In this paper, we present national maps of relative rates of mortality associated with short-term exposure to particulate matter < 10 µm in aerodynamic diameter (PM<sub>10</sub>). We report results for 88 of the largest metropolitan areas in the United States from 1987 to 1994 for all-cause mortality, combined cardiovascular and respiratory deaths, and other causes of mortality. Maximum likelihood estimates of the relative rate of mortality associated with PM<sub>10</sub> and the degree of statistical uncertainty were obtained for each of the 88 cities by fitting a separate log-linear regression of the daily mortality rate on air pollution level and potential confounders. We obtained Bayesian estimates of the relative rates by fitting a hierarchical model that takes into account spatial correlation among the true city-specific relative rates. We found that daily variations of PM<sub>10</sub> are positively associated with daily variations of mortality. In particular, the relative rate estimates of cardiovascular and respiratory mortality associated with PM<sub>10</sub> are larger on average than the relative rate estimates of all-cause and other-cause mortality. The estimated increase in the relative rate of death from cardiovascular and respiratory mortality, all-cause mortality, and other-cause mortality were 0.31% (95% posterior interval, 0.15–0.5), 0.22% (95% posterior interval, 0.1–0.38), and 0.13% (95% posterior interval, −0.05 to 0.29), respectively. Bayesian estimates of the city-specific relative rates ranged from 0.23% to 0.35% for cardiovascular and respiratory mortality, from 0.18% to 0.27% for all causes, and from 0.10% to 0.20% for other causes of mortality. The spatial characterization of effects across cities offers the potential to identify factors that could influence the effect of PM<sub>10</sub> on health, including particle characteristics, offering insights into mechanisms by which PM<sub>10</sub> causes adverse health effects. Key words: air pollution, Bayesian methods, hierarchical models, particulate matter, relative rate, spatial smoothing. Environ Health Perspect 111:39–43 (2003). [Online 13 November 2002] doi:10.1289/ehp.5181 available via http://dx.doi.org/
the health effects of air pollution within regions.

We applied a Bayesian hierarchical model that allows for the possible spatial correlation between city-specific estimates to the 88 largest metropolitan areas in the United States from 1987 to 1994, and we report national maps of MLEs and Bayesian estimates of the percentage increase in mortality associated with 10 μg/m³ increase in PM₁₀. Analyses were conducted for all-cause mortality, cardiovascular and respiratory deaths, and other causes of mortality.

Materials and Methods

We used the NMMAPS database of the largest 90 cities (3,4). Because one of the goals of this study was to graphically represent spatial correlation, we excluded Honolulu, Hawaii, and Anchorage, Alaska, from the analysis.

Figure 1 is a map showing the locations of the 88 cities and the 7 geographical regions used in this analysis. The database includes mortality, 24-hr average temperature and dew-point temperature, and 24-hr average PM₁₀ concentration for the 88 largest metropolitan areas in the United States for the 7-year period 1987–1994. The air pollution data were obtained from the AIRS database (5) maintained by the U.S. EPA. In some locations, a high percentage of days had missing values for PM₁₀ because measurements have been required only every 6 days since 1987 by the agency. These cities were retained, and the resulting increased uncertainty was taken into account in our analysis. We obtained daily cause-specific mortality data, aggregated at the level of the county, from the National Center for Health Statistics (Hyattsville, MD). After excluding deaths from external causes and in nonresidents of the counties, we classified the deaths by age group (< 65, 65–74, and ≥ 75 years) and by cause according to the International Classification of Diseases, Ninth Revision: cardiac (codes 390–448); respiratory, including chronic obstructive pulmonary disease and related disorders (codes 490–496), influenza (code 487), and pneumonia (codes 480–486 and 507); and other remaining diseases. The hourly temperature and dew point data for each site were obtained from the Earth Info CD-ROM database (10). The database is described in detail elsewhere (3,4).

We analyzed the data with a two-stage Bayesian hierarchical model (11). At the first stage, we obtained the MLE of the relative rate of mortality associated with a 10 unit change in PM₁₀, ̂β, and the corresponding statistical variance ̂σ² within each city, by fitting a log-linear generalized linear model with parametric adjustments for confounding factors. The outcome variable was the total number of deaths on a particular day, the exposure variable was the previous day’s PM₁₀ level, and the controlled potential confounders were longer term trends, seasonality and weather. No other pollutants were included in the model. The city-specific model specification is similar to the ones used by Kelsall et al. (8) and Samet et al. (3), but instead of using a generalized additive model with smoothing splines (12), we used a generalized linear model with natural cubic splines.

At the second stage, we assumed that the true but unknown city-specific relative rates, β城市发展future, have a common mean, α, and variance, ̂σ². We express the degree of similarity of the relative rates in locations  and  as a function of the distance between the cities. We define the distance between cities as the Euclidean distance of the longitude and latitude coordinates of the cities centroids. More specifically, we assume that cor(β城市发展future,β城市发展future) = \exp((-φ × d(,)²)/2). The parameter φ represents the rate of decay to zero of the correlation as the distance between the two cities increases.

The Bayesian estimate of β城市发展future, defined as the posterior mean ̂E[β城市发展future|α, ̂σ², φ, data], is a weighted average of the MLE, ̂β, and of the overall relative rate, ̂α:

\[
E[β城市发展future|α, ̂σ², φ, data] = \Lambda × 1̂α + (I − \Lambda) × ̂β城市发展future
\]

where

\[
Λ = \left[\frac{V^{-1} + \frac{R(φ)^{-1}}{σ²}}{σ²} + \frac{R(φ)^{-1}}{σ²} + 1\right] × \frac{R(φ)^{-1}}{σ²} \times 1̂α.
\]

Here ̂β城市发展future is the vector of the city-specific estimates; V is a diagonal matrix with  ²; R(φ) is the spatial correlation matrix with off-diagonal elements equal to cor(β城市发展future,β城市发展future); I is a vector of ones, and  is the identity matrix. Equation 1 points out that the Bayesian estimate of the city-specific air pollution effects is shrunk toward the overall mean (α), and the shrinkage factor (Λ) is proportional to the statistical uncertainty of the MLEs (V) and to the spatial correlation matrix [R(φ)], but inversely proportional to the degree of heterogeneity of the city-specific relative rates (σ²).

Model fitting was performed using a Bayesian statistical approach (13) and Bayesian software (14), which provides an estimate of the posterior distribution of the parameters of interest (α, ̂σ², ̂β, ̂φ). The posterior distribution was 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. A Bayesian estimate is defined as the mean of the posterior distribution. We carried out this analysis without making prior assumptions as to the value of the relative rate. More specifically, prior distributions for ̂β and ̂α were normal with large variances. Prior distribution for ̂σ² was gamma with scale and shape parameters equal to 0.001 and 0.001. Finally, the prior distribution for ̂φ was uniform in the interval (φmin, φmax). The parameters φmin and φmax were selected so that if ̂φ = φmin the prior correlation at the maximum and at the minimum distance was 0.01–0.82, and if ̂φ = φmax the prior correlation at the maximum and at the minimum distance was 0–0.52.

We used the posterior distribution to determine the probability that the relative rate of mortality associated with PM₁₀ is in a particular interval; it can also be used to determine the 95% posterior intervals. The 95% posterior interval encompasses 95% of the posterior distribution, a Bayesian formulation analogous to the 95% confidence interval. To approximate the posterior distributions of all the parameters of interest, we implemented simulation-based methods, and in particular the Geobugs software (14). Statistical models for analyzing correlated geographic cohort data based on Cox proportional hazards survival model with spatial correlated random effects have been proposed by Burnett et al. (15).

Results

Figure 2 shows the posterior distributions of the overall effects (α) and of the spatial correlation, cor(β城市发展future,β城市发展future), for total mortality, cardiovascular–respiratory mortality, and other-cause mortality. We found that the estimated overall relative rate of cardiovascular–respiratory mortality associated with PM₁₀ (percent increase in mortality per 10-μg/m³ increase in PM₁₀) was the highest (0.31%; 95% posterior interval, 0.15–0.5) compared to estimated overall relative rates of death for total mortality and other causes of mortality at 0.22% (95% posterior interval, 0.1–0.38), and 0.13% (95% posterior interval: –0.05 to 0.29), respectively.

Between-city standard deviation indicates the degree of heterogeneity of the relative rates for mortality across cities with respect to the overall relative rate. For example, if the overall relative rate, ̂α, equals 0.22 and the between-city standard deviation, ̂σ, equals...
Examples of city-pairs at the four distances are indicated in yellow (0.8), red (0.48), purple (1), and blue (2) in Figure 1.

| Distance | City Pairs                                      |
|----------|-------------------------------------------------|
| 0.08     | Kansas, KS, and Topeka, KS                      |
| 0.48     | San Jose, CA, and Oakland, CA                   |
| 1.00     | Kansas, KS, and Topeka, KS                      |
| 2.00     | Detroit, MI, and Akron, OH                       |

Posterior means and posterior quantiles of the spatial correlation $\phi \times$ of cardiovascular–respiratory mortality (CV/resp), and other causes of mortality (Other), based on the percent increase in mortality per 10 µg/m³ increase in PM$_{10}$. The curves indicate the posterior means.

The city-specific relative rates are within the inter-

Discussion

Particulate air pollution is a national public health problem, regulated under the provisions of the Federal Clean Air Act. Using national data, we attempted to characterize the effect of particulate air pollution on mortality for the largest cities in the United States. We used Bayesian methods to map the relative mortality rates associated with PM$_{10}$, grouping the nation into seven regions, following the regions designated by the U.S. EPA.

We found that there was some modest variation in the relative risks across the nation (Figures 3 and 4). In previously reported analyses, we were unable to explain the heterogeneity using descriptors of the population, air pollution characteristics, and reliability of the PM$_{10}$ measurement data (8).

Beyond random variation alone, the heterogeneity has several potential and nonexclusive explanations: across-region variation in the characteristics and sizes of the populations susceptible to air pollution and variation in the toxicity of PM$_{10}$. With regard to susceptibility, persons with underlying heart and lung disease, particularly the elderly, have been postulated to be at increased risk from exposure to PM$_{10}$ or other air pollutants (6). Both children and adults with asthma may also be at increased risk. Variation in the frequency of chronic heart and lung disease across the country is well documented. Mortality rates from chronic obstructive pulmonary disease and coronary heart disease vary widely, being highest in the Southeast and lowest across the mountain West (7). The range of age-adjusted mortality rates is approximately 2-fold, indicating an approximately similar range in prevalence. Asthma rates also vary, tending to be higher in inner cities with high proportions of minority children (18,19). Correspondence has not been found between indicators of the relative sizes of susceptible populations across the country and maps of comparative pollution effects.

Sources of airborne particulate matter vary across the country, as does the chemical composition and size distribution of particulate matter (20,21). Nationally, primary particulate emissions come from fugitive dust, biomass burning, agriculture, wind erosion, fossil-fuel combustion, and other sources; secondary particles are formed from the precursor gases.
sulfur dioxide and nitrogen dioxide and volatile organic compounds.

Some general conclusions can be made about regional differences in particle composition (20,21). In the eastern United States, secondary particles appear to dominate particulate matter ≤ 2.5 μm in aerodynamic diameter (PM2.5), whereas crystal dusts are prominent in agricultural areas and in desert regions. Comparative data for the eastern and western United States show that PM2.5 particles have a greater proportion of sulfate and less organic carbon in the eastern portion of the country. We cannot yet, however, link specific particle characteristics to toxicity (22); this topic is a focus of intense research as recommended by the National Research Council’s Committee on Research Priorities for Airborne Particulate Matter (23). Concentrations of PM10 and PM2.5 vary across the country, as does their ratio. If, in fact, the smaller particles are the component of airborne particulate matter causing increased mortality, we would anticipate the greatest effects in those regions having the highest concentrations of PM2.5, regardless of PM10 concentration. The 1999 data from the U.S. EPA, although still incomplete, indicate the highest levels in California and across the Midwest and Southeast. This pattern is only partially concordant with the mortality maps in Southern California and in the Midwest, but not concordant with the mortality pattern found in the Northeast.

We have also found that the effect of PM10 on mortality is negatively modified by the PM10 level itself; that is, the effect of PM10 per unit concentration declines at increasing concentrations (4,7). The maps of risks associated with PM10 (Figures 3 and 4) are not consistent with this pattern of modification. With further characterization of particles across the country from new monitoring initiatives, a richer database will be available to explore variation in health risk in relation to the heterogeneity of particle characteristics.

Our mapping strategy represents a starting point for refinement. In our modeling strategy, we assumed a similarity of the relative rates within regions based on the Euclidean distance between the cities. This simplistic assumption was necessitated by a lack of additional, external information on factors that might drive heterogeneity of risk estimates. The modeling approach might be refined by incorporating relevant geographic and meteorologic characteristics as well. One enhancement would be to incorporate priors based on results of receptor models, which would integrate sources and meteorology to provide more credible priors. The finding of heterogeneity has potential implications with regard to research opportunities and public health protection. The heterogeneity in risk estimates offers an opportunity to perform hypothesis-driven research, assessing the consistency of hypotheses concerning toxicity of particles against the observed differences in risk. At present, the National Ambient Air Quality Standards (24) are set on mass alone. A more complete understanding of the causes of heterogeneity of risk might lead to more focused source control or even to standards directed at specific types of particles.

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