

The New England Journal of Medicine

© Copyright, 2000, by the Massachusetts Medical Society

VOLUME 343

DECEMBER 14, 2000

NUMBER 24



FINE PARTICULATE AIR POLLUTION AND MORTALITY IN 20 U.S. CITIES, 1987–1994

JONATHAN M. SAMET, M.D., FRANCESCA DOMINICI, PH.D., FRANK C. CURRIERO, PH.D., IVAN COURSAK, 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_{10} 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.)

©2000, Massachusetts Medical Society.

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.¹⁻³ 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.⁴ 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_{10}). In 1997, the agency added standards for particulate matter that is less than 2.5 μm in aerodynamic diameter ($\text{PM}_{2.5}$), 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.⁵⁻⁸ A more detailed version of our methods and findings is available elsewhere.⁹

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.³ With the exception of a few studies, such as the multi-city Air Pollution and Health: a European Approach (APHEA) project¹⁰ and an analysis of data from six U.S. cities,¹¹ most of these studies have been based on single locations selected without a defined sampling plan. Consequently, the generalizability of the find-

From the Departments of Epidemiology (J.M.S.) and Biostatistics (F.D., F.C.C., I.C., S.L.Z.), School of Hygiene and Public Health, Johns Hopkins University, Baltimore. Address reprint requests to Dr. Samet at Johns Hopkins University, School of Public Health, 615 N. Wolfe St., Suite 6041, Baltimore, MD 21205, or at jsamet@jhsph.edu.

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.^{7,12,13}

To address these limitations, we combined information on the associations of levels of the five major outdoor-air pollutants — PM₁₀, ozone, sulfur dioxide, carbon monoxide, and nitrogen dioxide — with daily mortality rates from 20 of the largest U.S. cities.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 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 (<65 years, 65 to 74 years, and ≥75 years) and cause (cardiovascular and respiratory and other).¹⁵ Data on selected demographic characteristics were obtained from the 1990 U.S. Census.¹⁶

Hourly temperature and dew-point data were available from the EarthInfo compact-disk¹⁷ 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,¹⁸ which is maintained by the Environmental Protection Agency. For population-oriented monitoring variables, we downloaded all available data for PM₁₀, 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.*

CITY OR METROPOLITAN AREA	COUNTIES	POPULATION	MEAN No. OF DEATHS/ DAY	MEAN No. OF DEATHS FROM CARDIOVASCULAR AND RESPIRATORY CAUSES/DAY	ANNUAL INCOME <\$12,675	percentage of population	
						HIGH-SCHOOL GRADUATE	ANNUAL INCOME >\$100,000
Los Angeles	Los Angeles	8,863,164	148	87	14.8	70.0	7.9
New York	Bronx, Kings, New York, Richmond, Queens, Westchester	7,510,646	190.9	108.3	17.6	71.4	7.5
Chicago	Cook	5,105,067	113.9	62	14.0	73.4	5.5
Dallas-Fort Worth, Tex.	Collin, Dallas, Rockwall, Tarrant	3,312,553	47.9	26	11.7	79.0	5.6
Houston	Harris	2,818,199	39.9	20	15.5	74.0	5.5
San Diego, Calif.	San Diego	2,498,016	41.6	22.6	10.9	81.9	6.0
Santa Ana-Anaheim, Calif.	Orange	2,410,556	32.4	18.7	8.3	81.2	11.0
Phoenix, Ariz.	Maricopa	2,122,101	38.4	20.9	12.1	81.5	4.2
Detroit	Wayne	2,111,687	46.9	26.5	19.8	70.0	3.1
Miami	Dade	1,937,094	43.8	23.6	17.6	65.0	4.9
Philadelphia	Philadelphia	1,585,577	42.3	21.5	19.8	64.3	2.2
Minneapolis	Hennepin, Ramsey	1,518,195	26.3	13.9	9.7	87.2	5.5
Seattle	King	1,507,319	25.6	13.4	7.8	88.2	15.9
San Jose, Calif.	Santa Clara	1,497,577	19.7	10.7	7.3	82.0	11.4
Cleveland	Cuyahoga	1,412,141	36.5	20.1	13.5	74.0	4.0
San Bernardino, Calif.	San Bernardino	1,412,140	20.6	12.1	12.3	75.4	3.9
Pittsburgh	Allegheny	1,336,449	37.6	21.0	11.3	79.0	4.1
Oakland, Calif.	Alameda	1,279,182	22.2	12.2	10.3	81.4	6.7
Atlanta	Fulton, DeKalb	1,194,788	17.5	8.8	14.5	80.6	7.3
San Antonio, Tex.	Bexar	1,185,394	20.1	10.5	19.4	72.7	3.0

*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.^{15,22,23} 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.^{14,15,22,23}

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 PM₁₀ 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 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 predictor variables. The second-stage analysis provided an estimate of the effect of each predictor variable on the relative rate of mortality associated with PM₁₀.

Model fitting was performed with use of a Bayesian statistical approach,²⁴ 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. One important calculation is the posterior probability that the relative rate of mortality associated with PM₁₀ 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.²⁵

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 50 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.*

CITY OR METROPOLITAN AREA	NO. OF MONITORS	NO. OF DAYS ON WHICH DATA WERE COLLECTED		OZONE	PM ₁₀	NITROGEN DIOXIDE	SULFUR DIOXIDE	CARBON MONOXIDE
		OZONE	PM ₁₀					
		ppb			μg/m ³	ppb		
Los Angeles	7	2922	580	22.8 (6.9, 40.2)	46.0 (21.5, 73.1)	39.4 (23.2, 58.6)	1.9 (-0.2, 5.0)	15.1 (5.9, 28.3)
New York	15	2922	489	19.6 (7.3, 34.0)	28.8 (16.1, 44.8)	38.9 (27.0, 53.7)	12.8 (4.3, 25.1)	20.4 (14.8, 27.6)
Chicago	16	2922	2683	18.6 (6.1, 32.5)	35.6 (15.7, 60.3)	24.3 (14.4, 35.0)	4.6 (0.3, 10.3)	7.9 (4.5, 11.9)
Dallas-Fort Worth, Tex.	2	2922	624	25.3 (11.4, 41.2)	23.8 (11.4, 39.8)	13.8 (5.9, 22.7)	1.1 (-0.9, 4.3)	7.4 (3.6, 12.0)
Houston	2	2922	793	20.5 (9.3, 35.1)	30.0 (13.5, 48.6)	18.8 (9.0, 29.4)	2.8 (0.6, 5.6)	8.9 (4.0, 14.2)
San Diego, Calif.	4	2922	521	31.6 (18.1, 45.8)	33.6 (18.1, 52.1)	22.9 (11.2, 38.4)	1.7 (-0.3, 4.8)	11.0 (4.5, 20.5)
Santa Ana-Anaheim, Calif.	2	2922	480	23.0 (7.5, 38.5)	37.4 (18.4, 59.2)	35.1 (18.0, 59.0)	1.3 (-0.4, 4.0)	12.3 (3.7, 25.2)
Phoenix, Ariz.	10	2919	436	22.9 (10.3, 35.3)	39.7 (21.4, 58.4)	16.6 (8.8, 26.0)	3.5 (1.0, 6.6)	12.6 (5.4, 22.6)
Detroit	3	1861	1348	22.6 (9.1, 37.5)	40.9 (16.4, 71.1)	21.3 (11.5, 32.2)	6.4 (1.8, 12.4)	6.6 (3.2, 11.1)
Miami	4	2882	484	25.9 (14.5, 40.0)	25.7 (16.0, 36.6)	11.0 (4.5, 20.2)	NA	10.6 (6.5, 15.9)
Philadelphia	8	2901	495	20.5 (3.9, 39.5)	35.4 (19.0, 56.0)	32.2 (20.7, 45.0)	9.9 (1.7, 19.8)	11.8 (7.0, 17.2)
Minneapolis	8	NA	2764	NA	26.9 (10.9, 45.2)	17.6 (8.6, 27.4)	2.6 (0.1, 5.9)	11.8 (7.0, 17.0)
Seattle	7	1820	2205	19.4 (8.7, 30.0)	25.3 (10.2, 44.8)	NA	NA	17.8 (10.5, 26.4)
San Jose, Calif.	2	2922	945	17.9 (7.7, 28.1)	30.4 (9.3, 61.6)	25.1 (11.7, 44.1)	NA	9.4 (1.7, 21.3)
Cleveland	3	1712	1298	27.5 (12.7, 44.9)	45.1 (19.7, 78.7)	25.2 (15.2, 36.7)	10.3 (2.7, 19.9)	8.5 (3.7, 13.8)
San Bernardino, Calif.	8	2922	538	35.9 (14.5, 60.2)	37.0 (16.1, 56.2)	27.9 (15.3, 41.5)	0.7 (-0.7, 3.0)	10.3 (4.0, 17.5)
Pittsburgh	30	2883	2899	20.7 (7.0, 36.6)	31.6 (8.9, 61.2)	27.6 (17.6, 38.6)	14.2 (4.5, 26.5)	12.2 (6.1, 19.8)
Oakland, Calif.	3	2922	508	17.2 (7.7, 26.9)	26.3 (9.3, 47.8)	21.2 (9.6, 35.2)	NA	9.1 (2.9, 17.0)
Atlanta	3	2200	482	24.5 (11.6, 37.4)	34.4 (15.8, 56.4)	20.4 (11.7, 30.4)	6 (0.4, 14.0)	8 (3.2, 14.3)
San Antonio, Tex.	2	2918	670	22.2 (11.4, 34.5)	23.8 (12.3, 36.3)	NA	NA	10.1 (4.1, 17.3)

*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. PM₁₀ denotes particulate matter that is less than 10 μm in aerodynamic diameter, and NA not available.

TABLE 3. CORRELATION COEFFICIENTS OF ALL PAIRWISE CORRELATIONS BETWEEN POLLUTANTS FOR THE 20 CITIES AND METROPOLITAN AREAS.*

POLLUTANT	PM ₁₀	OZONE	NITROGEN DIOXIDE	SULFUR DIOXIDE	CARBON MONOXIDE
	median (10th and 90th percentiles)				
PM ₁₀	1.00	0.24 (-0.21, 0.41)	0.53 (0.22, 0.74)	0.39 (0.16, 0.51)	0.45 (0.15, 0.67)
Ozone		1.00	0.02 (-0.34, 0.20)	-0.06 (-0.31, 0.09)	-0.19 (-0.52, -0.04)
Nitrogen dioxide			1.00	0.51 (0.32, 0.70)	0.64 (0.51, 0.86)
Sulfur dioxide				1.00	0.41 (0.30, 0.71)
Carbon monoxide					1.00

*The correlation coefficients were calculated for values for all monitors within the cities. PM₁₀ denotes particulate matter that is less than 10 μm in aerodynamic diameter.

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 PM₁₀ 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 proba-

bility 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 PM₁₀ 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-

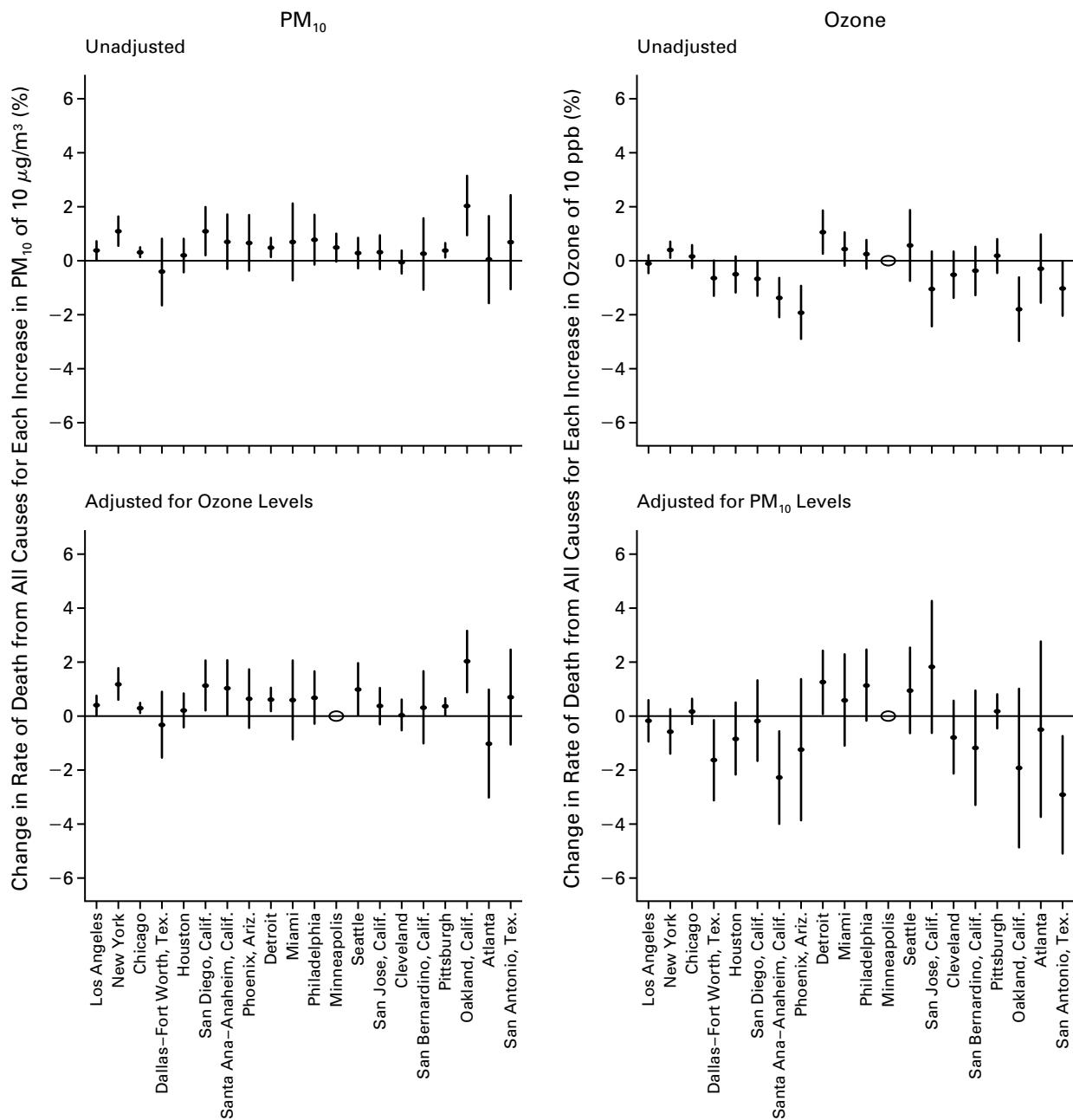


Figure 1. Regression Coefficients for the Changes in the Rate of Death from All Causes for Each Increase in the PM_{10} Level of $10 \mu\text{g}$ per Cubic Meter, before and after Adjustment for Ozone Levels, and for Each Increase in the Ozone Level of 10 ppb, before and after Adjustment for PM_{10} Levels in 20 Cities and Metropolitan Areas.

PM_{10} denotes particulate matter that is less than $10 \mu\text{m}$ in aerodynamic diameter. Bars indicate 95 percent confidence intervals. No data on ozone were available for Minneapolis.

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

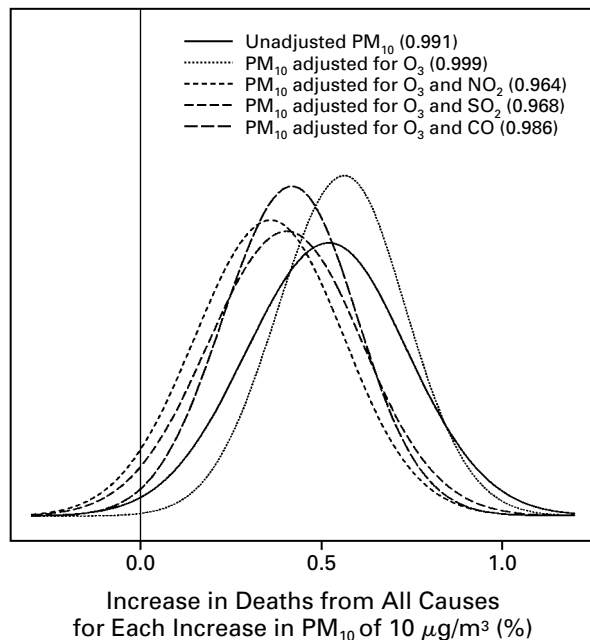


Figure 2. Posterior Distributions of the Overall Relative Rate of Increase in Death from All Causes for Each Increase in the PM₁₀ Level of 10 µg per Cubic Meter, before and after Adjustment for the Levels of Ozone (O₃), Nitrogen Dioxide (NO₂), Sulfur Dioxide (SO₂), and Carbon Monoxide (CO).

Values in parentheses are the posterior probabilities that the overall effects are greater than zero. PM₁₀ denotes particulate matter that is less than 10 µm in aerodynamic diameter.

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 PM₁₀ 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 PM₁₀ 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 PM₁₀ levels. After adjustment for PM₁₀ 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 PM₁₀ is associated with the rates of death from all causes and from cardiovascular and respiratory causes. The association of PM₁₀ was not affected by the inclusion of

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 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.³

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 $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$, so that the PM_{10} level is an imperfect surrogate for the $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$ 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.

Supported by a contract with the Health Effects Institute, an organization jointly funded by the Environmental Protection Agency (EPA R824835) and automotive manufacturers. The contents of this article do not necessarily reflect the views and policies of the Health Effects Institute, the Environmental Protection Agency, or manufacturers of motor vehicles or engines. Also supported by a grant from the National Institute of Environmental Health Sciences (P30 ES0 3819-12, to Johns Hopkins Center in Urban Environmental Health). Dr. Dominici is the recipient of a Rosenblith Young Investigator Award from the Health Effects Institute.

REFERENCES

1. Bascom R, Bromberg PA, Costa DL, et al. Health effects of outdoor air pollution. *Am J Respir Crit Care Med* 1996;153:477-98.
2. Dockery DW, Pope CA III. Acute respiratory effects of particulate air pollution. *Annu Rev Public Health* 1994;15:107-32.
3. Environmental Protection Agency, Office of Air Quality Planning and Standards. Review of the National Ambient Air Quality Standards for Particulate Matter: policy assessment of scientific and technical information. OAQPS staff paper no. EPA-452/R-96-013. Washington, D.C.: Government Printing Office, 1996.
4. Environmental Protection Agency (EPA). National ambient air quality standards for particulate matter. *Fed Regist* 1997;62:138.
5. Gamble JF, Lewis RJ. Health and respirable particulate (PM_{10}) air pollution: a causal or statistical association? *Environ Health Perspect* 1996; 104:838-50.
6. Gamble JF. $\text{PM}_{2.5}$ and mortality in long-term prospective cohort studies: cause-effect or statistical associations. *Environ Health Perspect* 1998; 106:535-49.
7. McClellan RO, Miller FJ. An overview of EPA's proposed revision of the particulate matter standard. *CIIT Act* 1997;17:1-24.
8. Kaiser J. Showdown over clean air science. *Science* 1997;277:466-9.
9. Samet JM, Zeger S, Dominici F, Dockery D, Schwartz J. The National Morbidity, Mortality, and Air Pollution Study (NMMAPS). 1. Methods and methodological issues. Cambridge, Mass.: Health Effects Institute, 2000. (See <http://healtheffects.org/pubs/samet.pdf>)
10. Katsouyanni K, Touloumi G, Spix C, et al. Short-term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from times series data from the APHEA project. *Air Pollution and Health: a European Approach*. *BMJ* 1997;314:1658-63.

11. Schwartz J, Dockery DW, Neas LM. Is daily mortality associated specifically with fine particles? *J Air Waste Manag Assoc* 1996;46:927-39.
12. Moolgavkar SH, Luebeck EG. A critical review of the evidence on particulate air pollution and mortality. *Epidemiology* 1996;7:420-8.
13. Lipfert FW, Wyzga RE. Air pollution and mortality: the implications of uncertainties in regression modeling and exposure measurement. *J Air Waste Manag Assoc* 1997;47:517-23.
14. Dominici F, Samet J, Zeger SL. Combining evidence on air pollution and daily mortality from the largest 20 U.S. cities: a hierarchical modeling strategy. *J R Stat Soc [A]* 2000;152:397-406.
15. Kelsall JE, Samet JM, Zeger SL, Xu J. Air pollution and mortality in Philadelphia, 1974-1988. *Am J Epidemiol* 1997;146:750-62.
16. Bureau of the Census. Statistical abstract of the United States. Washington, D.C.: Government Printing Office, 1990.
17. National Climatic Data Center databases TD-3280, TD-3281. Boulder, Colo.: EarthInfo, 1994.
18. Office of Air Quality Planning and Standards. Aerometric information retrieval system. Research Triangle Park, N.C.: Environment Protection Agency, 1999.
19. Hastie TJ, Tibshirani RJ. Generalized additive models. New York: Chapman & Hall, 1990.
20. Lindley DV, Smith AFM. Bayes estimates for the linear model. *J R Stat Soc [B]* 1972;34:1-41.
21. Morris CN, Normand S-L. Hierarchical models for combining information and for meta-analysis. In: Bernardo JM, Berger JO, Dawid AP, Smith AFM, eds. Bayesian statistics 4: proceedings of the Fourth Valencia International Meeting. Oxford, England: Oxford University Press, 1992: 321-44.
22. Samet JM, Zeger SL, Berhane K. The association of mortality and particulate air pollution. In: Particulate air pollution and daily mortality: replication and validation of selected studies: the phase I report of the Particle Epidemiology Evaluation Project. Cambridge, Mass.: Health Effects Institute, 1995.
23. Samet J, Zeger S, Kelsall J, Xu J, Kalkstein L. Air pollution, weather and mortality in Philadelphia. In: Particulate air pollution and daily mortality: analyses of the effects of weather and multiple air pollutants: the phase IB report of the Particle Epidemiology Evaluation Project. Cambridge, Mass.: Health Effects Institute, 1997.
24. Gelman A, Carlin JB, Stern HS, Rubin DB. Bayesian data analysis. London: Chapman & Hall, 1995.
25. S-PLUS version 3.4. Seattle: MathSoft, 1998 (software).
26. Samet JM, Zeger S, Dominici F, et al. The National Morbidity, Mortality, and Air Pollution Study (NMMAPS). 2. Morbidity and mortality from air pollution in the United States. Cambridge, Mass.: Health Effects Institute, 2000. (See <http://healtheffects.org/pubs/samet2.pdf>)
27. Dockery D, Pope A. Epidemiology of acute health effects: summary of time-series studies. In: Wilson R, Spengler JD, eds. Particles in our air: concentrations and health effects. Cambridge, Mass.: Harvard University Press, 1996:123-47.
28. Schwartz J. Air pollution and daily mortality: a review and meta analysis. *Environ Res* 1994;64:36-52.
29. Thurston GD, Ito K. Epidemiological studies of ozone exposure effects. In: Holgate ST, Samet JM, Koren HS, Maynard RL, eds. Air pollution and health. San Diego, Calif.: Academic Press, 1999:485-510.
30. Touloumi G, Katsouyanni K, Zmirou D, et al. Short-term effects of ambient oxidant exposure on mortality: a combined analysis within the APHEA project. *Am J Epidemiol* 1997;146:177-85.
31. Dockery DW, Pope CA III, Xu X, et al. An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 1993;329:1753-9.
32. Pope CA III, Thun MJ, Namboodiri MM, et al. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med* 1995;151:669-74.
33. Zeger SL, Dominici F, Samet J. Harvesting-resistant estimates of air pollution effects on mortality. *Epidemiology* 1999;10:171-5.
34. Schwartz J. Harvesting and long term exposure effects in the relation between air pollution and mortality. *Am J Epidemiol* 2000;151:440-8.