Short-term changes in ambient particulate matter with aerodynamic diameters < 10 µm (PM_{10}) have been associated with short-term fluctuations in mortality or morbidity in many studies. In this study, we tested whether those deaths are just advanced by a few days or weeks using a multicity hierarchical modeling approach for all-cause, respiratory, and cardiovascular deaths, for all ages and stratifying by age groups, within the APHEA-2 (Air Pollution and Health: A European Approach) project. We fit a Poisson regression and used an unconstrained distributed lag model to model the effect of PM_{10} exposure on deaths up to 40 days after the exposure. In baseline models using PM_{10} the day of and day before the death, we found that the overall PM_{10} effect (per 10 µg/m^3) was 0.74% (95% confidence interval 95% CI), –0.17 to 1.66] for respiratory deaths and 0.69% (95% CI, 0.31–1.08) for cardiovascular deaths. In unrestricted distributed lag models, the effect estimates increased to 4.2% (95% CI, 1.08–7.42) for respiratory deaths and to 1.97% (95% CI, 1.38–2.55) for cardiovascular deaths. Our study confirms that most of the effect of air pollution is not simply advanced by a few weeks and that effects persist for more than a month after exposure. The effect size estimate for PM_{10} doubles when we considered longer-term effects for all deaths and for cardiovascular deaths and becomes five times higher for respiratory deaths. We found similar effects when stratifying by age groups. These larger effects are important for risk assessment. Key words: air pollution, heart disease, hierarchical models, lung disease, mortality displacement. Environ Health Perspect 111:1188–1193 (2003). doi:10.1289/ehp.5712 available via http://dx.doi.org/ [Online 6 March 2003]
pattern between particulate matter with aerodynamic diameters < 10 µm (PM10) and cause-specific mortality in a multicity hierarchical model. Only one study (Schwartz 2000c) has looked at mortality displacement with regard to cause-specific deaths.

Some studies found an association between airborne particles and mortality by age groups (Bremner et al. 1999; Goldberg et al. 2000; Ostro et al. 1999a; Ponka et al. 1998; Verhoef et al. 1996), but again, none analyzed the distribution of the effect over time since exposure and whether the mortality displacement is short term.

In our previous study of the APHEA-2 (Air Pollution and Health: A European Approach) project (Zanobetti et al. 2002), we examined mortality displacement between air pollution and all daily deaths. The goal of that analysis was to estimate the dependence of daily deaths on day \( t \) on PM10 on that day, and up to the previous 40 days using an unconstrained distributed lag model. As a sensitivity analysis, in that study we used a fourth-degree polynomial and a cubic distributed lag model, and all the models showed similar results.

In the present study, within the APHEA-2 project, we examined mortality displacement by age, and we looked at the differences between cardiovascular and respiratory deaths compared with all causes of deaths. Following the same approach as in our previous analysis, we applied a fourth-degree polynomial and unconstrained distributed lag models in a multicity hierarchical framework.

Data and Methods

Health data. The APHEA-2 project was a multicenter study of 30 cities across Europe and adjacent countries (e.g., Istanbul, Turkey, and Tel Aviv, Israel). The goal of the APHEA-2 project was to examine the effects of air pollution on daily mortality. Details on APHEA-2 data have been reported previously (Katsouyanni et al. 2001).

To maximize the power of the study, we chose the largest cities in the project, subject to the constraint that only one city be chosen in each country. The 10 cities selected were Athens, Greece; Budapest, Hungary; Lodz, Poland; London, United Kingdom; Madrid, Spain; Paris, France; Prague, Czech Republic; Rome, Italy; Stockholm, Sweden; and Tel Aviv. Together, they comprise a population of about 28 million people, which is two-thirds of the population in the full study, and represents Northern Europe, Central Europe, and the Mediterranean region.

The data that we analyzed in this study included daily counts of all-cause mortality, excluding deaths from external causes (International Classification of Disease, 9th Revision (ICD-9) code > 800), daily counts of deaths from cardiovascular disease (CVD) (ICD-9 code 390–429), and daily counts of respiratory mortality (ICD-9 code 460–519). In all cities, the mortality counts were also divided in the following age groups: 15–64, 65–74, and >75 years.

The years of study were 1990 through 1997; however, mortality data in most cities were available only through 1995 or 1996.

Air pollution data were available as daily averages of the monitoring stations in each city. Particulate matter was measured as PM10 in four cities, as PM13 in Paris and PM15 in Rome. The Paris data were assumed to be equivalent to PM10 in this study. In Rome, Athens, Lodz, and Budapest, the data were converted from other measurements into PM10 using city-specific conversion factors. Further details have been published previously (Katsouyanni et al. 2001; Zanobetti et al. 2002).

We first analyzed respiratory and heart mortality; then, we examined deaths from all causes and stratified respiratory and cardiovascular deaths by age groups.

Methods. In the first step of the analysis, generalized additive regression models (Hastie and Tibshirani 1990) were fit in each of the 10 cities, and the models were built following the APHEA-2 methodology. Details on the models for all-cause mortality have been published (Katsouyanni et al. 2001).

Separate models were built in each city because of the substantial variability in seasonal patterns and weather between, for example, Stockholm and Tel Aviv.

In the generalized additive model, the outcome is assumed to depend on a sum of nonparametric smooth functions for each variable. This allows us to better model the nonlinear dependence of daily deaths on weather and season.

The model of interest was run for each general data set, and the sample standard deviation of the resulting regression coefficients serves as the bootstrap standard error.

We examined the dependence of daily deaths on PM10 of that day and of preceding days, up to the previous 40 days, using first an unconstrained distributed lag model and then a fourth-degree polynomial distributed lag model.

If the pollution-related deaths are only being advanced by a few days to a few weeks, we would see this “harvesting” effect expressed by negative associations between air pollution and deaths several days to weeks afterward. The effect of air pollution, net of any such short-term rebound up to 40 days, is the sum of the effect estimates for all 41 days.

For Poisson regression, the unconstrained distributed lag model can be written as

\[
\log \left( E[Y_{it}] \right) = \alpha + \text{covariates} + \beta_0 t + \ldots + \beta_r t^{r-1} + \ldots + \beta_q t^{q-1} \tag{1}
\]

The unconstrained distributed lag model is too noisy to provide any information about...
the shape of the effect versus lag, but it does give an unbiased estimate of the overall effect, computed as the sum of the $\beta_j$. To be able to have an idea of the shape of the curve, it is preferable to constrain the coefficients to vary smoothly with lag number (Almon 1965). A polynomial distributed lag constrains the $\beta_j$ to follow a polynomial pattern in the lag number; that is, $\beta_j = \sum_{j=0}^{q} \eta_j j^d$, for $j = 0 \ldots q$. [2]

where $j$ is the number of lags and $d$ is the degree of the polynomial.

Too much constraint risks bias, producing a distorted shape, whereas too little constraint produces estimates that are too noisy to be informative.

To fit a fourth-degree polynomial distributed lag model with 40 days of delay, in equation 2 we would have $d = 4$ and $j = 0, \ldots, 40$.

In the first stage of the analysis, a separate distributed lag model was fit for each of the 10 cities. The coefficients $\beta_j$ by lag for the distributed lag model were obtained from equation 2, and because we used the bootstrap method, we computed the sum of the effect estimates for all 41 days in each city. In the second stage, we combined the city-specific results using inverse variance weighted averages.

In our primary analysis we used an unconstrained distributed lag, because it makes the fewest assumptions. We used the polynomial distributed lag model as a sensitivity analysis because this gives more stable results and also allows us to examine the pattern of the mortality response over time. To verify whether the longer lags really contribute to the overall pollution effect, we also analyzed the unconstrained distributed lag model with 20 and 30 days of delay.

**Results**

Table 1 shows, for the 10 cities, the mean and standard deviation of the daily number of total and for the three age groups. It also shows the mean and standard deviation of the environmental variables. The mean of daily deaths for 15- to 64-year-olds is generally very low when subdivided by cause. For respiratory mortality, the second age group is also small; therefore, we decided that the cause-specific analysis by age could be done for the two older groups for cardiovascular mortality, and in the oldest age group for respiratory mortality.

Table 2 shows the percentage increase for the combined effect estimates across all of the 10 cities of PM$_{10}$ [per 10 µg/m$^3$ and its 95% confidence interval (95% CI)] for the fourth-degree polynomial (sum of the $\beta_j$ per 10 µg/m$^3$), and the unconstrained distributed lag models when stratified by cause of death.

Figures 1 and 2 show plots of the residuals from the unconstrained distributed lag model for Athens and London, respectively, with no residual seasonality in the models.

We found a 4.2% increase (95% CI, 1.08–7.42) in respiratory deaths for a 10 µg/m$^3$ increase in PM$_{10}$ concentration using the unconstrained distributed lag model. Similar results, but with tighter confidence intervals, were found with the polynomial distributed lag models. In contrast, the mean of PM$_{10}$ on the same and previous day was associated with only

| City/age group (years) | Years of study | Total | CVD | Respiratory | PM$_{10}$ (mg/m$^3$) | Temperature | Humidity |
|------------------------|----------------|-------|-----|------------|----------------------|-------------|----------|
| Athens                 | 1992–1996      | 72.9 ± 13.2 | 35.9 ± 8.7 | 4.6 ± 2.4 | 42.7 ± 12.9 | 17.8 ± 7.4 | 61.7 ± 13.6 |
| 15–64                  | 8.3 ± 3        | 2.9 ± 1.7  | 0.4 ± 0.5 | 1.1 ± 1.1 |
| 65–74                  | 21.3 ± 5.2     | 9.3 ± 3.4  | 0.7 ± 0.8 |
| ≥ 75                   | 42.4 ± 9.6     | 23.7 ± 6.7 | 3.2 ± 1.9 |
| Budapest               | 1992–1995      | 80 ± 11.6 | 40.1 ± 8 | 3.1 ± 2 | 41 ± 9.1 | 12.8 ± 8.8 | 70.1 ± 12.6 |
| 15–64                  | 22.1 ± 4.7     | 6.8 ± 2.7  | 0.6 ± 0.8 |
| 65–74                  | 19.5 ± 4.6     | 9.5 ± 3.2  | 0.7 ± 0.8 |
| ≥ 75                   | 37.6 ± 7.9     | 23.7 ± 6.6 | 1.7 ± 1.4 |
| Lodz                   | 1990–1996      | 29 ± 6.3  | 16.8 ± 4.6 | 1.2 ± 1.1 | 53.5 ± 15.5 | 8.4 ± 8.4 | 79 ± 12.4 |
| 15–64                  | 8.9 ± 3.2      | 3.7 ± 2    | 0.3 ± 0.5 |
| 65–74                  | 8.1 ± 3        | 4.5 ± 2.2  | 0.3 ± 0.6 |
| ≥ 75                   | 12.2 ± 3.9     | 8.7 ± 3.2  | 0.5 ± 0.8 |
| London                 | 1992–1996      | 168.5 ± 25.2 | 71.4 ± 12.9 | 29 ± 11 | 28.8 ± 13.7 | 11.8 ± 5.4 | 69.3 ± 11.3 |
| 15–64                  | 28.8 ± 5.7     | 9.5 ± 3.2  | 2.4 ± 1.7 |
| 65–74                  | 37 ± 7.4       | 15.7 ± 4.5 | 4.6 ± 2.6 |
| ≥ 75                   | 101.7 ± 18.5   | 46.1 ± 9.6 | 218 ± 8.9 |
| Madrid                 | 1992–1995      | 60.8 ± 11.1 | 21.7 ± 6.1 | 6.1 ± 3.1 | 37.8 ± 17.7 | 14.5 ± 7.4 | 61.8 ± 16.7 |
| 15–64                  | 13.7 ± 3.9     | 3.1 ± 1.8  | 0.7 ± 0.9 |
| 65–74                  | 13 ± 3.8       | 4 ± 2.1    | 1.1 ± 1.1 |
| ≥ 75                   | 33 ± 7.7       | 14.6 ± 4.6 | 4.2 ± 2.5 |
| Paris                  | 1992–1996      | 123 ± 15.7 | 37.9 ± 7.5 | 9 ± 3.8 |
| 15–64                  | 30 ± 5.7       | 4.1 ± 2    | 0.9 ± 0.9 |
| 65–74                  | 20.8 ± 5       | 5.4 ± 2.5  | 1.2 ± 1.1 |
| ≥ 75                   | 69.9 ± 11.8    | 28.3 ± 6.4 | 6.9 ± 3.2 |
| Prague                 | 1992–1995      | 38.2 ± 7.2 | 22.2 ± 5.2 | 1.4 ± 1.2 | 76.2 ± 45.7 | 11 ± 8 | 69.4 ± 14.1 |
| 15–64                  | 7.5 ± 3        | 2.8 ± 1.7  | 0.3 ± 0.5 |
| 65–74                  | 10 ± 3.2       | 5.3 ± 2.3  | 0.4 ± 0.6 |
| ≥ 75                   | 20.1 ± 5.1     | 14.4 ± 4.2 | 0.7 ± 0.9 |
| Rome                   | 1992–1996      | 56.2 ± 10.4 | 23.2 ± 6.4 | 3.1 ± 2 |
| 15–64                  | 11.3 ± 3.6     | 2.7 ± 1.7  | 0.2 ± 0.5 |
| 65–74                  | 13 ± 4         | 4.5 ± 2.3  | 0.6 ± 0.8 |
| ≥ 75                   | 30.9 ± 7.5     | 16 ± 5.1   | 2.2 ± 1.7 |
| Stockholm              | 1994–1996      | 28.9 ± 6.1 | 14 ± 4.1 | 2.5 ± 1.7 | 15.5 ± 7.9 | 7.7 ± 8.1 | 71.4 ± 15.8 |
| 15–64                  | 3.2 ± 2        | 1 ± 1.1    | 0.1 ± 0.4 |
| 65–74                  | 5.3 ± 2.4      | 2.2 ± 1.6  | 0.3 ± 0.6 |
| ≥ 75                   | 19.7 ± 5.1     | 10.7 ± 3.5 | 2 ± 1.5 |
| Tel Aviv               | 1993–1996      | 27.4 ± 6.3 | 11.5 ± 4.1 | 1.4 ± 1.4 | 50.3 ± 57.5 | 20.6 ± 5.4 | 65.6 ± 11 |
| 15–64                  | 4.2 ± 2        | 1 ± 1.1    | 0.1 ± 0.4 |
| 65–74                  | 5.8 ± 2.5      | 2.3 ± 1.5  | 0.2 ± 0.5 |
| ≥ 75                   | 17.2 ± 4.9     | 8.2 ± 3.3  | 1 ± 1.2 |
a 0.74% increase (95% CI, –0.17 to 1.66) in respiratory mortality.

We found a 1.97% (95% CI, 1.38–2.55) increase in CVD deaths using the unconstrained distributed lag model. Again, the results for the polynomial distributed lag models were similar, whereas the results for the mean of lag 0 and 1 of PM10 were lower (0.69% increase, 95% CI, 0.31–1.08).

Overall, the estimated effect of PM10 was doubled for CVD when the lagged effects were considered; the effect was five times larger for respiratory mortality.

Table 3 shows the percentage increase for the combined effect estimates across all of the 10 cities for PM10 (per 10 µg/m³ and its 95% CI) derived by applying the unconstrained distributed lag model with 20, 30, and 40 days of delay. The results show a clear increasing trend with increasing numbers of lags.

Figure 3 shows the estimated effect of PM10 at each lag (and its confidence interval) for CVD deaths. PM10 has a major immediate impact, declining rapidly to near 0 with a lag of 1 week. It then shows a prolonged, delayed effect, declining rapidly to near 0 with a lag of 40 days. The results show a clear increasing trend with increasing numbers of lags.

The pattern for respiratory deaths is quite different, as shown in Figure 4. The estimated immediate effect of PM10 decreases much more slowly than it does for CVD deaths. It again rises to a second smaller peak about a month later, before decreasing again by lag 40.

We then repeated the same analyses stratifying by age groups. Table 4 shows the results for all the outcomes by age group. Within each age group, we found similar results using the constrained and unconstrained distributed lag models. When looking across the age groups, we found that the risks increase. For example, using the fourth-degree polynomial distributed lag model, we found a –0.25% (95% CI, –0.876 to 0.36) increase in total mortality in the 15- to 64-year-olds, a 0.78% (95% CI, 0.23–1.33) increase in the 65- to 74-year-olds, and a 1.84% (95% CI, 0.92–2.78) increase in the ≥75-year-old group.

Applying the fourth-degree polynomial distributed lag model, we found a 2.06% increase (95% CI, 1.05–3.09) in CVD mortality for the 64- to 75-year-olds for a 10 µg/m³ increase in PM10. For the ≥75 year group, we obtained a 2.35% (95% CI, 1.42–3.29) increase in CVD deaths. We found a 4.57% (95% CI, 1.25–7.99) increase in respiratory disease for the ≥75 year group, which is similar to the results for all ages.

Table 2. Results for the combined estimated PM10 effect for the fourth degree and unrestricted models, with 40 days of delay using bootstrap methods.

|                | CVD                  | Respiratory disease |
|----------------|----------------------|---------------------|
|                | Percent              | 95% CI              | Percent              | 95% CI              |
| Mean lag 01    | 0.69                 | 0.31–1.09           | 0.74                 | 0.17–1.86           |
| Distributed lag|                      |                     |                      |
| Fourth degree  | 1.99                 | 1.44–2.54           | 4.21                 | 1.70–6.79           |
| Unrestricted   | 1.97                 | 1.38–2.55           | 4.20                 | 1.08–7.42           |

Increases are for a 10 µg/m³ increase in PM10.

Figure 3. The estimated shape of the association of PM10 for each lag with daily deaths for CVD with a fourth-degree distributed lag model with random effect in 10 cities (percentage increase in deaths for a 10 µg/m³ increase in PM10). The shaded area represents the 95% CIs.

Figure 4. The estimated shape of the association of PM10 for each lag with daily deaths for respiratory disease with a fourth-degree distributed lag model with random effect in 10 cities (percentage increase in deaths for a 10 µg/m³ increase in PM10). The shaded area represents the 95% CIs.
would expect a distribution of times until the exacerbation required hospitalization, which might account for this pattern. We also found an increasing PM$_{10}$ effect with increasing age.

Up to now, no study has examined long distributed lags or short-term mortality displacement for specific ages and causes of death. This study confirms the basic finding of the previous analysis on mortality displacement (Schwartz 2000c, 2001; Zanobetti et al. 2002; Zeger et al. 1999), because we did not find any near short-term harvesting. Rather, our results are consistent with the report of higher risk estimates in cohort studies (Dockery et al. 1993; Pope et al. 1995b), although still smaller in magnitude. However, there may be short-term displacement for some, but this is outweighed by the longer-term effect.

This study provides insight into the shape of the longer-term responses to particulate air pollution. In particular, it suggests that the adverse response to pollution persists to a month or longer after exposure not only for total mortality but also for respiratory and cardiovascular mortality. This was also observed in the 1952 London smog episode (Anderson 1999).

If we focus on assessing the difference in the lag pattern between particulate matter and cause-specific mortality, we can identify two different patterns. For respiratory deaths, the decline in impact of particle exposure over time is noticeably slower than that for cardiovascular deaths. For both causes, there is a small elevation of death risk that persists for about one month. The confidence intervals prevent us from drawing strong conclusions about the shape of the curves.

Based on the biologic plausibility, the differences on the lag structure here observed are admissible. CVD presents an acute response to trigger agents, and both experimental (Godleski 2000) and human (Peters et al. 2000) studies have supported this point. On the other hand, a wider and more lagged structure can be expected for respiratory diseases such as pneumonia and chronic disease, because these events have slower progression.

The APHEA-2 project was one of three large multicility studies of the effects of particulate air pollution that have recently been reported. The largest study (National Mortality and Morbidity Air Pollution Study) examined 90 U.S. cities (Samet et al. 2000). Because of the limitations of U.S. monitoring networks, results were reported only for 24-hr PM exposure. Those results were quite similar to the main APHEA-2 results (Katsouyani et al. 2001) but slightly lower. The APHEA-2 main results used the mean of PM exposure over 2 days (Katsouyani et al. 2001).

Another U.S. study restricted to 10 U.S. cities with daily PM monitoring showed a gradient in effect size when comparing 1-day averages, 2-day averages, and a 5-day distributed lag (Schwartz 2000a).

Our results, taken together with the reports described above, as well as other approaches to the harvesting question, confirm that estimates of the effects of subchronic exposure to air pollution that rely on 24- or 48-hr exposure measures seriously underestimate the impact of particle exposure.

Moreover, because part of the effect appears to involve triggering myocardial infarctions (Peters et al. 2001), the previous literature showing persons surviving for a month after their infarct have a life expectancy measured in years, suggests that deaths associated with air pollution include many that are advanced by a considerable amount.

Together with previous studies, this study suggests that risk assessment based on the short-term associations likely underestimate the number of early deaths that are advanced by a significant amount, and that estimates based on the cohort studies, or studies such as this one, would more accurately assess the public health impact.

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