
JONATHAN M. SAMET, M.D., FRANCESCA DOMINICI, PH.D., FRANK C. CURRIERO, PH.D., IVAN COURSAC, M.S., AND SCOTT L. ZEGER, PH.D.

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$_{10}$), 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$_{10}$ 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$_{10}$ 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$_{2.5}$ 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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Findings are 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.\textsuperscript{7,12,14}

To address these limitations, we combined information on the associations of levels of the five major outdoor-air pollutants — PM\textsubscript{10}, ozone, sulfur dioxide, carbon monoxide, and nitrogen dioxide — with daily mortality rates from 20 of the largest U.S. cities.\textsuperscript{14} 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 beginning information from multiple locations.

**Statistical Analysis**

We used a two-stage log-linear regression model.\textsuperscript{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 the
debased calculations.

**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.\textsuperscript{*}

<table>
<thead>
<tr>
<th>City or Metropolitan Area</th>
<th>Counties</th>
<th>Population</th>
<th>Mean No. of Deaths/Day</th>
<th>Mean No. of Deaths from Cardiovascular Causes/Day</th>
<th>Annual Income &lt;$12,675 (10% Trimmed Mean)</th>
<th>High-School Graduate</th>
<th>Annual Income &gt;$100,000 (10% Trimmed Mean)</th>
<th>Percentage of Population</th>
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<tbody>
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*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 (3 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. 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.

In the first-stage analysis, 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 PM\textsubscript{10} alone and ozone alone. We then considered the effects of these 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 PM\textsubscript{10} 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 predictor variables. The second-stage analysis provided an estimate of the effect of each predictor variable on the relative rate of mortality associated with PM\textsubscript{10}.

Model fitting was performed with use of a Bayesian statistical approach, 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 PM\textsubscript{10} has a particular value—that is, it is a measure of the strength of the evidence. One important calculation is the posterior probability that the relative rate of mortality associated with PM\textsubscript{10} is 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.

**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 PM\textsubscript{10} to be measured only every six days, data for ozone and other pollutants were generally available on more days. The mean daily values for PM\textsubscript{10} ranged from about 20 µg per cubic meter to nearly 50 µg per cubic meter; the present maximal allowable level of PM\textsubscript{10} 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 PM\textsubscript{10}. The level of ozone was only slightly correlated with that of PM\textsubscript{10} and was not correlated with the levels of other gaseous pollutants.

In initial univariate analyses, the level of PM\textsubscript{10} 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 PM\textsubscript{10} 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 PM\textsubscript{10} 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.

The combined analysis for all 20 cities and metropolitan areas confirmed the association between PM\textsubscript{10} 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 PM\textsubscript{10} 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 PM\textsubscript{10} 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 from all causes of 1.003. In the model that included PM\textsubscript{10} alone, the estimated increase in the relative rate of death from all causes was 0.51 percent for each increase in the PM\textsubscript{10} level of 10 µg per cubic meter (95 percent posterior interval, 0.07 to 0.93 percent). The posterior distributions of the PM\textsubscript{10} levels did 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.
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$_{10}$ 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$_{10}$ 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-
son. 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

Figure 1. Regression Coefficients for the Changes in the Rate of Death from All Causes for Each Increase in the PM$_{10}$ 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 PM$_{10}$ Levels in 20 Cities and Metropolitan Areas.

PM$_{10}$ 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.
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.26 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), PM10, and PM2.5. 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.5

In a meta-analysis of U.S. studies of particulate air pollution published between 1990 and 1993, Dockery and Pope2 estimated that each increase in the PM10 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.27 Schwartz28 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 PM10 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 PM10 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 PM10 level of 10 µg per cubic meter is very similar to the estimate of the APHEA project.10 The fact that our estimate was lower than those of Dockery and Pope2 and Schwartz28 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 poste-
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 photochemical pollution during the various seasons. Other recent studies have generally found an association between ozone levels and the risk of death.29 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 quantitatively similar effect was found with additional data from three cities (Amsterdam; and Basel and Zurich, Switzerland) that were not part of the APHEA project.30 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. Thurston and Ito29 pooled data from 15 studies and estimated that the relative risk of death was 1.036 for each increase of 100 ppb in the daily one-hour maximal 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, our 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 Ito. 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 considered. Data on levels of PM$_{2.5}$ are not yet available nationally, 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 surrogate for the PM$_{2.5}$ level.3 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 exposure 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 persons made frail by advanced heart and lung disease are more susceptible to the adverse effects of air pollution. The findings from several epidemiologic studies 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.31,32 Several analyses of daily mortality data also indicate that the effect of air pollution may go beyond shortening life by a few days.33,34 We found no evidence that key socioeconomic factors 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 particular 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 variation 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 particulate 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 Europe. Our analyses provide evidence that particulate air pollution continues to have an adverse effect on the public’s health and strengthen the rationale for limiting levels of respirable particles in outdoor air.

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REFERENCES