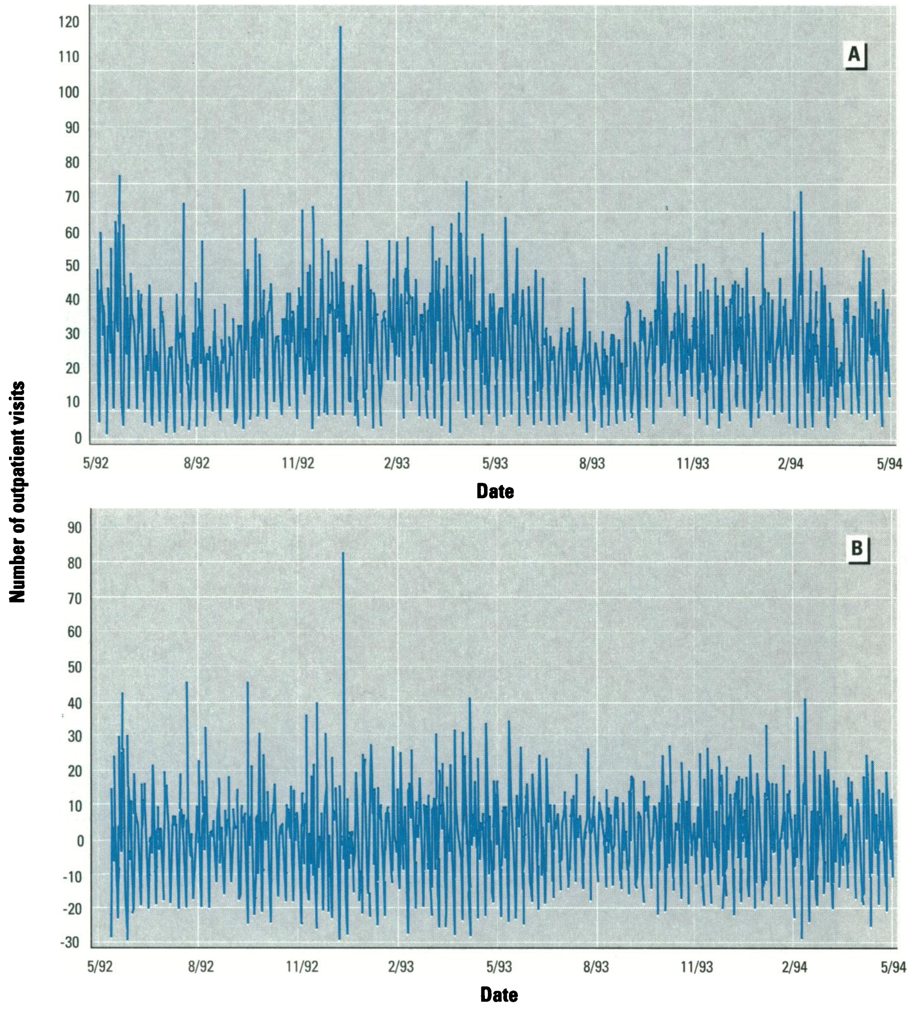

Figure 2. Daily outpatient visits for upper respiratory infections in Anchorage, Alaska. (A) Original data; (B) filtered data.

under the three age categories: <10 years, between 11 and 45 years, and >46 years (but typically less than 65 years). Table 3 presents these regression results. Due to the small number of daily counts, some of the age-specific regression estimates were not significant. For asthma visits, the effect estimate for the 11- to 45-years age group (2.6% excess visits) was not significant (p = 0.14) but similar in magnitude to one previously found for the all ages combined. However, statistically significant associations were found between PM_{10} and URI-related outpatient visits for children under 10 years of age and adults over 46 years of age. The predicted PM_{10} effect on URI visits associated with an increase of 10 µg/m^3 PM_{10} was 1.9% and 1.2%, respectively, in these two age categories. Outpatient visits for diarrhea were not significant either in models 2 or 3.

We examined the association between daily CO and outpatient visits using the regression models (i.e., models 3 and 4). Table 4 presents the estimated regression coefficients for CO from models of outpatient visits for asthma, bronchitis, and URI. These results are based on model 3 specifications. Models with lag 1, 2, or 3 CO variables did not result in statistically significant coefficients. Same-day CO was highly significantly associated with outpatient visits for bronchitis and URI using the available CO series, obtained during fall/winter of 1992–1993. In comparison to the estimated PM_{10} effect, the magnitude of the estimated CO effect on URI and bronchitis outpatient visits seems to be greater. For an increase of 1 ppm (8-hr maximum) CO, it is estimated that doctor visits for bronchitis and URI will rise by 10% and 13%, respectively. The significant associations found between CO and bronchitis and URI are not influenced or confounded by PM_{10}. Models in which both PM_{10} and CO variables were included produced results essentially the same as the single pollutant regression models.

Temperature and Volcano Effects

The temperature coefficient was significant in only one of the models. The estimated coefficient for the filtered temperature variable from the model of CO and temperature on URI was 0.24 (p<0.04). We examined further the temperature and PM_{10}, and temperature and CO relationships, and found those to be weak. We re-ran the PM_{10} regression models with lag 1 temperature instead of the same-day temperature and obtained identical results. We assume that the 19-day weighted Shumway filter adequately removes not only the seasonal trends in the data, but multiday variations in the temperature observations that may influence respiratory diseases and symptoms more than the day-to-day variations in temperature. Variations in temperature may have less effect on health in a young, working population than on a more vulnerable population.

We also examined whether the estimated PM_{10} coefficients were influenced by seasonal or other compositional factors. We ran nested regression models to estimate separate PM_{10} slopes for summer (April–October) versus winter (November–March) seasons and also for the period influenced by the volcano eruption (18 August 1992–31 December 1992) versus the period not expected to be influenced by the Mt. Spurr volcano (1 May 1992–17 August 1992; 1 January 1993–1 March 1994). Table 5 presents the estimated PM_{10} and (lag 1) PM_{10} coefficients obtained from these models. The association found with PM_{10} and asthma does not seem to be influenced much by season. A significant (lag 1) PM_{10} coefficient is estimated for the winter season from models of asthma-related outpatient visits. For URI, because of sample size limitations, PM_{10} coefficient loses its significance when the data set is split by summer and winter season. However, the magnitude of the estimated summer and winter coefficients remain similar to the PM_{10} coefficient estimated from the full data set (see Tables 4 and 5). Interestingly, the period immediately after the volcanic eruption resulted in nonsignificant PM_{10} coefficients in models of asthma and URI outpatient visits. In contrast, the period not affected by the volcanic eruption resulted in statistically significant PM_{10} coefficients. Average PM_{10} concentration during the period influenced by the volcano was around 70 µg/m^3 , whereas the period not affected by the volcano had an average PM_{10} of 40 µg/m^3. The magnitude of the estimated same-day or previous-day PM_{10} effect on doctors visits for asthma, during the period not influenced by the volcano, was about 6%, corresponding to an increase of 10 µg/m^3 PM$_{10}$. Likewise, doctors visits for URI are expected to increase by about 3% corresponding to increase of 10 µg/m^3 PM$_{10}$ during the period not influenced by volcanic activity.

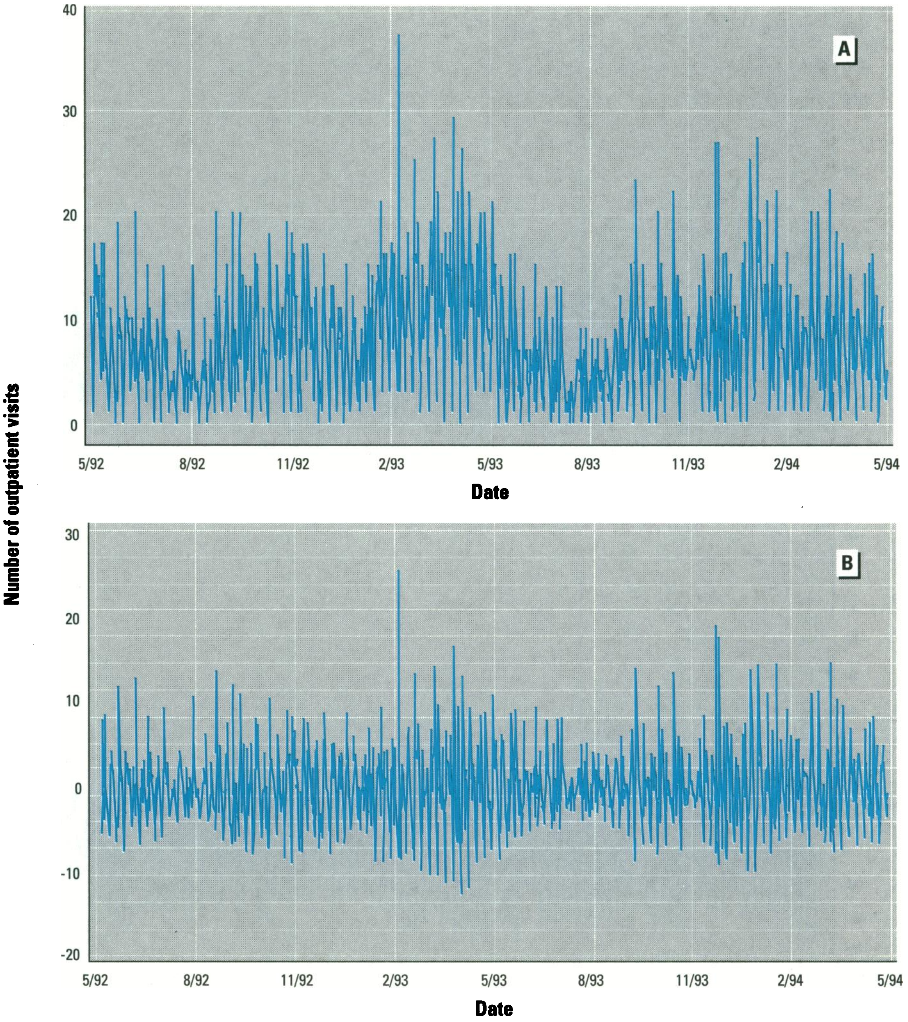

Figure 3. Daily outpatient visits for bronchitis in Anchorage, Alaska. (A) Original data; (B) filtered data.

Discussion

We analyzed 22 months of daily PM$_{10}$, temperature, and daily cause-specific outpatient visit data from Anchorage, Alaska, to study the acute relationship between PM$_{10}$ and respiratory illnesses. The health data were obtained from a large health insurance provider to state and municipal employees in Anchorage. Even though the coverage was only partial (80–90%) and records may have included repeat visits to a doctor by the same individuals, the data set is considered to be representative. Furthermore, we applied conservative statistical methods to control for potential seasonal, weekly, and daily confounders of PM$_{10}$ health effects. In particular, we controlled for potential influences of temperature on daily outpatient visits.

In Anchorage, continuous records for other pollutants such as ozone (O_3), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2) were not available for the period of analysis. It is unlikely that these omitted variables could confound potential associations between PM$_{10}$ and outpatient visits. Limited monitoring data available for these pollutants indicate very low levels [below the National Ambient Air Quality Standards (NAAQS) for O_3, NO_2, and SO_2. SO_2 concentrations measured in 1983–1985 were less than 10% of the NAAQS. The maximum hourly O_3 was 40 ppb, one-third the NAAQS during the 2 years of monitoring in 1983–1985. Although only 6 months of NO_2 data are available for Anchorage, levels of this pollutant did not exceed one-third the NAAQS. SO_2 was measured at the base of the volcano during the eruption, but not in the city of Anchorage. The measurement taken at the base of the volcano 60 miles from Anchorage was considered low for a volcanic eruption (750 tons/day) and unlikely to affect Anchorage.

Two pollutants do occur in Anchorage in significant amounts. They are CO and benzene. A year-long monitoring study of ambient air for volatile organic compounds in Anchorage was completed in 1994. The study showed that the levels of benzene in the winter in Anchorage were higher than the levels reported in any other U.S. city in a national study done in 1987 (*10*). Benzene monitoring was done at the CO monitoring sites and was highly correlated with CO concentrations (ρ ≈ 0.97) (*11*). Both benzene and CO are exhaust emissions of incomplete combustion of Alaskan gasoline, which is high in benzene (5%) and other aromatic compounds. An increase of 1 ppm CO is equivalent to an increase of 3 ppb benzene in Anchorage.

CO measurements at five sites in Anchorage were available for October–March during fall and winter of 1992 and 1993. CO measurements were not correlated with PM$_{10}$ measured at the Gambell site ($R^2 \approx 0.14$). Nevertheless, potential confounding of PM$_{10}$ associations due to CO were examined by running the outpatient visit-pollution models using average maximum CO as an independent exposure variable. Models were also run with both CO and PM$_{10}$ together in model 3 and model 4 specification. Results indicated that CO and PM$_{10}$ associations with outpatient visits are independent of each other. Because CO data are only collected in the cold season, the sample size for the CO models was about half of that used in the PM$_{10}$ models. Therefore, we have less confidence in the associations detected for CO than those found for PM$_{10}$. Nevertheless, the significant associations detected for CO are intriguing. These results suggest that wintertime emissions from automobiles, CO, NO_2, fine particles, and volatile organic compounds (VOCs), may significantly contribute to bronchitis and URI in the Anchorage population. Because CO is a surrogate for many of the vehicular emissions, it is not possible to directly link CO with the inferred respiratory effects. Available epidemiologic data on respiratory effects of CO and VOCs are quite limited. A recent article by Morris et al. (*12*) showed that ambient CO levels were positively associated with hospital admissions for CHF among elderly people in seven large U.S. cities. Ware et al. (*13*) had shown respiratory and irritant health effects associated with ambient VOCs in Kanawha Valley, West Virginia. Both petroleum or auto-related compounds and chemical manufacturing emissions were determined to be the likely source of ambient VOCs and the estimated health effects in that

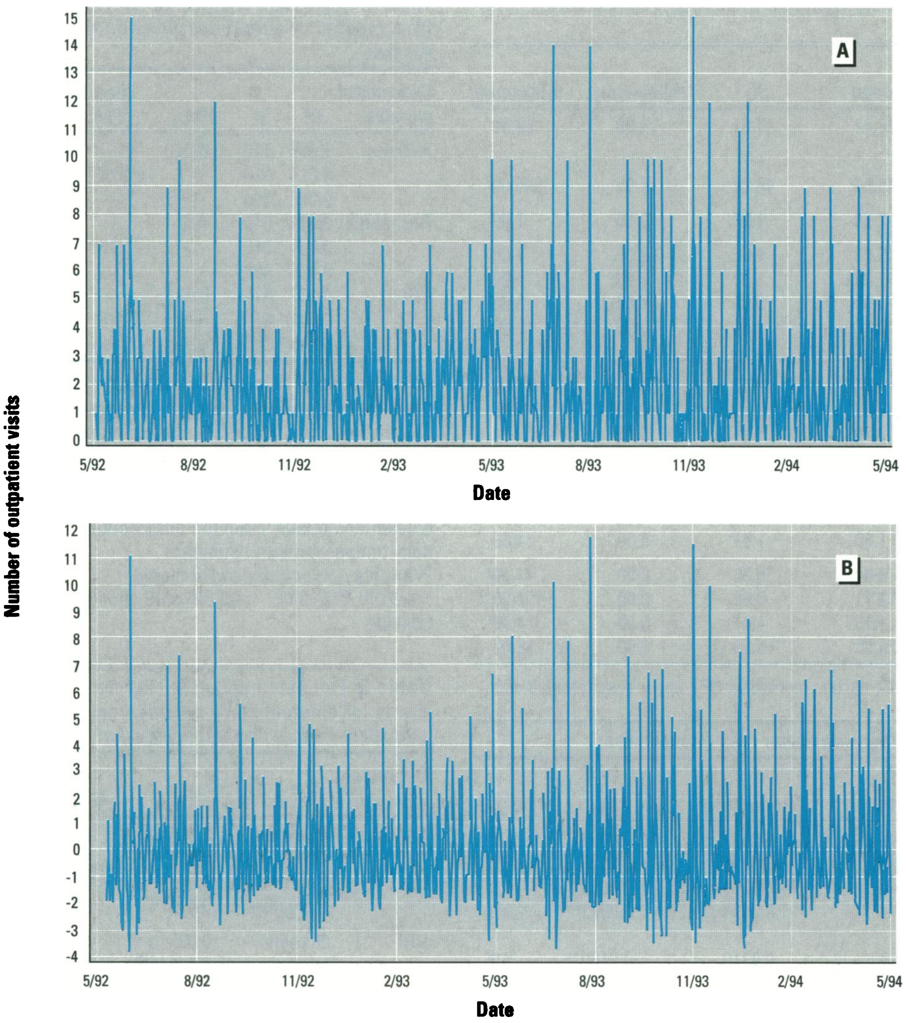

Figure 4. Daily outpatient visits for asthma in Anchorage, Alaska. (A) Original data; (B) filtered data.

community. Analyses of many years of daily mortality and daily air pollution in Los Angeles as well as in Toronto, Canada, have also shown statistically significant associations between CO, NO_2, index of fine particulate or carbonaceous air pollution, and daily total mortality (*14,15*).

We have found statistically significant associations between either the same-day or previous-day PM_{10} and outpatient visits for illnesses due to asthma and URI, in a location where the primary sources of PM_{10} are not combustion or secondary aerosols. Earth crustal and volcanic ash sources are believed to dominate the respirable particle mass in Anchorage. However, the association found between PM_{10} and asthma-related doctors visits seems to be higher and significant only during the period excluding volcanic activity. These findings could be explained by various exposure or source-related factors. After the volcanic eruption, PM_{10} levels exceeded hundreds of micrograms per cubic meter. Anchorage residents were advised to minimize their ambient exposures by staying indoors or limiting their outdoor activities. Businesses closed, work was curtailed, and events were postponed immediately after the eruption. Many people wore dust masks. Consequently, use of ambient PM_{10} concentrations following the period of volcanic eruption can lead to misclassification of personal exposures to PM_{10}. Past studies have shown that potential pulmonary toxicity of volcanic ash may be quite low. Beck et al. (*16*) showed that based on short-term animal bioassays, toxicity of Mt. St. Helens volcanic ash was low and similar to responses to aluminum oxide, a dust considered to be relatively inert. This result is also compatible with the possibility that volcanic ash is not toxic until after mixing with combustion-related fine particles.

Based on our findings, the toxicity of the aerosol mixture in Anchorage seems to be comparable to that inferred from other epidemiologic studies conducted in areas with more typical ambient or urban particulate pollution. For example, previous reports of estimated acute effects associated with a 10 $\mu g/m^3$ increase in daily PM_{10} on respiratory morbidity were: 1) Seattle, Washington (*17*): asthma emergency room visits for <65 year olds were increased by 4% ± 1%; 2) Utah Valley, Utah (*18*): hospital admissions for asthma and bronchitis for children under 5 years were increased by 7.1% ± 3%; 3) Birmingham, Alabama (*19*): hospital admissions for the elderly (>65 years) for COPD were increased by 2.4% ± 0.8% and for pneumonia by 1.8% ± 0.5; 4) Detroit, Michigan (*20*): hospital admissions for the elderly (>65 years) for COPD were increased by 2% ± 0.6% and for pneumonia by 1.1% ± 0.4%; 5) Minneapolis–St. Paul, Minnesota (*21*): hospital admissions for the elderly (>65 years) for COPD were increased by 4.5% ± 1.4% and for pneumonia by 1.6% ± 0.7%; 6) U.S. and European sites combined (*22*): URI increased by 0.7% and asthmatic attacks by 3%.

In comparison, our findings are quite similar in magnitude and indicate a 1–3% increase in outpatient visits for upper respiratory illness and about a 3–6% increase in asthma-related outpatient visits associated with 10 $\mu g/m^3$ increase in daily PM_{10} concentrations. Most of these other investigations, however, have been conducted in urban settings with numerous industrial and vehicular sources of particles, leading some researchers and regulators to suggest that these adverse particle-linked health effects are due to fine particles (<2.5 µm in diameter) generated largely by combustion sources. Because of the high ratio of coarse to fine particles in Anchorage, our analyses is somewhat unique in that it suggests that some morbidity may be related to coarse particles (>2.5 µm in diameter) that are primarily of geologic origin. This is consistent with a study done in rural, western Washington state, where PM_{10} also has a predominantly earth crustal component (*23*). If our results are confirmed through additional studies, they would bear significantly on some of the critical scientific uncertainties facing the U.S. EPA in its ongoing review of the ambient air quality standard for PM_{10}.

We believe that it is important to further examine the robustness of these results by analyzing a longer series of available records for outpatient visits and PM_{10} in Anchorage. It is also important to extend the analysis to more vulnerable segments of the population such as persons 65 years of age and older. The association found between CO and doctors visits for bronchitis and URI clearly needs to be pursued further, particularly in light of the recent findings associating urban CO with con-

Table 1. Descriptive statistics for air pollution and doctors' visits in Anchorage, Alaska, 1 May 1992–1 March 1994

Variable	Age category (years)	n	Mean	SD	Minimum	Maximum
Average temperature (°F)		669	38.44	17.51	-14.00	69.00
PM_{10}		626	45.54	48.81	5.00	565.00
CO		364	2.54	1.24	0.50	7.00
Asthma	<10	669	0.50	1.05	0.00	7.00
	11–45	669	1.22	1.97	0.00	15.00
	>46	669	0.39	0.95	0.00	9.00
	All ages	669	2.12	2.54	0.00	15 00
Bronchitis	<10	669	1.59	2.07	0.00	17.00
	11–45	669	4.54	3.83	0.00	24.00
	>46	669	1.57	1.91	0.00	15.00
	All ages	669	7.70	5.85	0.00	37.00
Diarrhea	<10	669	0.36	0.77	0.00	8.00
	11–45	669	0.81	1.26	0.00	12.00
	>46	669	0.33	0.82	0.00	10.00
	All ages	669	1.50	1.81	0.00	14.00
URI	<10	669	6.64	5.36	0.00	41.00
	11–45	669	13.71	9.86	0.00	104.00
	>46	669	4.15	4.47	0.00	34.00
	All ages	669	24.50	15.65	0.00	116.00

Abbreviations: PM_{10}, particulate matter <10 µm in diameter; CO, carbon monoxide; URI, upper respiratory illness.

Table 2. Regression results for outpatient visits in Anchorage, Alaska for respiratory illness[a]

Dependent variable	Model[b]	R^2	Intercept	*WD*	Temperature	PM_{10}	E(%)
Asthma	1	0.017	-0.0422	—	-0 0077	0.0088**	4.2
	2	0.194	0.6783†	2.3025†	-0.0226	0.0036	NS
	3	0.009	0.0124	—	-0.0171	0.0053*	2.5
	4	0.014	0.0056	—	0 0094	0.0073**	3.5
Bronchitis	1	0.014	0.1191	—	0.0310	0.0180**	2.3
	2	0.378	2.0924†	-7.1426†	0.0154	0.0020	NS
	3	0.003	0.0018	—	0.0006	0.0067	NS
URI	1	0.237	0.2958	—	0.1284	0.0650†	2.7
	2	0.568	6.9938†	-24.2439†	-0.0292	0.0105	NS
	3	0.007	0.0301	—	0.0332	0.0295*	1.2

Abbreviations: PM_{10}, particulate matter < 10 µm in diameter; URI, upper respiratory illness.

[a] All variables are detrended using the Shumway filter (*9*); n = 610. *W_D* , weekend/weekday dummy; *E*, predicted percent change in outpatient visits for each 10 µg/m^3 increase in PM_{10} pollution.

[b] Model definitions: model 1: regression of daily doctors visits on daily PM_{10} and temperature; model 2: regression of daily doctors' visits on daily PM_{10}, temperature, and *W_D*; model 3: regression of weekend/weekday adjusted daily doctors' visits on daily PM_{10} and temperature; model 4: regression of weekend/weekday adjusted daily doctors' visits on previous day's PM_{10} and same-day temperature.

*p<0 05; **p<0.01; †p<0.001; NS, not significant (p>0.05).

Table 3. Regression results by different age groups for respiratory illness in Anchorage, Alaska[a]

Dependent variable	Age category (years)	R^2	Intercept	Temperature	PM_{10}	E(%)
Asthma	10	0.006	-0.0042	-0.0121	0.0012	NS
	11–45	0.005	0.0035	-0.0081	0.0032 (p = 0.14)	2.6 (NS)
	46	0.002	0.0081	0.0031	0.0009	NS
	All ages	0.009	0.0124	-0.0171	0.0053*	2.5
URI	10	0.009	0.0137	0.0025	0.0129*	1.9
	11–45	0.001	-0.1086	0.0072	0.0062	NS
	46	0.008	0.0260	0.0230	0.0102*	11
	All ages	0.007	-0.0301	0.0332	0.0295*	1.2

Abbreviations: PM_{10}, particulate matter < 10 µm in diameter; URI, upper respiratory illness.

[a] All variables are detrended using the Shumway filter (*9*); n = 610. All regressions are weekend/weekday adjusted (filtered) daily doctors visits on (filtered) daily PM_{10} and temperature; *E*, predicted percent change in daily outpatient visits for each 10 µg/m^3 increase in PM_{10} pollution.

*p<0.05; NS, not significant (p>0.05).

Table 4. Regression coefficients for PM_{10} and CO from models of outpatient visits in Anchorage, Alaska[a]

Dependent variable	R^2	n	PM_{10}	(lag 1) PM_{10}	CO
Asthma	0.009	610	0.0053*	—[b]	—
	0.014	610	—	0.0073**	—
	0.003	304	—	—	NS
Bronchitis	0.003	610	NS	—	—
	0.001	610	—	NS	—
	0.036	306	—	—	0.89†
URI	0.007	610	0.03	—	—
	0.001	610	—	NS	—
	0.075	306	—	—	3.43†

Abbreviations: PM_{10}, particulate matter < 10 µm in diameter; (lag 1)PM_{10}, PM_{10} on the previous day; CO, carbon monoxide; URI, upper respiratory illness.

[a] All variables are detrended using the Shumway filter (*9*). All models are regressions of weekend/weekday adjusted daily doctors visits on daily temperature and pollution .

[b] Variable not included in the model.

*p<0.05; **p<0.01; †p<0.001; NS, not significant (p>0.05).

Table 5: Estimated pollution regression coefficients for different study periods from models of outpatient visits in Anchorage, Alaska[a]

Dependent variable	Period	PM_{10}	(lag 1)PM_{10}
Asthma	Summer	0.004 (NS)	0.008**
	Winter	0.011*	0.006 (NS)
	Volcano	0.001 (NS)	0.005 (NS)
	No volcano	0.013**	0.012**
URI	Summer	0.027 (p<0.1)	-0.008 (NS)
	Winter	0.039 (NS)	0.011 (NS)
	Volcano	0.012 (NS)	-0.016 (NS)
	No volcano	0.064**	0.022 (NS)

Abbreviations: PM_{10}, particulate matter < 10 µm in diameter; (lag 1)PM_{10}, PM_{10} on the previous day; URI, upper respiratory illness.

[a] All variables are detrended using the Shumway filter (*9*); n = 610. All models are regressions of weekend/weekday adjusted daily doctors visits on daily temperature and pollution. Summer = April–October; Winter = November–March; volcano period = 18 August 1992–31 December 1992; No volcano period = 1 May 1992–17 August 1992 and 1 January 1993–1 March 1994.

*p<0.05; **p<0.01; NS, not significant (p>0.05).

gestive heart failure. Additional years of data should be analyzed to confirm that the results are not influenced by limited sample size. It is also important to better characterize the combustion or petroleum pollutants by collecting and analyzing long-term measurements of fine particles ($PM_{2.5}$ $<d_a<$2.5 µm), coarse particles (2.5 µm$<d_a<$10 µm), NO_2, CO, selected VOCs (e.g., benzene, toluene, xylenes), and trace elements of combustion (Br, Pb, V, Ni, etc.). Finally, the feasibility of examining other health records from Anchorage, such as emergency room visits, hospital admissions, and mortality should also be considered in subsequent studies.

Conclusions

The results from analysis of 22 months of daily PM_{10} and outpatient visits for respiratory illness in Anchorage, Alaska showed that an increase of 10 $\mu g/m^3$ in PM_{10} is associated with a 3–6% increase in medical visits for asthma and a 1–3% increase in medical visits for upper respiratory illness. This study is one of few which shows that silicaceous or earth crustal coarse particulate pollution may have an acute, adverse effect on respiratory health even at relatively low ambient concentrations. It also suggests that the increased morbidity is associated not just with a vulnerable segment of the population, but with a relatively young, healthy working group as well. These findings could have important implications to U.S. EPA in the ongoing review of the ambient air quality standard for PM_{10}. Whereas most of the past epidemiologic studies have linked particulate air pollution with daily health effects in urban settings, our results suggest that anthropogenic sources are not the only sources that may have an impact on respiratory health. Additional studies in Alaska or other environments with similar aerosol composition are highly recommended to confirm the statistical associations found between respiratory illness and coarse particle dominated PM_{10}.

REFERENCES

1. Özkaynak H, Thurston GD. Associations between 1980 U.S. mortality rates and alternate measures of airborne particle concentrations. Risk Anal 7:449–461 (1987).
2. Dockery D, Schwartz J, Spengler J. Air pollution and daily mortality: associations with particulates and acid aerosols. Environ Res 59: 362–373 (1992).
3. Pope III CA, Thun M, Namboodiri M, Dockery D, Evans J, Speizer F, Heath C Jr. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Resp Crit Care Med 151: 669–674 (1995).
4. Xu X, Li B, Huang, H. Air pollution and unscheduled hospital outpatient and emergency room visits. Environ Health Perspect 103:286–289 (1995) .
5. Pope III CA, Dockery D, Spengler J, Raizenne M. Respiratory health and PM_{10} pollution. Am Rev Resp Dis 144: 668–674 (1991).
6. R.J. Lee Group, Inc. Composition of PM_{10} material from Anchorage, AK, by computer controlled scanning electron microscopy. Pittsburg, PA:R.J. Lee Group, Inc., March 1995.
7. NEA. Aerosol characterization study of Anchorage, Alaska: chemical analysis and source apportionment, analysis of sources of Anchorage particulates. Beaverton, OR:Nuclear Environmental Analysis, Inc., 1985.
8. Spengler, JD and Thurston, GD. Mass and elemental composition of fine and coarse particles in six U.S. cities. J Air Pollut Control Assoc 33:1162–1171 (1983).
9. Shumway RH. Applied statistical time series analysis. Englewood Cliffs, NJ: Prentice–Hall, 1988.
10. Wallace LA. The total exposure assessment methodology (TEAM) study: summary and analysis, vol I. Report no. EPA/600/6–87/0022. Washington, DC:Environmental Protection Agency, 1987.
11. Taylor L, Morris S. Final report on the operations and findings of the Anchorage VOC monitoring project. Anchorage, AK:Anchorage Air Pollution Agency, 1995.
12. Morris RD, Naumora EN, Munasinghe RL. Ambient air pollution and hospitalization for congestive heart failure among elderly people in seven large US cities. Am J Public Health 85: 1361–1365 (1995).
13. Ware J, Spengler J, Neas L, Samet J, Wagner G, Coultas D, Özkaynak H, Schwab M. Respiratory and irritant health effects of ambient volatile organic compounds: The Kanawha County Health Study. Am J Epidemiol 137:1287–1301(1993).
14. Kinney PL, Özkaynak H. Associations of daily mortality and air pollution in Los Angeles County. Environ Res 54:99–120 (1991).
15. Özkaynak H, Xue J, Severance P, Burnett R, Raizenne M. Associations between cause and location-specific daily mortality and air pollution in Toronto, Canada. Presented at the Annual conference of the International Society for Exposure Analysis, 30 August–1 September 1995, Noordwijkerhout, The Netherlands.
16. Beck B, Brain J, Bohannon D. The pulmonary toxicity of an ash sample from Mt. St. Helens volcano. Exp Lung Res 3:389–401 (1981).
17. Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis 147:826–831 (1993).
18. Pope CA III. Respiratory hospital admissions associated with PM_{10} pollution on symptomatic and asymptomatic children. Am Rev Respir Dis 145:1123–1128 (1991).
19. Schwartz J. Air pollution and hospital admissions for the elderly in Birmingham, Alabama. Am J Epidemiol 139:589–598 (1994).
20. Schwartz J. Air pollution and hospital admissions for the elderly in Detroit, Michigan. Am J Respir Crit Care Med. 150:648–655 (1994).
21. Schwartz J. PM_{10}, ozone, and hospital admissions for the elderly in Minneapolis–St. Paul, Minnesota. Arch Environ Health 49:366–374 (1994).
22. Dockery DW, Pope CA III Acute respiratory effects of particulate air pollution. Annu Rev Public Health 15: 107–132 (1994).
23. Hefflin BJ, Jalaludin B, McClure E, Cobb N, Johnson C, Jecha L, Etzel RA. Surveillance for dust storms and respiratory diseases in Washington State, 1991. Arch Environ Health 49:170–174 (1994).