

Particulate Air Pollution and Mortality in 20 U.S. Cities: 1987-1994

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Abstract

Background: Time-series analyses have linked contemporary levels of air pollution, particularly particles, to daily mortality counts. These findings have contributed to the rationale for tightening air quality standards, but the validity of these findings has been questioned. We have developed analytic methods to address limitations of prior single-city time-series analyses by combining evidence across multiple locations.

Methods: Using a two-stage analytic approach that pools evidence from multiple locations, we have assessed the effects of five major outdoor air pollutants, particulate matter less than 10 μm in aerodynamic diameter (PM_{10}), ozone (O_3), carbon monoxide (CO), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2), on daily mortality in 20 of the largest cities in the United States, from 1987-1994.

Results: We found consistent evidence that PM_{10} is associated with total and cardiorespiratory mortality after taking into account potential confounding by other pollutants. For total mortality, the estimated relative rate was approximately a 0.5% increase in mortality per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} and the effect was not likely to be due to chance. There was weaker evidence that ozone increased mortality during the summer but not the winter months. Other pollutants did not have effects on mortality.

Conclusions: The analyses provide evidence that air pollution with particles is still adversely affecting the public's health and strengthen the rationale for limiting concentrations of respirable particles in outdoor air.

Key words: air pollution, mortality, epidemiology, particles, ozone, time series

Introduction

Recent studies showing that current levels of air pollution in cities of many developed and developing countries are associated with increased mortality and morbidity have raised concern that air pollution still poses a threat to public health¹⁻³. The evidence suggests that airborne particles are the toxic component of urban air pollution. Using this interpretation of the evidence as a rationale, the U.S. Environmental Protection Agency has implemented a new standard for fine particulate matter⁴. The existing standard, promulgated in 1987, specified 24-hour and annual standards for particulate matter less than 10 μ in aerodynamic diameter (PM₁₀). In 1997, the Agency added 24-hour and annual standards for PM_{2.5}, particulate matter less than 2.5 μ in aerodynamic diameter, corresponding better to the particles that penetrate to the lung's airways and alveoli. This decision has been controversial; critics question if the scientific evidence is sufficiently certain to take regulatory action⁵⁻⁸.

Key findings on particulate air pollution have come from time-series analyses of the association of air pollution levels with daily mortality counts³. With the exception of a few studies, the multi-city European APHEA (Air Pollution and Health: a European Approach) project⁹ and an analysis of data for six U.S. cities,¹⁰ most of these studies have been based on single locations selected without a defined sampling plan. Consequently, the generality of findings is uncertain and analysis 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^{7,11,12}.

To address these limitations, we combined information on the associations of the five major outdoor air pollutants -- PM₁₀, ozone (O₃), sulfur dioxide (SO₂), carbon monoxide (CO) and nitrogen dioxide (NO₂) -- with daily mortality from 20 of the largest U.S. cities¹³. Our estimates are based on a defined sample of the cities; statistical precision was enhanced by combining information from multiple locations.

Methods

Data

We began with the 20 largest counties by population (or with logical groupings of counties) and for analysis used data for the counties making up the associated cities, thus covering a population of over 50 million. Analysis was carried out at the county-level because county was the common coding unit for the various data sets. In this paper, we refer to “city” rather than county. Daily mortality counts were obtained from the National Center for Health Statistics (**Table 1**). After excluding deaths from external causes and of non-residents, we classified the deaths by age group (< 65 years, 65-74 years, and ≥ 75 years) and by cause (cardiorespiratory, and other)¹⁴. Data on selected demographic characteristics were obtained from the 1990 census¹⁵.

Hourly temperature and dew point data were available from the National Climatic Data Center, as assembled in the EarthInfo Compact Disc¹⁶ database. For analysis we used the 24-hour mean for each day. The air pollution data were obtained from the Aerometric Information Retrieval Service¹⁷ database

maintained by the U.S. Environmental Protection Agency. For population-oriented monitors, we downloaded all available data for PM₁₀, O₃, CO, SO₂, and NO₂. For the pollutants measured on an hourly basis, we calculated the 24-hour average. If multiple monitors were available for a metropolitan area, we averaged the data. To protect against the potential consequences of outlying values, we excluded the highest and lowest 10% of values (10% trimmed mean) and then averaged across monitors, after each monitor was corrected for its yearly average. Data were for 1987 to 1994.

Data Analysis

We used a two-stage log-linear regression model¹⁸⁻²⁰. In the first stage, a separate log-linear regression of observed daily mortality on air pollution measures and other confounders was fit to obtain estimates of the pollution relative rate and its statistical uncertainty for each of the 20 cities. In the second stage, the relative rate estimates were combined across cities (adjusting for the different levels of uncertainty) to obtain an overall estimate and to assess whether city specific characteristics were modifiers of the estimated effect of air pollution on mortality.

In the first stage log-linear regressions, we controlled for possible confounding by longer-term trends resulting from changes in population size and characteristics, health status, and health care and from shorter-term effects of seasonality and influenza epidemics. To do this, we used a flexible function that took into account variation in mortality over periods of several months (a smooth function of calendar time with seven degrees of freedom per year per city, allowed to differ across the three age groups). We

also allowed for the short-term effect of weather on mortality by including similar functions of the same day's temperature and the average temperature for the three previous days (6 degrees of freedom) along with comparable functions for dew point (3 degrees of freedom). Finally, we included indicator variables for day of the week. This model specification was based upon extensive, previously reported exploratory analyses^{14,21,22}. In this paper our results do not reflect the choice of degrees of freedom. We have found that the air pollution relative rates were not sensitive to the number of degrees of freedom selected for the smooth functions of time, temperature and dew point^{13,14,21,22}.

We explored lags for the pollutant variables in both the first stage analysis, ranging from using the current day's pollution data to data from the prior day or the day before the prior day (one-day and two-day lags individually). The overall effect did not vary with the selection of lag interval. Consequently, we show data for a one-day lag between pollution variables and mortality.

We considered the effects of multiple pollutants on mortality. We initially conducted univariate analyses for PM₁₀ alone and O₃ alone. We then considered the effects of these two pollutants in a bivariate model and then developed trivariate models adding either SO₂, NO₂, or CO to the bivariate model. The three-pollutant models provided estimates of the individual effects of CO, SO₂, and NO₂ on mortality with control for PM₁₀ and O₃.

The second stage of the analysis provides pooled estimates of the pollutant-specific relative rates and a characterization of the effects of air pollutants across the cities. We can also examine factors determining heterogeneity. In the second stage of the analysis for determinants of heterogeneity, the

first-stage estimates of the pollution relative rates are assumed to follow a linear regression with the selected city-specific demographics (**Table 1**) as predictor variables. The second-stage analysis provides an estimate of the effect of each predictor variable on the relative rate for PM₁₀.

Model fitting was performed using a Bayesian statistical approach²³ which provides, in addition to a point estimate and standard error, an estimate of the posterior distribution of the parameter of interest, e.g., the regression coefficient of mortality on PM₁₀. This analysis was carried out without making a strong prior assumption as to the relative rate's value (uninformative prior). The posterior distribution gives the probability that the relative rate of mortality for PM₁₀ has a particular value; that is, it is a measure of the strength of evidence. One posterior summary of interest is the posterior probability that the PM₁₀ relative rate of mortality is greater than zero. Using the posterior distribution it is also possible to generate 95% posterior intervals, another summary of interest. The 95% posterior intervals encompasses 95% of the posterior distribution, a Bayesian formulation comparable to the 95% confidence interval. All analyses were carried out using the statistical program Splus²⁴.

Results

The 20 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 PM₁₀ monitoring only every sixth day, data for O₃ and other pollutants were generally available for more days. Mean daily values for PM₁₀ ranged from about 20 µg/m³ to near 50 µg/m³ compared to the present 24-hr

standard for PM₁₀ of 150 µg/m³. The average numbers of daily deaths were substantial, ranging from 20 to nearly 200 (**Table 1**). Mean, 10th and 90th percentiles of all correlations between pollutants across the 20 cities are provided in **Table 3**. The correlation structure is generally consistent with the common sources of the primary combustion-related gases (SO₂, NO₂, and CO) and of PM₁₀. The concentration of O₃ was only slightly correlated with that of PM₁₀ and not with the gaseous pollutants.

In initial univariate analyses PM₁₀ was positively associated with total mortality in 19 of the 20 cities (**Figure 1**). The left side of **Figure 1** shows the individual-city analysis results for PM₁₀, the right for O₃. The associations of PM₁₀ with mortality were changed little with the addition of O₃ to the model, while the effects of O₃ tended to be more variable with the addition of PM₁₀. The one-pollutant analysis was also stratified by cause of mortality. The city-specific PM₁₀ mortality associations for cardiorespiratory mortality were similar to the patterns shown for total mortality. Univariate analysis stratified for age, reported in Dominici et al.¹³ showed no trend with age.

The analysis across the 20 cities confirmed the association of PM₁₀ with total mortality (**Figure 2**) and with cardiorespiratory deaths. **Figure 2** provides the posterior distributions of the relative rates of total mortality associated with PM₁₀ for the one-pollutant, two-pollutant, and three-pollutant statistical models. Numbers in the box indicate the probability that the PM₁₀ relative rate of mortality is greater than zero for each model. For total mortality, the distributions are shifted towards the right with their means (i.e. estimated relative rates) ranging between approximately 0.3% and 0.6% increase in daily mortality count per 10 µg/m³ increase in PM₁₀. A relative rate of 0.3% increase in mortality per 10 µg/m³ increase

in PM₁₀ corresponds to a relative risk of 1.003. In the model for total mortality including PM₁₀ alone, the estimated relative rate was 0.51% per 10 µg/m³ (95% Posterior Interval 0.07, 0.93). For cardiorespiratory deaths, the effect of PM₁₀ was somewhat greater than for total mortality, and there was an even greater probability that the effect is larger than zero. The estimated relative rate for cardiorespiratory mortality was 0.68% per 10 µg/m³ increase in PM₁₀ (95% Posterior Interval 0.20, 1.16). The posterior distributions for PM₁₀ did not change substantially with inclusion of other pollutants, suggesting that the univariate findings were not affected by confounding by other pollutants (**Figure 2**).

The univariate effects of O₃ were examined across the whole year and by season. Overall, the posterior probability distributions for the effects of O₃ were concentrated about zero and there was only an even chance that the effect was larger than zero separately for both total and cardiorespiratory deaths. Because ozone levels vary strongly with season, we compared the effects of O₃ during the three hottest summer months (June, July, August) when levels are highest and winter months (November, December, and January). With this stratification, the estimated relative rates of mortality change with O₃ (95% posterior interval) were 0.41% per 10 ppb (-0.20, 1.01) during the summer and -1.83% per 10 ppb (-2.69, -0.96) during the winter.

The between city differences in relative rates did not depend on city average PM₁₀ or O₃ levels nor on inclusion of city-specific demographics; for these variables, all associated 95% posterior intervals contained zero. Consequently, the analyses and results for PM₁₀ were not adjusted for these city-specific characteristics.

We also analyzed effects of CO, SO₂, and NO₂ in similar fashion to the PM₁₀ analysis. After controlling for PM₁₀ and O₃, we found little evidence in support of effects of these pollutants on mortality.

Discussion

We found consistent evidence that PM₁₀ is associated with total and cardiorespiratory mortality. The association of PM₁₀ was robust to the inclusion of other pollutants in the statistical model and to the choice of current or previous days' pollutant variables. This pooled analysis strongly supports the evidence from prior studies of particulate matter and mortality. These studies, largely based on data from single cities, used a variety of measures of particulate matter, including total suspended particles (TSP), Black Smoke, PM₁₀, and particulate matter less than 2.5 μ in aerodynamic diameter (PM_{2.5}). The statistical methodology used to assess the pollution/mortality relationships was also heterogeneous among the studies; for example, there was no uniformity in the approaches used to control for temporally varying factors or for other pollutants. Nonetheless, using a weight-of-evidence approach, the U.S. Environmental Protection Agency interpreted the study results as indicating a possibly causal association between particulate matter and adverse effects on health³.

In a meta-analysis of U.S. studies published through 1993, Dockery and Pope² estimated the relative rate of particulate air pollution on total mortality as an increase of 1 % per 10 μg/m³ of PM₁₀. In a subsequent update using reports published through 1995, there was little change in this estimate²⁵.

Schwartz²⁶ also carried out a meta-analysis of studies published through 1993, but included London and Minneapolis in addition to the eight cities considered by Dockery and Pope. The resulting estimated

relative rate was 0.7% increase in total mortality per $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} . In the APHEA project, a common analysis technique was applied to data from 12 European cities. Summary measures were then estimated in a second step. For the six western European cities, mortality was estimated to increase by 0.4% per $10 \mu\text{g}/\text{m}^3$ of PM_{10} . The estimate in our 20-city analysis, approximately 0.5% increase in mortality per $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} , is closely comparable to the APHEA project's⁹ estimate. The lower value, in comparison with the prior meta-analyses by Dockery and Pope and by Schwartz, may reflect differences in analysis techniques and the cities selected. The initial reports included in the meta-analysis may have been biased through selection for publication based on positive rather than null findings. Our 20-city estimate is not subject to bias from the selection of particular cities and should be preferred for the U.S.

The shape of the dose-response relationship between levels of air pollution and relative rate of mortality has implications for regulatory proposals. Daniels et al.²⁷ used this same data base of the 20 largest cities to characterize the shape of the dose-response curve for PM_{10} and mortality. Findings indicate that linear models are appropriate for assessing the effect of particulate air pollution on daily mortality even at current levels.

We did not find an effect of O_3 on total or cardiorespiratory mortality across the full year. Ozone was positively associated with mortality in the summer months when O_3 levels were highest, although the 95% interval included no effect of O_3 on mortality. The finding of a positive effect of O_3 in summer only may reflect the higher levels of O_3 during these months or possibly differing characteristics of

photochemical pollution across the seasons. Other recent studies have generally shown associations of O₃ with mortality²⁸. In the APHEA study, one-hour maximum O₃ levels were associated with daily numbers of deaths in four cities (London, Athens, Barcelona, and Paris) and a quantitatively similar effect was found in a group of four additional cities considered by the authors (Amsterdam, Basel, Geneva, and Zurich)²⁹. For an increase of 50 µg/m³ in the one-hour maximum, the estimated relative risk was 1.029 (1.1% per 10 ppb), using a random effects model for combining the city-specific data. Thurston and Ito²⁸ pooled data from 15 studies and estimated the relative risk of death to be 1.036 per 100 ppb (0.36% per 10 ppb) increase in the daily one-hour maximum. For the summer months, we estimated a comparable level of effect, 0.41% per 10 ppb. The findings of these three analyses (APHEA, Thurston and Ito, and the present 20-city study) provide consistent data that O₃ exposure also increases mortality.

Although we have analyzed a large data set based on 20 of the largest U.S. cities, limitations of the analyses need to be considered. Data on concentrations of PM_{2.5}, the respirable particles now also regulated by the Environmental Protection Agency, are not yet available nationally, as a monitoring network for particles in this size range is just being implemented. We used PM₁₀ because it has been monitored since 1987; there is variation across the United States in the proportion of PM₁₀ mass that is made up of PM_{2.5}³. Additionally, for regulatory purposes, monitoring of PM₁₀ is required only every six days, limiting the extent of available data.

These daily time series analyses also do not address the extent of life-shortening associated with these daily associations. The finding that the association is strongest for cardiorespiratory causes of

death is consistent with the hypothesis that persons made frail by advanced heart and lung disease are susceptible to air pollution. The findings from several epidemiologic studies of longer-term effects of air pollution on mortality suggest that air pollution may have a more severe effect than simply advancing death by a few days^{30,31}. Analyses of daily time-series data, conducted at different temporal frequencies, also indicate that the effect of air pollution may go beyond only shortening life by a few days^{32,33}.

We have not found evidence that key socioeconomic factors like poverty and race are modifiers, on the linear of the regression models, of the effect of PM₁₀ on mortality. It is also possible that the medical conditions and ill-health that predispose to higher risk are not be well captured by the socioeconomic indicators recorded by the Census, and more specific information on health status, rather than social factors may be needed to explore effect modification, particularly in relation to personal susceptibility. Finally, we have used county-level data for these social factors because our admissions and mortality data are on that level. But, the variation in socioeconomic status within the typical urban county is usually considerably larger than the variation across counties and the sociodemographic factors considered in the second stages of the models may be too ecologically coarse to be informative.

The epidemiologic evidence on particulate matter and mortality and morbidity has prompted the promulgation of a new standard for PM_{2.5} in the United States, and a rethinking of guidelines for particulate matter in Europe. Our analyses provide evidence that air pollution with particles is still adversely affecting the public's health and strengthen the rationale for limiting concentrations of respirable particles in outdoor air.

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Air Pollution and Mortality in 20 U.S. Cities

Table 1: Selected cities and counties, populations, average daily deaths for total mortality and for cardiorespiratory mortality, percentages of people in poverty, graduated from high school, and households with income greater than \$100,000.

City	County (ies)	Population	Mean Daily Deaths Total	Mean Daily Deaths Cardiovascular and Respiratory Deaths	% In Poverty	% High School Graduate	% Income > \$100K
1. Los Angeles	Los Angeles	8863164	148	87	14.8	70.0	7.9
2. New York	Bronx, Kings, New York, Richmond, Queens, Westchester	7510646	190.9	108.3	17.6	71.4	7.5
3. Chicago	Cook	5105067	113.9	62	14.0	73.4	5.5
4. Dallas-Ft. Worth	Collin, Dallas, Rockwall, Tarrant	3312553	47.9	26	11.7	79.0	5.6
5. Houston	Harris	2818199	39.9	20	15.5	74.0	5.5
6. San Diego	San Diego	2498016	41.6	22.6	10.9	81.9	6.0
7. Santa Ana-Anaheim	Orange	2410556	32.4	18.7	8.3	81.2	11.0
8. Phoenix	Maricopa	2122101	38.4	20.9	12.1	81.5	4.2
9. Detroit	Wayne	2111687	46.9	26.5	19.8	70.0	3.1
10. Miami	Dade	1937094	43.8	23.6	17.6	65.0	4.9
11. Philadelphia	Philadelphia	1585577	42.3	21.5	19.8	64.3	2.2
12. Minneapolis	Hennepin, Ramsey	1518195	26.3	13.9	9.7	87.2	5.5
13. Seattle	King	1507319	25.6	13.4	7.8	88.2	15.9

Air Pollution and Mortality in 20 U.S. Cities

City	County (ies)	Population	Mean Daily Deaths Total	Mean Daily Deaths Cardiovascular and Respiratory Deaths	% In Poverty	% High School Graduate	% Income > \$100K
14. San Jose	Santa Clara	1497577	19.7	10.7	7.3	82.0	11.4
15. Cleveland	Cuyahoga	1412141	36.5	20.1	13.5	74.0	4.0
16. San Bernardino	San Bernardino	1412140	20.6	12.1	12.3	75.4	3.9
17. Pittsburgh	Allegheny	1336449	37.6	21.0	11.3	79.0	4.1
18. Oakland	Alameda	1279182	22.2	12.2	10.3	81.4	6.7
19. Atlanta	Fulton, De Kalb	1194788	17.5	8.8	14.5	80.6	7.3
20. San Antonio	Bexar	1185394	20.1	10.5	19.4	72.7	3.0

Air Pollution and Mortality in 20 U.S. Cities

Table 2: Number of monitors (N_{Mon}) recording PM₁₀ levels, days with O₃ and PM₁₀ data, means and 10-th and 90-th percentiles () of pollutants by city.

Locations	N _{Mon}	Days with O ₃ Data	Days with PM ₁₀ Data	O ₃ (ppb)	PM ₁₀ (µg/m ³)	NO ₂ (ppb)	SO ₂ (ppb)	CO (ppb/100)
1. 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)
2. 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)
3. 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)
4. Dallas-Fort Worth	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)
5. 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)
6. San Diego	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)
7. Santa Ana – Anaheim	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)
8. Phoenix	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)
9. 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)
10. 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)
11. 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)
12. Minneapolis	8	NA ²	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)
13. Seattle	7	1820	2205	19.4 (8.7,30.0)	25.3 (10.2,44.8)	NA	NA	17.8 (10.5,26.4)
14. San Jose	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)
15. 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)
16. San Bernadino	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)

Air Pollution and Mortality in 20 U.S. Cities

Locations	N _{Mon}	Days with O ₃ Data	Days with PM ¹⁰ Data	O ₃ (ppb)	PM ₁₀ (µg/m ³)	NO ₂ (ppb)	SO ₂ (ppb)	CO (ppb/100)
17. 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)
18. Oakland	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)
19. 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)
20. San Antonio	2	2918	670	22.2 (11.4,34.5)	23.8 (12.3,36.3)	NA	NA	10.1 (4.1,17.3)

NA = Data Not Available.

Table 3: Median (10th and 90th percentiles) of all pairwise correlations* of pollutants for the 20 cities.

	PM ₁₀	O ₃	NO ₂	SO ₂	CO
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)
O ₃		1.00	0.02 (-0.34,0.20)	-0.06 (-0.31,0.09)	-0.19 (-0.52,-0.04)
NO ₂			1.00	0.51 (0.32,0.70)	0.64 (.51,0.86)
SO ₂				1.00	0.41 (0.30,0.71)
CO					1.00

* The correlation coefficients were calculated for values for all monitors within the cities. The table provides the median and the 10th and 90th percentile values for these correlation coefficients.

Table 4: Means and 95% confidence intervals () of the relative rate of PM₁₀ on total and cardiorespiratory mortality for the univariate, bivariate, and trivariate results shown in Figures 3 and 4.

Statistical Model	Total Mortality	Cardiorespiratory Mortality
PM ₁₀	0.51 (0.07,0.93)	0.68 (0.20,1.16)
PM ₁₀ +O ₃	0.55 (0.21,0.90)	0.74 (0.35,1.15)
PM ₁₀ +O ₃ +NO ₂	0.35 (-0.04,0.76)	0.56 (0.08,1.05)
PM ₁₀ +O ₃ +SO ₂	0.40 (-0.03,0.82)	0.52 (0.07,1.00)
PM ₁₀ +O ₃ +CO	0.41 (0.05,0.78)	0.50 (0.08,0.93)