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ABSTRACT

Background Air pollution in cities has been linked to increased rates of mortality and morbidity in developed and developing countries. Although these findings have helped lead to a tightening of air-quality standards, their validity with respect to public health has been questioned.

Methods We assessed the effects of five major outdoor-air pollutants on daily mortality rates in 20 of the largest cities and metropolitan areas in the United States from 1987 to 1994. The pollutants were particulate matter that is less than 10 μm in aerodynamic diameter (PM<sub>10</sub>), ozone, carbon monoxide, sulfur dioxide, and nitrogen dioxide. We used a two-stage analytic approach that pooled data from multiple locations.

Results After taking into account potential confounding by other pollutants, we found consistent evidence that the level of PM<sub>10</sub> is associated with the rate of death from all causes and from cardiovascular and respiratory illnesses. The estimated increase in the relative rate of death from all causes was 0.51 percent (95 percent posterior interval, 0.07 to 0.93 percent) for each increase in the PM<sub>10</sub> level of 10 μg per cubic meter. The estimated increase in the relative rate of death from cardiovascular and respiratory causes was 0.68 percent (95 percent posterior interval, 0.20 to 1.16 percent) for each increase in the PM<sub>10</sub> level of 10 μg per cubic meter. There was weaker evidence that increases in ozone levels increased the relative rates of death during the summer, when ozone levels are highest, but not during the winter. Levels of the other pollutants were not significantly related to the mortality rate.

Conclusions There is consistent evidence that the levels of fine particulate matter in the air are associated with the risk of death from all causes and from cardiovascular and respiratory illnesses. These findings strengthen the rationale for controlling the levels of respirable particles in outdoor air. (N Engl J Med 2000;343:1742-9.)

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STUDIES showing that current levels of air pollution in the cities of many developed and developing countries are associated with increased rates of mortality and morbidity have heightened concern that air pollution continues to pose a threat to public health. The evidence suggests that small airborne particles are a toxic component of urban air pollution. Using this interpretation of the evidence as a rationale, the Environmental Protection Agency implemented a new standard for fine particulate matter. The existing standard, promulgated in 1987, specified the maximal levels allowable in a 24-hour period and on an annual basis for particulate matter with an aerodynamic diameter (the diameter of a unit-density sphere that has the same settling velocity in gas as the particle of interest) that was less than 10 μm (PM<sub>10</sub>). In 1997, the agency added standards for particulate matter that is less than 2.5 μm in aerodynamic diameter (PM<sub>2.5</sub>), since the size of such particles better corresponds to the size of particles that can penetrate to the airways and alveoli of the lung. This decision has been controversial; critics question whether the scientific evidence is strong enough to take regulatory action.

A more detailed version of our methods and findings is available elsewhere.

Key findings on particulate air pollution have come from time-series analyses of the association of air-pollution levels with the number of deaths per day. With the exception of a few studies, such as the multi-city Air Pollution and Health: a European Approach (APHEA) project and an analysis of data from six U.S. cities,

most of these studies have been based on single locations selected without a defined sampling plan. Consequently, the generalizability of the find-
ings is uncertain, and analytic strategies have differed among studies. Citing these limitations, critics have questioned whether the findings indicate an effect of air pollution generally or of particles specifically.2,3

To address these limitations, we combined information on the associations of levels of the five major outdoor-air pollutants — PM<sub>10</sub>, ozone, sulfur dioxide, carbon monoxide, and nitrogen dioxide — with daily mortality rates from 20 of the largest U.S. cities.4 Our estimates are based on a defined sample of the cities; statistical precision was enhanced by combining information from multiple locations.

**METHODS**

**Data Collection**

Data were collected from 1987 through 1994. We began with the 20 counties deemed the largest in the 1990 U.S. Census on the basis of population (or with logical groupings of counties), and for the analysis, we used data for the counties that included the associated cities, thus encompassing a population of more than 50 million. Analysis was carried out at the county level because the county was the common coding unit for the various data sets. In this article, we refer to cities and metropolitan areas rather than counties. Daily mortality rates were obtained from the National Center for Health Statistics (Table 1). After excluding deaths from external causes (e.g., accidents, suicide, and homicide) and deaths of nonresidents, we classified the deaths according to age group (<55 years, 55 to 74 years, and ≥75 years) and cause (cardiovascular and respiratory and other).4 Data on selected demographic characteristics were obtained from the 1990 U.S. Census.4

Hourly temperature and dew-point data were available from the EarthInfo compact-disk7 data base of the National Climatic Data Center. For analysis we used the 24-hour mean value for each day. The air-pollution data were obtained from the data base of the Aerometric Information Retrieval System,8 which is maintained by the Environmental Protection Agency. For population-oriented monitoring variables, we downloaded all available data for PM<sub>10</sub>, ozone, carbon monoxide, sulfur dioxide, and nitrogen dioxide. For the pollutants measured on an hourly basis, we calculated the 24-hour average. If the levels of pollutants were monitored at multiple locations in a metropolitan area, we averaged the data. To avoid the potential consequences of outlying values, we excluded the highest and lowest 10 percent of values (10 percent trimmed mean) and then averaged the values for each set of monitors, after the value for each monitor had been corrected for its yearly average.

**Statistical Analysis**

We used a two-stage log-linear regression model.19-21 In the first stage, a separate log-linear regression of the daily mortality rate on air-pollution measures and other confounders was fitted to obtain estimates of the relative rate of mortality associated with the pollution variable and the degree of statistical uncertainty for each of

**Table 1. Rates of Death from All Causes and from Cardiovascular and Respiratory Causes in 20 U.S. Cities and Metropolitan Areas, According to Various Socioeconomic Characteristics, 1987-1994.**

<table>
<thead>
<tr>
<th>CITY OR METROPOLITAN AREA</th>
<th>COUNTRY</th>
<th>POPULATION</th>
<th>MEAN NO. OF DEATHS/ DAY</th>
<th>MEAN NO. OF DEATHS FROM CARDIOVASCULAR AND RESPIRATORY CAUSES/DAY</th>
<th>ANNUAL INCOME &lt;$12,679 HIGH SCHOOL GRADUATE</th>
<th>ANNUAL INCOME &lt;$100,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Los Angeles</td>
<td>Los Angeles</td>
<td>6,863,164</td>
<td>148</td>
<td>87</td>
<td>14.8</td>
<td>70.9</td>
</tr>
<tr>
<td>New York</td>
<td>Bronx, Kings, New York, Richmond, Queens, Westchester</td>
<td>7,510,666</td>
<td>190.9</td>
<td>108.3</td>
<td>175</td>
<td>71.4</td>
</tr>
<tr>
<td>Chicago</td>
<td>Cook</td>
<td>5,105,067</td>
<td>113.9</td>
<td>62</td>
<td>14.0</td>
<td>73.4</td>
</tr>
<tr>
<td>Dallas—Fort Worth, Tex.</td>
<td>Collin, Dallas, Rockwall, Tarrant</td>
<td>3,312,553</td>
<td>479</td>
<td>26</td>
<td>11.7</td>
<td>79.0</td>
</tr>
<tr>
<td>Houston</td>
<td>Harris</td>
<td>2,818,199</td>
<td>39.9</td>
<td>20</td>
<td>15.5</td>
<td>74.0</td>
</tr>
<tr>
<td>San Diego, Calif.</td>
<td>San Diego</td>
<td>2,498,016</td>
<td>41.6</td>
<td>22.6</td>
<td>10.9</td>
<td>81.9</td>
</tr>
<tr>
<td>Santa Ana—Anaheim, Calif.</td>
<td>Orange</td>
<td>2,410,556</td>
<td>22.4</td>
<td>18.7</td>
<td>8.3</td>
<td>81.2</td>
</tr>
<tr>
<td>Phoenix, Ariz.</td>
<td>Maricopa</td>
<td>2,132,101</td>
<td>38.4</td>
<td>20.9</td>
<td>12.1</td>
<td>81.5</td>
</tr>
<tr>
<td>Detroit</td>
<td>Wayne</td>
<td>2,111,687</td>
<td>46.9</td>
<td>25.5</td>
<td>19.8</td>
<td>70.0</td>
</tr>
<tr>
<td>Miami</td>
<td>Dade</td>
<td>1,937,094</td>
<td>43.8</td>
<td>23.6</td>
<td>17.6</td>
<td>68.0</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>Philadelphia</td>
<td>1,885,577</td>
<td>42.3</td>
<td>21.5</td>
<td>19.8</td>
<td>64.3</td>
</tr>
<tr>
<td>Minneapolis</td>
<td>Hennepin, Ramsey</td>
<td>1,318,195</td>
<td>26.3</td>
<td>13.9</td>
<td>9.7</td>
<td>82.2</td>
</tr>
<tr>
<td>Seattle</td>
<td>King</td>
<td>1,507,319</td>
<td>25.6</td>
<td>15.4</td>
<td>7.8</td>
<td>88.2</td>
</tr>
<tr>
<td>San Jose, Calif.</td>
<td>Santa Clara</td>
<td>1,497,577</td>
<td>19.7</td>
<td>10.7</td>
<td>7.3</td>
<td>82.0</td>
</tr>
<tr>
<td>Cleveland</td>
<td>Cuyahoga</td>
<td>1,412,141</td>
<td>36.5</td>
<td>20.1</td>
<td>13.5</td>
<td>74.0</td>
</tr>
<tr>
<td>San Bernardino, Calif.</td>
<td>San Bernardino</td>
<td>1,412,140</td>
<td>36.5</td>
<td>20.1</td>
<td>13.5</td>
<td>74.0</td>
</tr>
<tr>
<td>Pittsburgh</td>
<td>Allegheny</td>
<td>1,336,449</td>
<td>37.6</td>
<td>21.0</td>
<td>11.3</td>
<td>79.0</td>
</tr>
<tr>
<td>Oakland, Calif.</td>
<td>Alameda</td>
<td>1,279,183</td>
<td>22.2</td>
<td>12.2</td>
<td>10.3</td>
<td>81.4</td>
</tr>
<tr>
<td>Atlanta</td>
<td>Fulton, DeKalb</td>
<td>1,194,783</td>
<td>175</td>
<td>8.8</td>
<td>14.5</td>
<td>80.6</td>
</tr>
<tr>
<td>San Antonio, Tex.</td>
<td>Bexar</td>
<td>1,185,394</td>
<td>20.1</td>
<td>10.5</td>
<td>19.4</td>
<td>72.7</td>
</tr>
</tbody>
</table>

*The demographic information was obtained from the 1990 U.S. Census.*
the 20 cities. In the second stage, the estimates of the relative rates were combined for all cities (after adjustment for the various levels of uncertainty) to obtain an overall estimate and to assess whether city-specific characteristics modified the estimated effect of air pollution on the relative rate of death.

In the first-stage log-linear regressions, we controlled for possible confounding by longer-term trends resulting from changes in the size and characteristics of the population, health status, and health care and from shorter-term effects of seasonality and the presence or absence of influenza epidemics. To do this, we used a flexible function that took into account the variation in the mortality rate over periods of several months (a smoothing function with respect to calendar time with 7 degrees of freedom per year per city, which was allowed to differ in the three age groups). We also adjusted for the short-term effect of weather on the risk of death by including similar smoothing functions with respect to a specific day's temperature and the average temperature for the three days preceding it (6 degrees of freedom) and to dew point (5 degrees of freedom). Finally, we included indicator variables for the day of the week. This model specification was based on extensive, previously reported exploratory analyses,10,11,12. In this article, our results do not reflect the degrees of freedom used. We have found that the relative rates of air pollution were not sensitive to the number of degrees of freedom selected for the smoothing functions of time, temperature, and dew point,10,11,12.

In the first stage, we analyzed the effect of the day on which the pollution data were obtained (the current day, the day before, or two days before) on the association with mortality rates. The overall effect did not vary with the lag interval selected. Consequently, we report data for a one-day lag between pollution variables and mortality.

We considered the effects of multiple pollutants on the relative rate of mortality. We initially conducted univariate analyses that included PM10 alone and ozone alone. We then considered the effects of the two pollutants in a bivariate model and developed trivariate models that also included sulfur dioxide, nitrogen dioxide, or carbon monoxide. The trivariate models provided estimates of the individual effects of carbon monoxide, sulfur dioxide, and nitrogen dioxide on the risk of death after adjustment for PM10 and ozone levels.

The second stage of the analysis provided pooled estimates of the relative rates of mortality associated with specific pollutants and a characterization of the effects of air pollutants among the cities. We also examined factors determining heterogeneity in the effect of air pollution on mortality. With respect to determinants of heterogeneity in the second stage of the analysis, we assumed that first-stage estimates of the relative mortality rates associated with specific pollutants followed a linear regression with the selected city-specific demographic characteristics (Table 1) as predictors. The second-stage analysis provided an estimate of the effect of each predictor on the relative rate of mortality associated with PM10.

Model fitting was performed with use of a Bayesian statistical approach,14 which provides an estimate of the posterior distribution of the variable of interest. We carried out this analysis without making a strong prior assumption as to the value of the relative rate. The posterior distribution is used to determine the probability that the relative rate of mortality associated with PM10 has a particular value — that is, it is a measure of the strength of the evidence on the relative rate of mortality associated with PM10. Greater than zero. The posterior distribution can also be used to determine the 95 percent posterior intervals. The 95 percent posterior interval encompasses 95 percent of the posterior distribution, a Bayesian formulation similar to the 95 percent confidence interval. All analyses were performed with use of S-Plus statistical software.31

RESULTS

The 20 cities and metropolitan areas broadly represented the United States. The number of days for which pollution data were available varied (Table 2). Since the Environmental Protection Agency requires levels of PM10 to be measured only every six days, data for ozone and other pollutants were generally available on more days. The mean daily values for PM10 ranged from about 20 μg per cubic meter to nearly 500 μg per cubic meter; the present maximal allowable level of PM10 in a 24-hour period is 150 μg per cubic meter. The average numbers of deaths per day were substantial, ranging from less than 20 to nearly 200 (Table 1). The correlation coefficients of all correlations between pollutants for all 20 cities and metropolitan areas are provided in Table 3. The correlation structure generally reflects the common sources of the primary combustion-related gases (sulfur dioxide, nitrogen dioxide, and carbon monoxide) and of PM10. The level of ozone was only slightly correlated with that of PM10 and was not correlated with the levels of other gaseous pollutants.

In initial univariate analyses, the level of PM10 was positively associated with the rate of death from all causes in most of the 20 cities and metropolitan areas (Fig. 1). Adjustment for the effect of ozone levels had little effect on the association, whereas the effects of the ozone level, before and after adjustment for PM10 levels, tended to be more variable. The analysis of each pollutant was also stratified according to the cause of death. The city-specific associations between PM10 levels and the rate of death from cardiovascular and respiratory causes were similar to those for the rate of death from all causes. A previous univariate analysis stratified according to age showed no age-associated trend.14

The combined analysis for all 20 cities and metropolitan areas confirmed the association between PM10 levels and the rate of death from all causes (Fig. 2) and of death from cardiovascular and respiratory causes. Figure 2 shows the posterior distributions of the estimated increases in the relative rates of death from all causes associated with each increase in the PM10 level of 10 μg per cubic meter before and after adjustment for levels of ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide, as well as the probability that overall effects are greater than zero for each model. With respect to death from all causes, the distributions are shifted toward the right, with the respective mean increases in the number of deaths per day for each increase in the PM10 level of 10 μg per cubic meter (i.e., estimated relative rates) ranging between approximately 0.3 percent and 0.6 percent. An increase in the relative rate of 0.3 percent corresponds to a relative risk of death of 1.003. In the model that included PM10 alone, the estimated increase in the relative rate of death from all causes was 0.51 percent for each increase in the PM10 level of 10 μg per cubic meter (95 percent posterior interval, 0.07 to 0.93 percent). The posterior distributions of the PM10 levels did
**Table 2. Mean Levels of Pollutants in 20 U.S. Cities and Metropolitan Areas.**

<table>
<thead>
<tr>
<th>City or Metropolitan Area</th>
<th>No. of Monitors</th>
<th>No. of Days on Which Data Were Collected</th>
<th>Ozone (ppb)</th>
<th>PM&lt;sub&gt;10&lt;/sub&gt; (μg/m&lt;sup&gt;3&lt;/sup&gt;)</th>
<th>Nitr. Dioxide (ppm)</th>
<th>Sulfur Dioxide (ppm)</th>
<th>Carbon Monoxide (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Los Angeles</td>
<td>7</td>
<td>2922 580</td>
<td>22.8 (6.9, 40.2)</td>
<td>46.0 (21.5, 73.1)</td>
<td>39.4 (12.6, 58.6)</td>
<td>1.9 (0.5, 2.0)</td>
<td>15.1 (5.9, 23.3)</td>
</tr>
<tr>
<td>New York</td>
<td>15</td>
<td>2922 489</td>
<td>19.6 (7.3, 34.0)</td>
<td>28.8 (16.1, 44.8)</td>
<td>38.9 (27.0, 53.7)</td>
<td>2.4 (0.8, 2.1)</td>
<td>20.4 (2.1, 27.6)</td>
</tr>
<tr>
<td>Chicago</td>
<td>16</td>
<td>2922 2683</td>
<td>35.6 (11.3, 63.5)</td>
<td>35.6 (15.7, 60.3)</td>
<td>24.1 (14.4, 35.0)</td>
<td>0.3 (0.1, 0.5)</td>
<td>7.9 (4.5, 11.9)</td>
</tr>
<tr>
<td>Dallas–Fort Worth, Tex.</td>
<td>2</td>
<td>2922 624</td>
<td>3.3 (0.1, 4.1)</td>
<td>23.8 (11.4, 39.8)</td>
<td>12.4 (5.9, 22.7)</td>
<td>0.1 (0.8, 3.0)</td>
<td>7.4 (5.6, 12.0)</td>
</tr>
<tr>
<td>Houston</td>
<td>2</td>
<td>2922 793</td>
<td>20.5 (9.3, 35.1)</td>
<td>30.0 (13.5, 48.6)</td>
<td>18.3 (9.0, 29.0)</td>
<td>0.1 (0.8, 3.0)</td>
<td>8.9 (4.4, 12.2)</td>
</tr>
<tr>
<td>San Diego, Calif.</td>
<td>4</td>
<td>2922 521</td>
<td>13.6 (18.1, 55.8)</td>
<td>23.6 (18.1, 52.1)</td>
<td>22.9 (11.2, 38.4)</td>
<td>4.1 (0.9, 4.0)</td>
<td>11.0 (4.5, 20.5)</td>
</tr>
<tr>
<td>Santa Ana–Anaheim, Calif.</td>
<td>2</td>
<td>2922 460</td>
<td>30.3 (25.1, 33.5)</td>
<td>27.4 (15.4, 59.2)</td>
<td>25.1 (18.0, 59.0)</td>
<td>1.1 (0.1, 0.5)</td>
<td>12.3 (4.7, 23.2)</td>
</tr>
<tr>
<td>Phoenix, Ariz.</td>
<td>10</td>
<td>2919 426</td>
<td>21.8 (7.0, 35.0)</td>
<td>39.7 (21.4, 58.4)</td>
<td>16.6 (8.8, 26.0)</td>
<td>0.4 (0.1, 0.5)</td>
<td>6.6 (3.2, 11.1)</td>
</tr>
<tr>
<td>Denver</td>
<td>3</td>
<td>1861 1348</td>
<td>22.6 (9.1, 37.5)</td>
<td>40.9 (16.4, 71.1)</td>
<td>21.3 (11.5, 32.1)</td>
<td>1.0 (0.1, 0.5)</td>
<td>6.6 (3.2, 11.1)</td>
</tr>
<tr>
<td>Miami</td>
<td>4</td>
<td>2822 405</td>
<td>25.4 (14.5, 40.0)</td>
<td>25.7 (15.0, 36.6)</td>
<td>11.0 (4.5, 20.2)</td>
<td>NA</td>
<td>10.6 (6.5, 15.9)</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>8</td>
<td>2901 495</td>
<td>20.5 (3.9, 39.5)</td>
<td>35.4 (19.0, 56.0)</td>
<td>23.2 (20.7, 46.0)</td>
<td>2.6 (0.1, 0.5)</td>
<td>11.8 (7.0, 17.2)</td>
</tr>
<tr>
<td>Minneapolis</td>
<td>8</td>
<td>NA 2764</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>17.8 (10.5, 26.4)</td>
</tr>
<tr>
<td>Seattle</td>
<td>7</td>
<td>1820 2205</td>
<td>19.4 (8.7, 30.0)</td>
<td>25.3 (10.2, 44.8)</td>
<td>25.1 (11.7, 44.1)</td>
<td>NA</td>
<td>9.4 (5.1, 21.3)</td>
</tr>
<tr>
<td>San Jose, Calif.</td>
<td>2</td>
<td>2922 945</td>
<td>17.9 (7.7, 28.1)</td>
<td>30.4 (9.3, 61.6)</td>
<td>25.1 (11.7, 44.1)</td>
<td>NA</td>
<td>2.7 (0.1, 0.5)</td>
</tr>
<tr>
<td>Cleveland</td>
<td>3</td>
<td>1712 1298</td>
<td>22.5 (12.7, 49.9)</td>
<td>45.1 (19.7, 76.7)</td>
<td>25.2 (15.2, 36.7)</td>
<td>10.3 (2.7, 19.9)</td>
<td>8.5 (4.3, 13.8)</td>
</tr>
<tr>
<td>San Bernardino, Calif.</td>
<td>8</td>
<td>2922 258</td>
<td>5.9 (4.5, 60.3)</td>
<td>37.0 (16.1, 56.2)</td>
<td>25.2 (15.2, 36.7)</td>
<td>0.7 (0.1, 0.5)</td>
<td>10.3 (4.0, 17.5)</td>
</tr>
<tr>
<td>Pittsburgh</td>
<td>30</td>
<td>2883 2899</td>
<td>20.7 (7.0, 36.6)</td>
<td>31.6 (8.9, 61.3)</td>
<td>27.6 (17.6, 83.6)</td>
<td>14.2 (4.5, 26.5)</td>
<td>12.2 (6.1, 19.8)</td>
</tr>
<tr>
<td>Oakland, Calif.</td>
<td>3</td>
<td>2922 508</td>
<td>17.2 (7.7, 26.9)</td>
<td>26.3 (9.3, 47.8)</td>
<td>21.3 (9.6, 35.2)</td>
<td>3.1 (0.1, 0.5)</td>
<td>9.1 (2.9, 17.0)</td>
</tr>
<tr>
<td>Atlanta</td>
<td>3</td>
<td>2200 482</td>
<td>24.5 (11.6, 37.4)</td>
<td>34.4 (15.8, 56.4)</td>
<td>20.4 (11.7, 30.4)</td>
<td>0.5 (0.1, 0.5)</td>
<td>8.8 (3.2, 14.3)</td>
</tr>
<tr>
<td>San Antonio, Tex.</td>
<td>2</td>
<td>2918 670</td>
<td>22.2 (11.4, 34.5)</td>
<td>27.8 (12.3, 36.3)</td>
<td>NA</td>
<td>NA</td>
<td>10.1 (4.1, 17.3)</td>
</tr>
</tbody>
</table>

*Cities are listed according to sample population size. Values shown are 10 percent trimmed means, as described in the Methods section. Values in parentheses are the 10th and 90th percentiles. PM<sub>10</sub> denotes particulate matter that is less than 10 μm in aerodynamic diameter, and NA not available.

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**Table 3. Correlation Coefficients of All Pairwise Correlations between the 20 Cities and Metropolitan Areas.**

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>PM&lt;sub&gt;10&lt;/sub&gt;</th>
<th>Ozone</th>
<th>Nitr. Dioxide</th>
<th>Sulfur Dioxide</th>
<th>Carbon Monoxide</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM&lt;sub&gt;10&lt;/sub&gt;</td>
<td>1.00</td>
<td>0.24 (−0.21, 0.41)</td>
<td>0.53 (0.22, 0.74)</td>
<td>0.39 (0.16, 0.51)</td>
<td>0.46 (0.15, 0.67)</td>
</tr>
<tr>
<td>Ozone</td>
<td>1.00</td>
<td>0.02 (−0.34, 0.20)</td>
<td>−0.06 (−0.31, 0.09)</td>
<td>0.01 (0.12, 0.70)</td>
<td>0.19 (−0.52, −0.04)</td>
</tr>
<tr>
<td>Nitr. Dioxide</td>
<td>1.00</td>
<td>0.51 (0.32, 0.70)</td>
<td>1.00 (1.00, 1.00)</td>
<td>0.64 (0.51, 0.86)</td>
<td>0.61 (0.30, 0.71)</td>
</tr>
<tr>
<td>Sulfur Dioxide</td>
<td>1.00</td>
<td>0.01 (0.12, 0.70)</td>
<td>0.01 (0.12, 0.70)</td>
<td>1.00 (1.00, 1.00)</td>
<td>1.00 (1.00, 1.00)</td>
</tr>
</tbody>
</table>

*The correlation coefficients were calculated for values for all monitors within the cities. PM<sub>10</sub> denotes particulate matter that is less than 10 μm in aerodynamic diameter.

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not change substantially after adjustment for the other pollutants, suggesting that the univariate findings were not affected by confounding by other pollutants (Fig. 2).

The PM<sub>10</sub> level had a somewhat greater effect on the rate of death from cardiovascular and respiratory causes than on the rate of death from all causes and was associated with a correspondingly larger probability that the effect was greater than zero. The estimated increase in the relative rate of death from cardiovascular and respiratory causes was 0.68 percent for each increase of 10 μg per cubic meter in the PM<sub>10</sub> level (95 percent posterior interval, 0.20 to 1.16 percent).

The univariate effects of ozone levels were examined during a one-year period and according to sea-
Figure 1. Regression Coefficients for the Changes in the Rate of Death from All Causes for Each Increase in the PM₁₀ Level of 10 µg per Cubic Meter, before and after Adjustment for Ozone Levels, and for Each Increase in the Ozone Level of 10 ppb, before and after Adjustment for PM₁₀ Levels in 20 Cities and Metropolitan Areas.

PM₁₀ denotes particulate matter that is less than 10 µm in aerodynamic diameter. Bars indicate 95 percent confidence intervals. No data on ozone were available for Minneapolis.

Overall, the posterior distributions of the effects of ozone were concentrated near zero, and there was only an even chance that the effect was larger than zero when death from all causes and death from cardiovascular and respiratory causes were considered separately. Because ozone levels vary strongly with the season, we compared the effects of ozone levels during the three hottest summer months (June, July, and August), when levels are highest, and three cold months (November, December, and January), when levels tend to be lowest. With the use of this stratification, the estimated relative rates of death from all...
other pollutants in the statistical model or by the time at which data were collected. Our findings strongly support the findings of prior studies of particulate matter and mortality. These studies, which were largely based on data from single cities, used a variety of measures of particulate matter, including levels of total suspended particles, black smoke (a measure of soiling of a filter that provides an index of particle levels), PM_{10}, and PM_{2.5}. The statistical methods used to assess the relations between levels of pollution and the risk of death were also heterogeneous; for example, there was no uniformity in the approaches used to control for factors that varied over time or for other pollutants. Nonetheless, using a weight-of-evidence approach, the Environmental Protection Agency interpreted the results of the studies as indicating a possibly causal association between levels of particulate matter and adverse effects on health.

In a meta-analysis of U.S. studies of particulate air pollution published between 1990 and 1993, Dockery and Pope estimated that each increase in the PM_{10} level of 10 μg per cubic meter increased the relative rate of death from all causes by 1 percent. In a subsequent update that included data from reports published through 1995, Dockery and Pope found little change in this estimate. Schwartz also performed a meta-analysis of studies published between 1990 and 1993 but included data from London and Minneapolis in addition to the data on the eight cities considered by Dockery and Pope. The resulting estimated increase in the relative rate of death from all causes was 0.7 percent for each increase in the PM_{10} level of 10 μg per cubic meter. The APHEA project analyzed data from 12 European cities and then estimated summary measures. For the six western European cities in the study, the mortality rate was estimated to increase by 0.4 percent for each increase in the PM_{10} level of 10 μg per cubic meter. In our 20-city analysis, our estimate of an increase of approximately 0.5 percent in the rate of death from all causes for each increase in the PM_{10} level of 10 μg per cubic meter is very similar to the estimate of the APHEA project. The fact that our estimate was lower than those of Dockery and Pope and Schwartz may reflect differences in analytic techniques and the cities selected. The initial reports included in the meta-analyses may have been biased by the fact that studies with positive findings are more likely to be selected for publication than those with negative findings. Our 20-city estimate is not subject to such bias and our results should thus be more applicable to the United States in general.

We did not find an effect of ozone levels on the overall rate of death from all causes or from cardiovascular and respiratory causes during the full year period. Ozone levels were positively associated with mortality rates during the summer months when ozone levels were highest, although the 95 percent post-

 causes with each increase in the ozone level of 10 ppb were 0.41 percent (95 percent posterior interval, −0.20 to 1.01 percent) during the summer months and −1.83 percent (95 percent posterior interval, −2.69 to −0.96 percent) during the cold months.

The differences between cities in the relative rates did not depend on average PM_{10} or ozone levels in a city or on city-specific demographic characteristics; for these variables, all associated 95 percent posterior intervals included zero. Consequently, the analyses and results for PM_{10} were not adjusted for these city-specific characteristics.

We also analyzed the effects of levels of carbon monoxide, sulfur dioxide, and nitrogen dioxide in a fashion similar to that of the analysis of PM_{10} levels. After adjustment for PM_{10} and ozone levels, we found little evidence that these pollutants had a significant effect on the relative rate of death.

DISCUSSION

We found consistent evidence that the level of PM_{10} is associated with the rates of death from all causes and from cardiovascular and respiratory causes. The association of PM_{10} was not affected by the inclusion of

Figure 2. Posterior Distributions of the Overall Relative Rate of Increase in Death from All Causes for Each Increase in the PM_{10} Level of 10 μg per Cubic Meter, before and after Adjustment for the Levels of Ozone (O_3), Nitrogen Dioxide (NO_2), Sulfur Dioxide (SO_2), and Carbon Monoxide (CO).

Values in parentheses are the posterior probabilities that the overall effects are greater than zero. PM_{10} denotes particulate matter that is less than 10 μm in aerodynamic diameter.
rior interval extended into the range indicating no
effect of ozone levels on mortality. The finding of an
effect of ozone levels only during the summer may
reflect the higher levels of ozone during these months
or, possibly, differences in the characteristics of pho-
tochemical pollution during the various seasons. Oth-
er recent studies have generally found an association
between ozone levels and the risk of death. In the
APHEA project, the maximal ozone levels during a
one-hour period were associated with the numbers of
deaths per day in four cities (London; Athens,
Greece; Barcelona, Spain; and Paris), and a quanti-
tatively similar effect was found with additional data
from three cities (Amsterdam; and Basel and Zür-
ich, Switzerland) that were not part of the APHEA
project. For each increase of 50 μg per cubic meter
in the one-hour maximal level, the estimated relative
risk of death was 1.029 (i.e., a 1.1 percent increase
in the rate of death for each increase in the ozone
level of 10 ppb), with the use of a random-effects
model for combining the city-specific data. Thurso-
nton and Iró9 pooled data from 15 studies and esti-
imated that the relative risk of death was 1.036 for
each increase of 100 ppb in the daily one-hour max-
imal level of ozone (i.e., a 0.36 percent increase in
the rate of death for each increase in the ozone level
of 10 ppb). For the summer months, we estimate (a
0.41 percent increase in the rate of death for each
increase in the ozone level of 10 ppb) was similar to
those of Thurston and Iró. Taken together, the results
of these three studies provide consistent evidence that
exposure to ozone also increases the risk of death.

The limitations of our analyses should be consid-
ered. Data on levels of PM_{2.5} are not yet available na-
tionally, since a monitoring network for particles in
this size range is currently being implemented. We
used PM_{10} levels because they have been monitored
since 1987; there is variation across the United States
in the proportion of PM_{10} mass that is made up of
PM_{2.5}, so that the PM_{10} level is an imperfect surro-
gate for the PM_{2.5} level. In addition, for regulatory
purposes, PM_{10} levels must only be measured every
six days, limiting the extent of available data.

Our analyses also did not address the extent to
which life is shortened in association with daily ex-
posure to the various pollutants. The finding that the
association between PM_{10} levels and the risk of death
was strongest for cardiovascular and respiratory causes
of death is consistent with the hypothesis that per-
sions made frail by advanced heart and lung disease
are more susceptible to the adverse effects of air pol-
ution. The findings from several epidemiologic stud-
ies of the longer-term effects of air pollution on the
risk of death suggest that exposure to air pollution may
do more than simply shorten life by a few days. Several
analyses of daily mortality data also indicate that the
effect of air pollution may go beyond short-
ening life by a few days.

We found no evidence that key socioeconomic fac-
tors such as low socioeconomic status affect the
association between PM_{10} levels and the risk of death
in linear regression models. The medical conditions
and poor health that increase the risk of death may
not be adequately reflected by the socioeconomic
indicators recorded by the U.S. Census. Thus, more
specific information on health status, rather than on
social factors, may be needed to explore this issue,
particularly in relation to the susceptibility of partic-
ular groups of people. Finally, we used county-level
data for these social factors because most of our data
were categorized according to county. The variation
in socioeconomic status in a typical urban county,
however, is usually considerably larger than the vari-
ation among counties. Thus, the demographic factors
considered in the second stages of the models may
be too broad to be informative.

The epidemiologic evidence that levels of particu-
late matter are associated with the risk of mortality
and morbidity has prompted the promulgation of a
new standard for PM_{10} in the United States and a
rethinking of guidelines for particulate matter in Eu-

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