

# The Lag Structure Between Particulate Air Pollution and Respiratory and Cardiovascular Deaths in 10 US Cities

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*To assess differences in the lag structure pattern between particulate matter <math><10\ \mu\text{m}/100\ \mu\text{m}</math> in diameter ( $\text{PM}_{10}$ ) and cause-specific mortality, we performed a time-series analysis in 10 US cities using generalized additive Poisson regressions in each city; nonparametric smooth functions were used to control for long time trend, weather, and day of the week. The  $\text{PM}_{10}$  effect was estimated based on its daily mean, 2-day moving average, and the cumulative 7-day effect by means of an unconstrained distributed lag model. A  $10\text{-}\mu\text{g}/\text{m}^3$  increase in the 7-day mean of  $\text{PM}_{10}$  was associated with increases in deaths due to pneumonia (2.7%, 95% confidence interval [CI]: 1.5, 3.9), chronic obstructive pulmonary disease (1.7%, 95% CI: 0.1, 3.3), and all cardiovascular diseases (1.0%, 95% CI: 0.6, 1.4). A  $10\text{-}\mu\text{g}/\text{m}^3$  increase in the 2-day mean of  $\text{PM}_{10}$  was associated with a 0.7% (95% CI: 0.3, 1.1) increase in deaths from myocardial infarction. When the distributed lag was assessed, two different patterns could be observed: respiratory deaths were more affected by air pollution levels on the previous days, whereas cardiovascular deaths were more affected by same-day pollution. These results contribute to the overall efforts so far in understanding how exposure to air pollution promotes adverse health effects. (J Occup Environ Med. 2001;43:927-933)*

**I**n past decades, numerous studies have reported associations between daily fluctuations in air pollution and daily fluctuations in deaths in urban areas.<sup>1-7</sup> These associations have been seen with pollution levels on the same day or within a few previous days.<sup>8</sup>

Recently, we demonstrated that the effects of air pollution on daily deaths from all causes were distributed over several subsequent days.<sup>9</sup> One possible reason for this is that different causes of death are affected with different latency periods by air pollution. Further support for this comes from Rossi and coworkers,<sup>10</sup> who examined cause-specific mortality in Milan and reported different lags for different causes. A study in a single location risks variations in the lag between pollution and daily deaths that are due to sampling variability rather than related to a causal pathway.

A multicity approach to this issue would be much more convincing. Further, even for cause-specific mortality, the effects of pollution may be spread over more than 1 day, and discovering that relationship for specific causes of deaths may provide some insight into possible mechanisms. A systematic approach to investigate the distribution of effect over time offers the possibility of elucidating this question, and we applied such a methodology to examine the association of pollution from particulate matter <math><10\ \mu\text{m}/100\ \mu\text{m}</math> in diameter and cause-specific mortal-

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ity in 10 cities across the United States.

## Data

To effectively analyze the distributed lag between particulate matter <10  $\mu\text{m}$ /100  $\mu\text{m}$  in diameter ( $\text{PM}_{10}$ ) and daily deaths, we obtained daily  $\text{PM}_{10}$  measurements. Most of the US cities measured  $\text{PM}_{10}$  only on 1 day of 6, but a number of locations monitored daily. We selected 10 US cities with daily  $\text{PM}_{10}$  monitoring to provide a reasonable number of locations for a combined analysis: Birmingham, Alabama; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis–St. Paul, Minnesota; New Haven, Connecticut; Pittsburgh, Pennsylvania; and Seattle and Spokane, Washington.

Daily counts of deaths due to pneumonia (International Classification of Diseases, 9th Revision [ICD-9]: 480–487), chronic obstructive pulmonary diseases (COPD) (ICD-9: 490–496), all cardiovascular diseases (ICD-9: 390–429), and specifically myocardial infarction (ICD-9: 410) in the metropolitan county containing each city were extracted from National Center for Health Statistics mortality tapes for the years 1986 through 1993. Minneapolis and St. Paul were combined and treated as one city. Daily weather data were obtained from the nearest airport station (EarthInfo CD NCDC Surface Airways; EarthInfo Inc, Boulder, CO), and daily concentrations of  $\text{PM}_{10}$  were obtained from the US Environmental Protection Agency's Aerometric Retrieval System monitoring network.

Many of the cities have more than one monitoring location for  $\text{PM}_{10}$ . Some operate on a daily basis; others on every third or sixth day. If the monitoring results were simply averaged, the daily mean would change on days when new monitors were included merely because their annual average differed from the monitoring station that operated on a daily basis. The variance of  $\text{PM}_{10}$  measure-

ments also can differ from monitoring location to location. Day-to-day changes in which monitoring results are averaged would also result in changes in the day-to-day variation in the exposure measurements that would not represent true changes in exposure but rather changes in the sampling of the monitors. To remove these influences, we used the following algorithm. The annual mean was computed for each monitor for each year and was subtracted from the daily values of that monitor. We then standardized these daily deviances from each monitor's annual average by dividing by the standard deviation of that monitor. The daily-standardized deviations for each monitor on each day were averaged, producing a daily averaged standardized deviation. We multiplied this by the standard deviation of all centered monitor readings for the entire year, and we added back in the annual average of all of the monitors. This gave a daily average  $\text{PM}_{10}$  concentration for each day in each city. This approach has been applied successfully in previous analyses.<sup>9,11</sup>

## Analytical Methods

For each city, a generalized additive Poisson regression was fit,<sup>12,13</sup> modeling the logarithm of the expected value of daily deaths as a sum of the smooth functions of the predictor variables. The generalized additive model allows regressions to include nonparametric smooth functions to model the potential nonlinear dependence of the daily admissions on weather and season. It was assumed that:

$$\log(E(Y)) = \beta_0 + S_1(X_1) + \dots + S_p(X_p), \quad (1)$$

where  $Y$  is the daily count of deaths,  $E(Y)$  is the expected value of that count,  $X_i$  is the covariate, and  $S_i$  is the smooth (ie, continuously differentiable) function. For the  $S_i$  we used loess,<sup>14</sup> a moving regression smoother. This approach is now standard in air pollution time-series.<sup>15</sup> For each covariate, it is necessary to

choose a smoothing parameter that determines how smooth the function of that covariate should be. Three classes of predictor variables were used: a smooth function of time to capture seasonal and other long-term trends in the data, weather and day of the week variables to capture short-term potential confounding, and  $\text{PM}_{10}$ . The choice of smoothing parameter for each set of variables is described below. The purpose of the smooth function of time is to remove the basic long-term pattern from the data. Seasonal patterns can vary greatly between Birmingham and Spokane, for example, and a separate smoothing parameter was chosen in each city to eliminate seasonal patterns in the residuals and to reduce the residuals of the regression to "white noise"<sup>16</sup> (ie, remove serial correlation). This approach was used because each death is an independent event, and autocorrelation in residuals indicates there are omitted, time-dependent covariates whose variation may confound air pollution. If the autocorrelation is removed, the remaining variation in omitted covariates has no systematic temporal pattern; hence, confounding is less likely. This approach was described previously.<sup>16</sup> Sometimes it was necessary to incorporate autoregressive terms<sup>17</sup> to eliminate serial correlation from the residuals.

The other covariates were temperature, relative humidity, and barometric pressure on the same day; the previous day's temperature; and the day of the week. To allow for city-specific differences, the smoothing parameters for these covariates were also chosen separately in each location to minimize Akaike's Information Criteria.<sup>18</sup>

$\text{PM}_{10}$  was treated as having a linear association with deaths in this analysis to facilitate the combination of coefficients across cities and the examination of lag structure. Robust regression was used to reduce sensitivity to outliers in the dependent variable. To reduce sensitivity to outliers in the pollution variable, the

**TABLE 1**  
The Populations and Daily Means of Deaths in the 10 Locations

Cities	1990 Pop- ulation	Deaths*				
		Total	Pneumonia	COPD	CVD	MI
Birmingham	907,810	19.1	0.6	0.7	5.9	1.5
Canton	367,585	9.9	0.3	0.5	3.5	1.2
Chicago	5,105,067	133.4	5.0	4.1	48.4	15.2
Colorado Springs	397,014	6.0	0.3	0.4	1.6	0.5
Detroit	2,111,687	59.7	1.8	1.9	22.8	6.2
Minneapolis	1,518,196	32.3	1.3	1.4	9.7	2.4
New Haven	804,219	20.4	0.8	0.7	7.5	1.7
Pittsburgh	1,336,449	42.4	1.3	1.6	16.1	5.1
Seattle	1,507,319	29.3	1.1	1.4	9.4	2.3
Spokane	361,364	8.7	0.3	0.5	2.9	0.9

\* COPD, chronic obstructive pulmonary disease; CVD, all cardiovascular diseases; MI, myocardial infarction.

baseline analysis was restricted to days when PM<sub>10</sub> levels were below 150 µg/m<sup>3</sup>, the currently enforced ambient standard. This also ensured that the results were unambiguously relevant to questions of revision of those standards.

**Distributed Lag Models**

Distributed lag models have been used for decades in the social sciences,<sup>19</sup> and Pope and Schwartz<sup>20</sup> recently described the use of this approach in epidemiology. We recently applied this method to estimating the distributed lag between all-cause mortality and daily deaths in these 10 locations.<sup>9</sup> The motivation for the distributed lag model is the realization that air pollution can affect deaths occurring not only on the same day but also on several subsequent days. The converse is therefore also true: deaths today will depend on the same-day effect of today's pollution levels, the 1-day-lag effects of yesterday's PM<sub>10</sub> concentrations, and so forth. Therefore, suppressing covariates and assuming Gaussian data in this instance, the *unconstrained* distributed lag model assumes:

$$Y_t = \alpha + \beta_0 X_t + \dots + \beta_q X_{t-q} + \epsilon_t, \tag{2}$$

where  $X_{t-q}$  is the PM<sub>10</sub> concentration  $q$  days before the deaths. The overall effect of a unit increase in air pollution on a single day is its impact on

that day plus that on subsequent days; that is, it is the sum of  $\beta_0 + \dots + \beta_q$ .<sup>16</sup> To simplify this, equation 2 can be recast as:

$$Y_t = \alpha + \beta^*(\omega_0 X_t + \dots + \omega_q X_{t-q}) + \epsilon_t, \tag{3}$$

where the  $\omega_i$  are weights that sum to 1, and  $\beta^*$  is  $\beta_0 + \dots + \beta_q$ . That is,  $\beta^*$  is also interpretable as the marginal effect of a unit increase in a weighted average pollution variable. Because a unit of increase in pollution on a single day increases the weighted average on all  $q$  subsequent days, the effect of that single day's increase will be  $\beta^* \omega_i$  on each of the  $q$  subsequent days, or  $\beta^*$  overall.

Because there is substantial correlation between air pollution concentrations on days that are close together, the above regression will have a high degree of collinearity. This will result in unstable estimates of the individual  $\beta_q$ 's and, hence, poor estimates of the shape of the distribution of the effect over lag. However, the sum of the individual  $\beta_q$ 's are an unbiased estimate of the overall effect of a unit increase in pollution, and the individual  $\beta_q$ 's are also unbiased. Thus, by fitting the same model in 10 different locations, and by combining effect size estimates, by lag, over the cities, we can obtain an unbiased estimate of the distribution of the effect over time with no constraints. Note that the use of a single day's exposure is a con-

strained lag model. In that case, we fit equation 2 with the constrain that  $\beta_1 = \beta_2 = \dots = \beta_p = 0$ . If we are unsure that the pollution effects are limited to a single day, these constraints are quite restrictive and are therefore likely to introduce bias into the estimated overall effect. To see if the traditional approaches of using a 1- or 2-day moving average resulted in a downward bias in the estimated effects of PM<sub>10</sub>, we also fit those models in each city.

To combine results across cities, we used inverse variance-weighted averaging to estimate the overall effect for lag 0, lag 0-1. For the distributed lag model, we used a multivariate approach to pool the estimates of each day's lag simultaneously. The inverse variance-weighted average (and overall covariance matrix) was computed using the variance-covariance matrix of the estimated coefficients in each city. This approach incorporated the correlations among the estimated effects at different lags.

**Results**

Table 1 shows the populations and the mean values of the cause-specific deaths in the 10 study locations. Table 2 shows the mean values of the environmental variables in these cities.

As presented previously,<sup>9</sup> the correlation between PM<sub>10</sub> and barometric pressure was quite small, ranging

**TABLE 2**  
Daily Means of the Environmental Variables in the 10 Study Locations

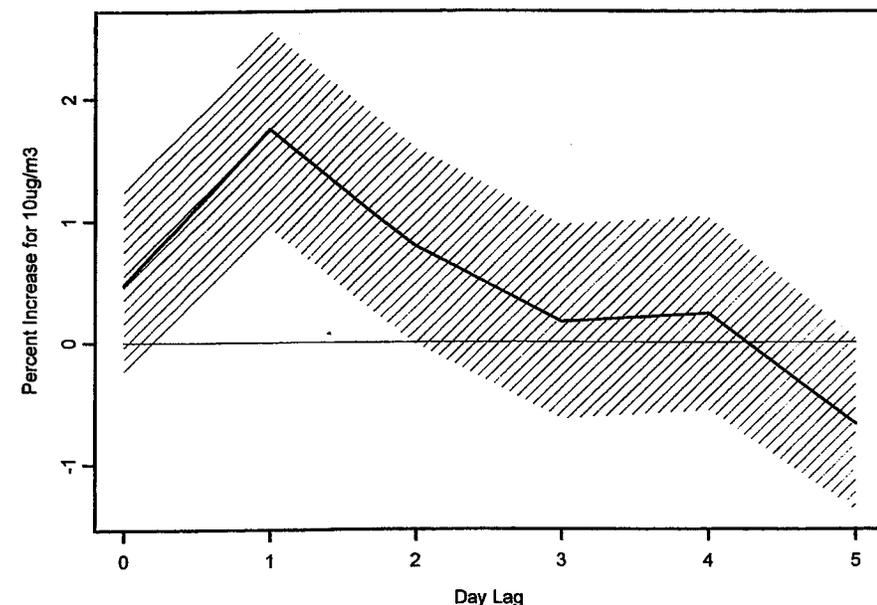
Cities	PM <sub>10</sub> ( $\mu\text{g}/\text{m}^3$ )*	Humidity (%)	Pressure (mm Hg)	Temperature (°C)
Birmingham	34.8	70.5	29.4	16.9
Canton	28.6	73.7	28.7	10.0
Chicago	36.1	70.8	29.3	10.1
Colorado Springs	27.2	52.5	24.0	9.5
Detroit	36.4	69.2	29.3	10.5
Minneapolis	28.0	68.7	29.1	7.9
New Haven	28.4	66.8	29.8	10.7
Pittsburgh	35.9	69.3	28.8	11.2
Seattle	32.1	75.5	29.6	11.4
Spokane	43.1	66.6	27.5	8.8

\* PM<sub>10</sub>, particulate matter >10  $\mu\text{m}/100 \mu\text{m}$  in diameter.

**TABLE 3**  
Percentage Increases in Daily Cause-Specific Deaths and 95% CI due to a 10- $\mu\text{g}/\text{m}^3$  Increase in PM<sub>10</sub> Levels\*

Causes	Increases in Daily Deaths					
	Distributed Lag Model		Lag 0-1		Lag 0	
	%	95% CI	%	95% CI	%	95% CI
PN	2.7	1.5, 3.9	6.6	-4.1, 17.3	1.4	0.2, 2.6
COPD	1.7	0.1, 3.3	1.5	0.7, 2.3	0.7	0.1, 1.3
CVD	1.0	0.6, 1.4	0.8	0.6, 1.0	0.6	0.4, 0.8
MI	0.6	0.0, 1.2	0.7	0.3, 1.1	0.6	0.2, 1.0

\* CI, confidence interval; PM<sub>10</sub>, particulate matter >10  $\mu\text{m}/100 \mu\text{m}$  in diameter; PN, pneumonia; COPD, chronic obstructive pulmonary disease; CVD, all cardiovascular diseases; MI, myocardial infarction.



**Fig 1.** Distributed lag for the association between a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> and deaths from pneumonia.

from -0.05 in Detroit to 0.24 in Seattle. The correlation between PM<sub>10</sub> and relative humidity was ever negative, ranging from -0.35 in Minneapolis to -0.11 in Colorado Springs and Seattle. The correlation between PM<sub>10</sub> and temperature ranged from -0.34 in Colorado Springs to 0.45 in Pittsburgh.

### PM<sub>10</sub> Results

Table 3 shows the estimated effect of a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> in each city using the concurrent day's pollution, the 2-day moving average, and the unconstrained distributed lag model. For deaths from respiratory and all cardiovascular diseases, the distributed lag models had substantially greater overall impact than models using only a single day's exposure. When the distributed lag models were compared with those using the 2-day moving average, no significant differences were detected, except for pneumonia. The estimated increase in myocardial infarction was similar whatever the approach used, suggesting a shorter response period for this outcome.

Figure 1 shows the distributed lag structure for the estimated increase in pneumonia deaths due to a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> in the 10 cities on the same day and on the 5 previous days. The particulate pollution of the previous day contributed more to the outcome, with a minor contribution of the pollution of 2 days before. For COPD (Fig. 2), exposure to air pollution on the 2 previous days presented almost the same contribution to COPD deaths. For both causes of deaths, exposure to the particulate air pollution on the same day of the event was not significant.

When the impact of a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> on cardiovascular deaths was analyzed, we could see a different pattern from that observed for respiratory deaths. The contribution of the air pollution levels on the same day of the event increased substantially when compared with its contribution to respiratory deaths,

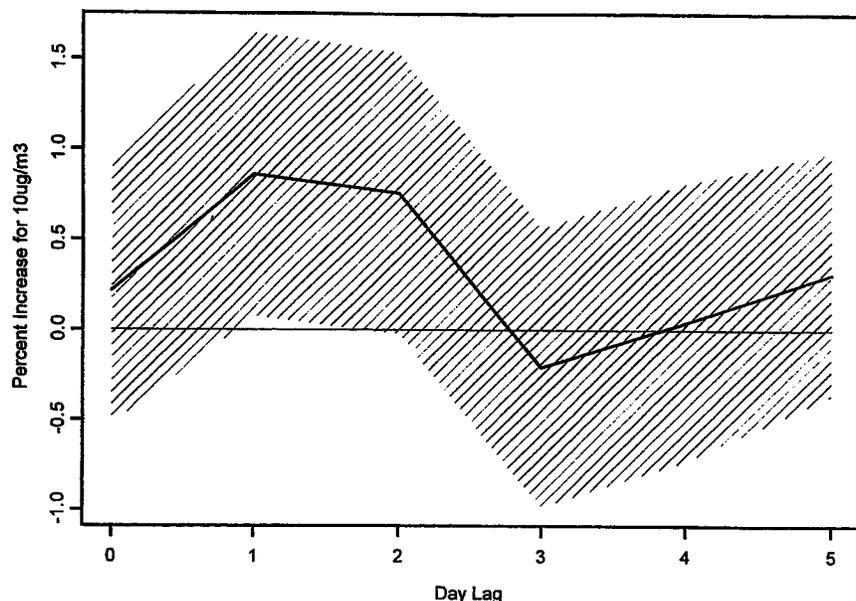


Fig 2. Distributed lag for the association between a 10-µg/m<sup>3</sup> increase in PM<sub>10</sub> and COPD deaths.

characterizing a more acute health effect. Figure 3 shows that for all cardiovascular diseases, the previous-day and the same-day exposure (and to a lesser extent, exposure 2 days before) contributed to the deaths. When deaths due to myocardial infarction were analyzed (Fig. 4), exposure on the same day seemed to be the main contributor

to deaths, with exposure on the day before also presenting a positive association.

### Discussion

The present study shows PM<sub>10</sub> positively associated with respiratory and cardiovascular mortality in a meta-regression analysis of 10 US counties. The study is coherent and

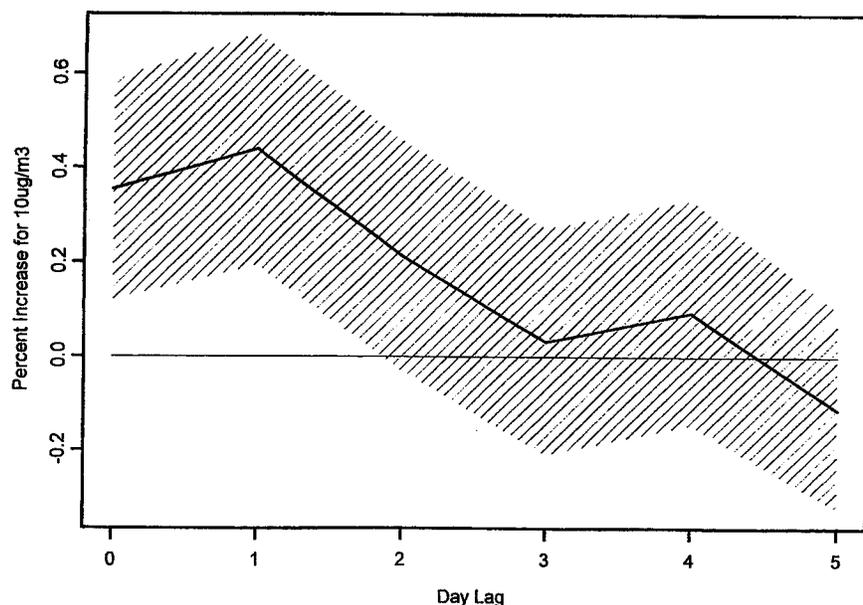


Fig 3. Distributed lag for the association between a 10-µg/m<sup>3</sup> increase in PM<sub>10</sub> and deaths from all cardiovascular diseases.

in agreement with a previous study of the same cities that used the same methodological approach to assess the effects of PM<sub>10</sub> on daily deaths from all causes.<sup>9</sup>

However, our interest was focused on assessing differences in the lag structure pattern between particulate matter and cause-specific mortality. When we looked at the unconstrained distributed lag models, two different patterns could be identified. For respiratory deaths, exposure to particulate pollution on the previous days (lag 1 for pneumonia deaths and lags 1 and 2 for COPD deaths) had a greater effect than same-day exposure. For cardiovascular deaths, immediate exposure (specifically, same-day) contributed more consistently to the deaths. When deaths from all cardiovascular diseases were analyzed, the effect of exposure to particulate pollution was almost the same for the same day and for the day before the event (and to a lesser extent, for the 2 days before). For myocardial infarction, a more acute response was observed, and same-day exposure to air pollution was seen as the main contributor to the deaths.

Based on the biological plausibility, the differences in the lag structure observed in this study are admissible. A wider and more lagged structure can be expected for deaths from respiratory diseases (eg, COPD, and even pneumonia, an infectious disease) that progress more slowly. On the other hand, cardiovascular diseases (particularly myocardial infarction) represent an acute response to a trigger. Therefore, fatal cardiovascular events occurring on the same day of the exposure are quite plausible.

The evidence for a more immediate impact of particles on cardiovascular disease has some support from both experimental and human studies. Godleski and coworkers<sup>21</sup> reported that heart rate variability in dogs in exposure chambers responded within hours to changes in the concentration of the particles

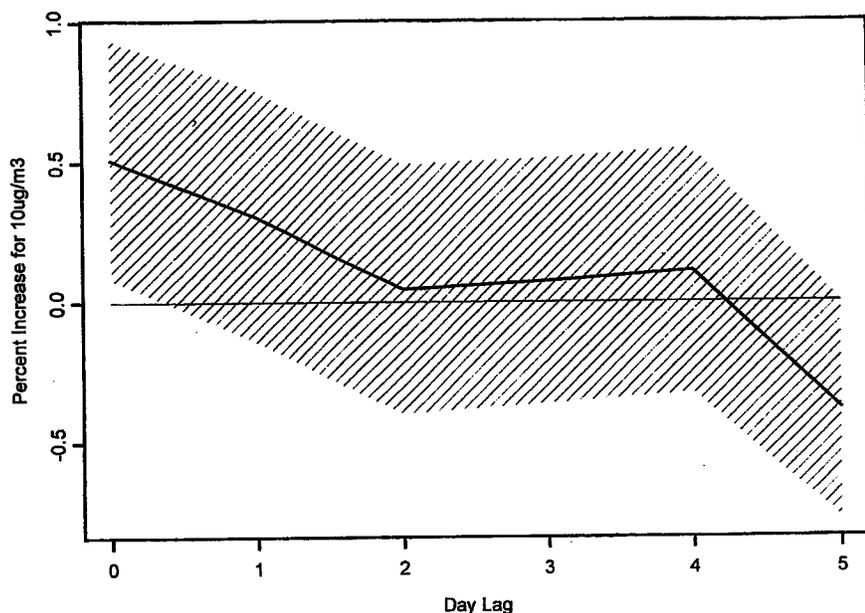


Fig 4. Distributed lag for the association between a 10- $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$  and deaths from myocardial infarction.

they were exposed to. Gold and coworkers<sup>22</sup> also reported the strongest association between heart rate variability and  $\text{PM}_{2.5}$  within 4 hours of exposure. Healthy young adults exposed to concentrated air particles in an exposure chamber also had increases in plasma fibrinogen in the same day.<sup>23</sup>

On the other hand, Peters and coworkers<sup>24</sup> reported a lag of over a day between exposure to air pollution and incidence of arrhythmias in a cohort of patients with implanted cardiovascular defibrillators. These results are not necessarily inconsistent with ours; it was primarily myocardial infarction that seemed to show an immediate response to air pollution in our study, with other cardiovascular deaths showing a more lagged response. However, Rossi and coworkers<sup>10</sup> reported lag structures for respiratory and cardiovascular deaths in Milan, Italy, that are different from those reported in this article. Same-day exposure was associated with pneumonia and heart failure deaths, whereas myocardial infarction and COPD deaths were associated with the mean of the 3 and 4 previous days.

When we adopted a metaanalytic approach to analyzing the association between  $\text{PM}_{10}$  and cause-specific deaths in 10 different cities, we attempted to control for stochastic variability and possible variability in exposure measurements or in the death records that could induce noise in both the estimated effect and the lag structure. The differences between respiratory and cardiovascular deaths, as presented in this study, define patterns of lag structure that seem to be more plausible and coherent estimates.

In summary, we present a strong and positive association between  $\text{PM}_{10}$  and cause-specific mortality across 10 US cities. Cardiovascular deaths represented a more acute response to air pollution exposure, with deaths occurring on the same day. For respiratory deaths, exposure to air pollution levels on the 1 or 2 previous days showed a greater effect. These results contribute to the overall efforts made toward understanding how exposure to air pollution promotes adverse health effects.

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#### Facts

- Number of members of Congress or their staffers who failed to file a tax return or pay back taxes in 1999: 856.
- Average number of network news minutes devoted to Presidents Clinton and Bush, respectively, during each of first 50 days in office: 18.04 and 9.24.
- Amounts pledged by Iraq last December to support Palestinian Intifada and in January for "poor Americans," respectively: \$930,000,000 and \$93,000,000.
- Estimated number of Americans eligible for food stamps who have not applied: 12,400,000.
- Ratio of the weight of a croissant at Au Bon Pain to that at a typical Parisian bakery: 2:1.
- Ratio of per capita use of Ritalin in Vermont to that in California: 3:1.
- Percentage of 77,000 dams in United States producing electricity: 3.
- Percentage of electricity produced by dams in Canada and United States, respectively: 62 and 10.
- Chance that a Catholic saint named in the past 400 years was canonized by Pope John Paul II: 1 in 2.

—HARPER'S INDEX. *Harper's.* 2001;302(1813):11.

