



Original Contribution

Particulate Air Pollution and Mortality in the United States: Did the Risks Change from 1987 to 2000?

Francesca Dominici¹, Roger D. Peng¹, Scott L. Zeger¹, Ronald H. White², and Jonathan M. Samet²

¹ Department of Biostatistics, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD.

² Department of Epidemiology, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD.

Received for publication September 7, 2006; accepted for publication January 19, 2007.

Evaluation of the public health impact of air quality regulations, referred to as accountability research, is increasingly viewed as a necessary component of responsible governmental policy interventions. The authors present an example of accountability assessment based on evaluating change in the short-term effect of airborne particles over a period of increasingly stringent regulation that might have changed the chemical composition and toxicity of these particles. They used updated data and methods of the National Morbidity Mortality Air Pollution Study to estimate national average relative rates of the effects of particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter on all-cause, cardiovascular, and respiratory mortality and on other-cause mortality for 1987–2000. They estimated national average relative rates of the effects of particulate matter $\leq 2.5 \mu\text{m}$ in aerodynamic diameter on all-cause mortality for 1999–2000. The authors found strong evidence that lag 1 exposures to particulate matter $\leq 10 \mu\text{m}$ and $\leq 2.5 \mu\text{m}$ in aerodynamic diameter continue to be associated with increased mortality. They also found a weak indication that the lag 1 effects of particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter on mortality declined during 1987–2000 and that this decline occurred mostly in the eastern United States. The methodology presented can be used to track the health effects of air pollution routinely on regional and national scales.

mortality; particulate matter; population surveillance; public policy; sentinel surveillance

Abbreviations: NMMAPS, National Morbidity Mortality Air Pollution Study; $\text{PM}_{2.5}$, particulate matter $\leq 2.5 \mu\text{m}$ in aerodynamic diameter; PM_{10} , particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter.

Editor's note: An invited commentary on this article appears on page 889, and the authors' response is on page 892.

Implementation of increasingly stringent national and local air quality standards in the United States over the past three decades has resulted in significant improvements in ambient air quality. Not surprisingly, regulators, regulated industries, and the public are looking for evidence of gains in public health that may have resulted from implementing

these costly regulatory policies. As a result, evaluation of the public health impact of air quality regulations, now also referred to as accountability research (1), is viewed as an emerging component of responsible governmental policy intervention and environmental health tracking and research. A 2003 report of the Health Effects Institute addresses the conceptual framework for research on accountability and related methodological challenges. Several studies have now been carried out that fit within this research framework, including, for example, a study of attainment status with the particulate matter National Ambient Air Quality Standard and infant and adult mortality (2, 3)

Correspondence to Dr. Francesca Dominici, Department of Biostatistics, Bloomberg School of Public Health, Johns Hopkins University, 615 North Wolfe Street, Baltimore, MD 21205 (e-mail: fdominic@jhsph.edu).

and studies of interventions that sharply changed pollution sources, such as banning coal sales in Dublin, Ireland, and reducing sulfur in fuels in Hong Kong, China (4–6).

The 1970 Clean Air Act Amendments provided a regulatory framework and prompted national air pollution control programs targeted at stationary and mobile sources of air pollution in the United States. With the passage of the Clean Air Act Amendments of 1990, a number of major, new air quality regulatory programs were adopted that further reduced air pollution emissions. These programs included the Acid Rain Control Program, California LEV1 (Low Emission Vehicle phase 1), and national tier 1 motor vehicle emission standards (more stringent national emission standards for trucks and buses), as well as state control program requirements that resulted from designation of areas as not attaining the National Ambient Air Quality Standards for particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter (PM_{10}) and ozone. As a result, levels of particulate matter have fallen progressively and chemical composition of particulate matter is changing, providing an opportunity to assess any parallel public health gains.

The National Morbidity Mortality Air Pollution Study (NMMAPS) is an ongoing study of air pollution and health aimed at periodically estimating short-term effects of ambient air pollution on mortality and morbidity on national and regional scales (7–10). The original NMMAPS database includes daily time series of air pollution, temperature, dew point temperature, and counts of all-cause and cause-specific mortality for the largest 100 US counties for the period 1987–1994. The NMMAPS database has been updated through the year 2000 and now includes particulate matter $\leq 2.5 \mu\text{m}$ in aerodynamic diameter ($\text{PM}_{2.5}$) data from 1999 onward. Because time-series data were available when several key particulate matter-related air pollution control programs were implemented, we used the NMMAPS data to assess whether there was a temporal trend in the short-term effect of particulate matter on mortality. Because we adjusted for longer-term trend in particulate matter concentration, we could not assess overall benefits to health of declining particulate matter concentration. Rather, we used all available data to provide evidence as to whether short-term effects of particulate matter on mortality might have changed, from 1987 through 2000, because of changes in the characteristics of particulate matter.

MATERIALS AND METHODS

The NMMAPS data, including a full description, and the boundaries of geographic regions are described in detail elsewhere (7–9, 10). Updated data through the year 2000 are available at <http://www.ihapss.jhsph.edu>.

To estimate national short-term effects of PM_{10} and $\text{PM}_{2.5}$ on mortality, we used statistical tools developed for NMMAPS (10, 11). Specifically, we applied Bayesian two-stage hierarchical models (12, 13) to estimate county-specific, regional, and national average associations between day-to-day variation in PM_{10} at lag 1 and county-level mortality counts, accounting for weather, seasonality, and long-term trends.

At the first stage, single-lag overdispersed Poisson regression models (14, 15) were used for estimating county-specific relative rates of mortality associated with ambient levels of PM_{10} . These county-specific models include as explanatory variables 1) indicator variables for the day of the week to allow for different baseline mortality rates for each day; 2) smooth functions of calendar time (natural cubic splines) with 7 degrees of freedom per year to adjust for seasonality, for example, due to influenza epidemics, and longer-term trends, for example, due to changes in medical practice patterns; and 3) smooth functions of same-day temperature (6 degrees of freedom) and dew point temperature (3 degrees of freedom) and of the 3 previous days' temperatures and dew point temperatures to control for the potential confounding effect of weather. Statistical properties of this modeling approach and alternative modeling specifications for confounding adjustment are reported elsewhere in detail (16, 17).

At the second stage, to produce a national average estimate of the short-term association between PM_{10} and mortality, we used Bayesian hierarchical models (12, 13, 18, 19) to combine relative rates across counties accounting for within-county statistical error and for between-county variability of the “true” relative rates (also called heterogeneity). Estimates of risks associated with PM_{10} and $\text{PM}_{2.5}$ exposures were calculated by using data for the periods 1987–2000 and 1999–2000 and by pooling city-specific estimates across 100 and 96 communities, respectively.

We estimated the average relative rate of mortality associated with lag 1 exposure to PM_{10} separately for three periods—1987–1994, 1995–2000, and 1987–2000—for the “eastern United States,” the “western United States,” and all the 100 US counties. We selected the end of the year 1994 for time stratification so that, for the two study periods 1987–1994 and 1995–2000, a similar number of counties with PM_{10} monitors would be collecting data regularly. The “eastern United States” includes 62 counties located in the industrial Midwest, Northeast, and Southeast regions; the “western United States” includes 38 counties located in the Northwest, Southwest, Upper Midwest, and Southern California regions, as defined in previous NMMAPS analyses (20). To produce regional estimates, we used the same two-stage hierarchical model described above but separately within each of the two geographic regions.

To estimate linear trends of short-term effects of PM_{10} on mortality, we adapted statistical tools developed by Peng et al. (21) for estimating time-varying relative rates (22). Specifically, at the first stage within each urban community, we estimated the percentage increases in all-cause and cause-specific mortality associated with a $10\text{-}\mu\text{g}/\text{m}^3$ increase in daily PM_{10} on the previous day as a linear function of calendar time for the period 1987–2000: $\beta^c(t) = \alpha_0^c + \alpha_1^c t$. At the second stage, we used Bayesian hierarchical models to pool the regression coefficients (α_0^c, α_1^c) across communities. We denoted by α_1 the national average slope, which can be interpreted as the change in the percentage increase in mortality for a $10\text{-}\mu\text{g}/\text{m}^3$ increase in daily PM_{10} at lag 1 associated with a change in time of 1 year. To assess the number of years and daily deaths necessary to detect interaction between the short-term effects of PM_{10} on mortality

and calendar time, we conducted sample size calculations. Details are provided in the Appendix.

Finally, to investigate the sensitivity of national average estimates for the period 1987–2000 to the degree of control for temporal confounding, we estimated the national average effects for PM_{10} and for $PM_{2.5}$ by varying the number of degrees of freedom from 1 per year to 20 per year (10, 17).

To facilitate replication, the R software used for carrying out these analyses is available at <http://www.biostat.jhsph.edu/~rpeng/RR/trend> (23).

RESULTS

Table 1 summarizes the key particulate matter–related control programs and years of implementation for the period 1987–2002. Implementation dates were estimated for those control programs (e.g., PM_{10} state implementation plans) that did not have statutorily mandated implementation deadlines.

Figures 1 and 2 show box plots of annual county-specific averages of PM_{10} and $PM_{2.5}$, respectively, for the years 1987–2003. In both figures, the numbers of US counties for which particulate data were available are denoted at the bottom of the box plots. Both PM_{10} and $PM_{2.5}$ annual averages declined. After 1997, PM_{10} was monitored less often, whereas the national network for $PM_{2.5}$ was first put into place in 1999.

Table 2 summarizes national- and regional-average (eastern United States and western United States) effects of lag 1 PM_{10} on all-cause, cardiorespiratory, and other causes of mortality for 1987–1994, 1995–2000, and 1987–2000. For the whole period 1987–2000, evidence shows that lag 1 exposure to PM_{10} continued to be associated with mortality. In addition, for all-cause and cardiorespiratory mortality, the national average estimates of the short-term effects were larger in the eastern United States. By comparing relative rate estimates for 1987–1994 versus 1995–2000, we found weak evidence that short-term effects declined and that most of the decline in the national estimate was attributable to the counties located in the eastern United States. The decline was primarily for all-cause and cardiorespiratory outcomes. However, the relative rate estimates for 1987–1994 were not significantly different from the relative rate estimates for 1995–2000.

We also estimated the linear trend over time in the national average effect of lag 1 PM_{10} on all-cause and cardiorespiratory mortality. The estimated slopes for all-cause mortality and for cardiovascular and respiratory mortality were $\hat{\alpha}_1 = -0.012$ (95 percent posterior interval: $-0.037, 0.014$) and $\hat{\alpha}_1 = -0.016$ (95 percent posterior interval: $-0.058, 0.027$), respectively, for a 1-year change in calendar time. Although these estimated trends are not statistically significant, they indicate that, on average, across the 100 counties, associations between day-to-day variation in ambient PM_{10} levels and all-cause and cardiorespiratory mortality counts became weaker over the period 1987–2000.

Figure 3 shows national average effects of PM_{10} and $PM_{2.5}$ on all-cause mortality plotted against the number of degrees of freedom in the smooth function of time used to

control for temporal confounding. The vertical segment denotes the national average relative rate estimates obtained under the NMMAPS basic model, which uses 7 degrees of freedom per year in the smooth function of time. Under the NMMAPS basic model, a $10\text{-}\mu\text{g}/\text{m}^3$ increase in PM_{10} at lag 1 is associated with 0.19 (95 percent posterior interval: 0.10, 0.28) and 0.24 (95 percent posterior interval: 0.13, 0.36) percent increases in all-cause mortality and cardiorespiratory mortality, respectively. In addition, a $10\text{-}\mu\text{g}/\text{m}^3$ increase in $PM_{2.5}$ at lag 1 is associated with 0.29 (95 percent posterior interval: 0.01, 0.57) and 0.38 (95 percent posterior interval: $-0.07, 0.82$) percent increases in all-cause mortality and cardiorespiratory mortality, respectively. National average effects for $PM_{2.5}$ are similar to estimates based on PM_{10} but with larger statistical uncertainty because of the availability of only 2 years of $PM_{2.5}$ data.

DISCUSSION

Environmental regulations intended to protect human health are based on a foundation of scientific evidence that comes from toxicologic, clinical, and epidemiologic research. Their purpose is to reduce exposure to agents that have been found to adversely affect health, and their implementation is presumed to lead to a reduction in the burden of health effects caused by the exposure. In a “chain of accountability” for an environmental regulation, its consequences could be traced from the reduction of emissions of a pollutant, and changes in its chemical composition in the environment, to altered population exposures and doses, and finally to reduced adverse health effects (1).

In this paper, we provide evidence as to whether the short-term effects of PM_{10} on mortality changed during 1987–2000, when several air quality regulations were implemented. Specifically, we assessed whether the risk of exposure to airborne particles changed over a period of substantial alterations in the sources and of declines in ambient concentrations of airborne particles. For this purpose, we used nearly the full set of available data for PM_{10} and mortality; we found evidence of a persistent effect of airborne particles on mortality with PM_{10} as well as $PM_{2.5}$, the more recent indicator.

We found weak evidence of a trend of decline in the short-term effect of PM_{10} on mortality from 1987 through 2000, as well as some indication of geographic heterogeneity in the trend. We also found that day-to-day variations in all-cause and cardiopulmonary mortality are associated with ambient concentrations of $PM_{2.5}$ and that these effects are larger than the ones for PM_{10} .

Our analytic approach provides one quantitative basis to assess whether the association between day-to-day changes in pollution levels and health outcomes is weakening over time. Short-term risks estimated from time-series studies are not confounded by factors that vary slowly over time, such as changes in the economy and in industrial activities. A declining trend in the short-term risk estimates is evidence that the day-to-day association between particulate matter and mortality is getting weaker over time, possibly as a result of changes in the composition and toxicity of the particulate

TABLE 1. Key PM₁₀*-related air pollution control programs, United States, 1987–2000

Program regulation	Program description	Year implemented	Reference
National Diesel-powered Truck and Bus Emission Standards	Reduced allowable emissions of diesel particulate matter from trucks by almost 60% (1991) and by an additional 25% (1994) from 1990 levels. Nitrogen oxide emission limits reduced by one third from 1990 levels. Urban bus particulate matter emission limits reduced by more than 80% (1993), with additional reductions of 5% (1994) and 3% (1996).	Trucks: 1991, 1994; buses: 1993, 1994, 1996	US EPA.* Emission Standards Reference Guide for Heavy-Duty and Nonroad Engines (EPA420-F-97-014). September 1997.
Moderate Area PM ₁₀ NAAQS* SIP*	Counties designated as moderate PM ₁₀ nonattainment areas under the Clean Air Act of 1990 required to submit state implementation plans by November 15, 1991, demonstrating attainment of the PM ₁₀ NAAQS by December 1994.	1992†	Clean Air Act. Section 189 (a).
Serious Area PM ₁₀ NAAQS SIP	Los Angeles Basin, Coachella Valley, Owens Valley, and San Joaquin Valley, California; Las Vegas, Nevada (1994); Phoenix, Arizona (1997)	1994–2000†	Clean Air Act. Section 189 (b).
Acid Rain Control Program Phase I	Set sulfur dioxide emissions levels for 263 fossil-fuel power plant units located in the eastern United States plus over 150 phase-II-affected units that opted in. In 1995, an annual reconciliation showed a 50% reduction from 1980 levels (39% beyond the phase I program requirement).	1995	Clean Air Act. Title IV. Section 404 (a). US EPA. 1995 Acid Rain Program Compliance report. http://www.epa.gov/airmarkets/cmprpt/index.html
California LEV1* Motor Vehicle Emissions Standards	Reduced allowable hydrocarbon and nitrogen oxide emission limits for model year 1995 motor vehicles in California.	1995	US EPA. Federal and California Exhaust and Evaporative Emission Standards for Light-Duty Vehicles and Light-Duty Trucks (EPA410-B-00-001). February 2000.
OTC* NOx* Budget Program	Required installation of NOx RACT* (phase 1), with additional NOx reductions by 1999 (phase 2) and 2003 (phase 3) in 12-state OTC region. Replaced in 2002 by the NOx SIP Call program.	1995, 1999	http://www.epa.gov/airmarkets/otc/index.html
National Tier 1 Motor Vehicle Emissions Standards	Reduced allowable emission limits for hydrocarbons and nitrogen oxides from model year 1996 cars and light-duty trucks except in California.	1996‡	US EPA. Federal and California Exhaust and Evaporative Emission Standards for Light-Duty Vehicles and Light-Duty Trucks (EPA410-B-00-001). February 2000.
National Low Emission Vehicle Program	Reduced hydrocarbon and nitrogen oxide emission limits from model year 1999 (Northeast states) and model year 2001 (national) motor vehicles by 70% from tier 1 limits.	1998, 2000	Federal Register/40 CFR*Parts 9, 85 & 86/Vol. 63, No. 4/January 7, 1998. US EPA. Regulatory Announcement: Final Rule for the National Low Emission Vehicle Program (EPA420-F-97-047). December 1997.
Acid Rain Control Program Phase II	Expanded the number of power plants included in the program and further tightened sulfur dioxide emissions and NOx emissions levels. Phase II limits designed to reach the goals of a 10 million ton reduction in sulfur dioxide (50% reduction) and a 2 million ton reduction in NOx from 1980 levels by 2010. A permanent cap of 8.95 million tons for sulfur dioxide emissions from power plants affected by phase I and phase II to take effect in 2010.	2000	Clean Air Act. Title IV. Section 404(a). National Acid Precipitation Assessment Program. 2005 Report to Congress.

* PM₁₀, particulate matter ≤10 μm in aerodynamic diameter; EPA, Environmental Protection Agency; NAAQS, National Ambient Air Quality Standard; SIP, State Implementation Plan; LEV1, Low Emission Vehicle phase 1; OTC, Ozone Transport Commission; NOx, oxides of nitrogen; RACT, Reasonably Available Control Technology; CFR, Code of Federal Regulations.

† Approximate implementation date(s).

‡ Model year of full implementation.

matter from the air quality control programs or of nonlinearity between daily particulate matter concentration and the associated risk. However, in our previous work, we found

that this exposure-response curve is linear for PM₁₀ and mortality (10, 24). In addition to a change in the toxicity of ambient particulate matter, other hypotheses for a time

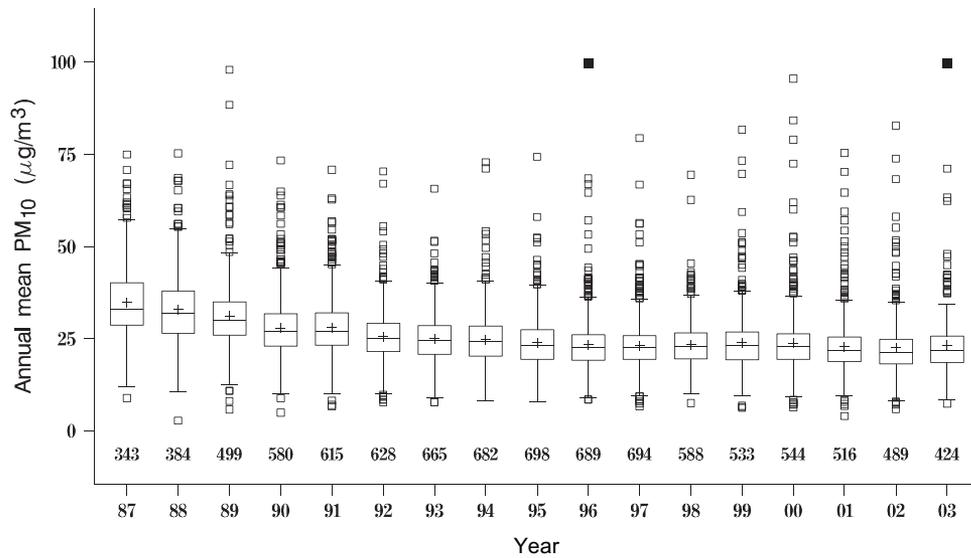


FIGURE 1. Box plots of county-specific averages of particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter (PM_{10}) for each year for the period 1987–2003 in the United States. The numbers of US counties for which PM_{10} data were available are denoted at the bottom of the box plots. The box denotes the low quartile, the median, and the upper quartile. The vertical extensions denote the upper 5th and 97.5th percentiles. The open squares denote the outliers. The two filled squares denote two clipped outliers.

trend of declining effect can be offered: 1) greater exposure measurement error at lower levels of PM_{10} ; or 2) flattening of the exposure-response relation at lower concentrations of particulate matter; or 3) a change in the underlying susceptibility of the population, reflecting, for example, a decline in smoking or dropping coronary heart disease rates.

Because our analyses estimated the short-term effects of particulate matter on mortality with simultaneous adjust-

ment for long-term trends in the levels of particulate matter concentration, they cannot directly evaluate the benefit of air quality standards that seek to reduce the mass concentration of airborne particles. Evidence relating to this question can be gathered by testing whether the county-specific long-term particulate matter average is an effect modifier of the county-specific short-term risks of particulate matter on mortality. We did not find any evidence of effect

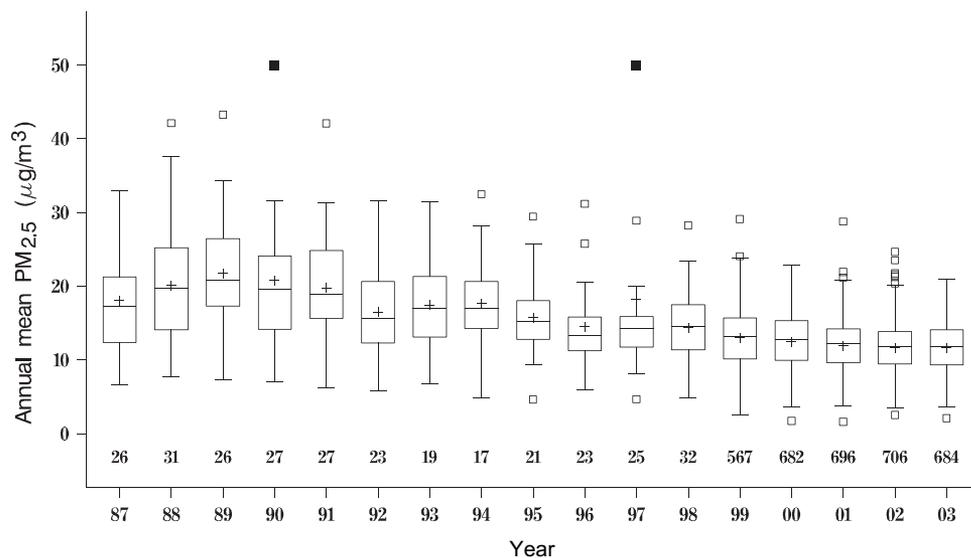


FIGURE 2. Box plots of county-specific averages of particulate matter $\leq 2.5 \mu\text{m}$ in aerodynamic diameter ($\text{PM}_{2.5}$) for each year for the period 1987–2003 in the United States. The numbers of US counties for which $\text{PM}_{2.5}$ data were available are denoted at the bottom of the box plots. The box denotes the low quartile, the median, and the upper quartile. The vertical extensions denote the upper 5th and 97.5th percentiles. The open squares denote the outliers. The two filled squares denote two clipped outliers.

TABLE 2. Estimates and 95% posterior intervals for the national and regional (eastern United States and western United States) percentage increase in all-cause, cardiorespiratory, and other-cause mortality associated with a 10- $\mu\text{g}/\text{m}^3$ increase in PM_{10} * at lag 1 for the periods 1987–1994, 1995–2000, and 1987–2000

	1987–1994	95% PI	1995–2000	95% PI	1987–2000	95% PI
All cause						
East	0.29	0.12, 0.46	0.13	-0.19, 0.44	0.25	0.11, 0.39
West	0.12	-0.07, 0.30	0.18	-0.07, 0.44	0.12	-0.02, 0.26
National	0.21	0.10, 0.32	0.18	0.00, 0.35	0.19	0.10, 0.28
Cardiorespiratory						
East	0.39	0.16, 0.63	0.30	-0.13, 0.73	0.34	0.15, 0.54
West	0.17	-0.07, 0.40	0.13	-0.23, 0.50	0.14	-0.05, 0.33
National	0.28	0.14, 0.43	0.21	-0.03, 0.44	0.24	0.13, 0.36
Other						
East	0.21	-0.03, 0.44	0.00	-0.49, 0.50	0.15	-0.09, 0.39
West	0.09	-0.21, 0.38	0.23	-0.15, 0.62	0.11	-0.10, 0.33
National	0.15	-0.02, 0.32	0.17	-0.07, 0.41	0.15	0.00, 0.29

* PM_{10} , particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter; PI, posterior interval.

modification by average level using the NMMAPS data for 1987–2000.

Our analytic approach could also be used to explore geographic heterogeneity in trends of risk as various pollution control measures are implemented in different regions. Analyses directed at comparing regional trends in short-term relative rates among geographic areas affected and not affected by a regulation may provide insights into the pollution sources of particular relevance to improving public health. For example, the first phase of the Acid Rain Control Program, initiated in 1995, required a 50 percent reduction in emissions of sulfur dioxide compared with 1980 levels, and it also required reductions in nitrogen oxides from fossil-fuel-powered electric utility power plants in the eastern part of the United States (25). Consequently, the

chemical composition of particulate matter in the eastern part of the United States affected by the Acid Rain Control Program has changed substantially, with a drop of 21 percent in the level of sulfate particulate matter between 1999 and 2003 (26). Thus, the sharp decline in sulfur dioxide emissions in the eastern United States as a result of the Acid Rain Control Program could explain the pattern of observed regional heterogeneity of trends in short-term effects of PM_{10} .

National pollution control programs targeting diesel engine emissions from heavy-duty trucks and buses would also be expected to reduce the contribution of elemental carbon to the composition of ambient particulate matter over time. Stricter emission standards for passenger vehicles would also be expected to reduce ambient organic carbon and ammonium nitrate particle levels. Such reductions would be

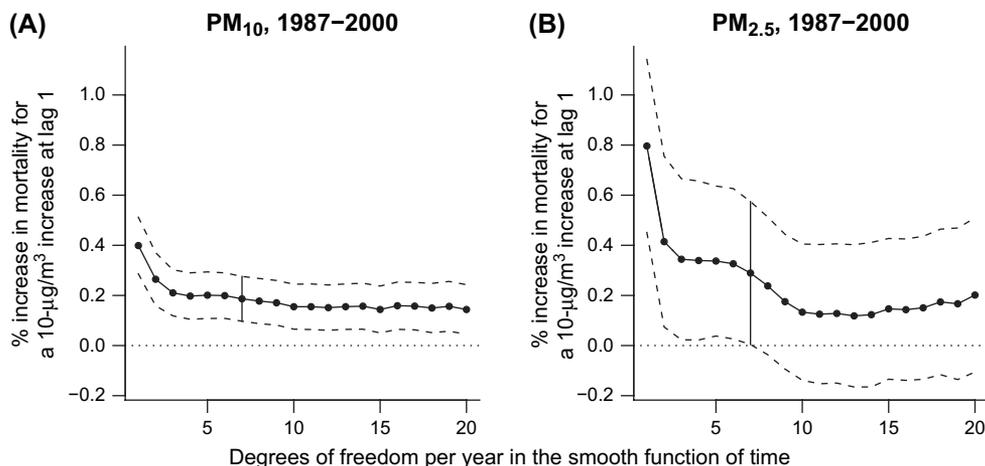


FIGURE 3. National average estimates of the percentage increase in all-cause mortality associated with a 10- $\mu\text{g}/\text{m}^3$ increase in particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter (PM_{10}) (A) and particulate matter $\leq 2.5 \mu\text{m}$ in aerodynamic diameter ($\text{PM}_{2.5}$) (B) at lag 1 as a function of the number of degrees of freedom in the smooth function of time for the United States for the periods 1987–2000 and 1999–2000, respectively. The vertical segment denotes the point estimate and the 95% posterior interval of the national average relative rate at 7 degrees of freedom per year.

specifically anticipated in California, where significantly stricter motor vehicle emission limits for hydrocarbons and nitrogen oxides than national standards were adopted. However, fleet turnover rates lead to relatively long lead times between implementation of motor vehicle emission standards, and their full impact on PM_{10} composition was probably not achieved during our study period.

Few studies have addressed changes in health impacts associated with changes in levels and composition of air pollution. Recent studies cited as examples of accountability research have focused on acute changes in sources and levels of air pollution (1). Even by the 1960s, however, Martin (27) recognized that the health effects of air pollution could be tracked over time; before time-series methods were in wide use, he examined correlations of pollution measures with mortality counts over successive winters in London, United Kingdom. Lawther et al. (28) followed the relation of symptoms with air pollution indicators in persons with chronic obstructive pulmonary disease in London across the 1960s. The association weakened as pollution levels declined. In Germany, air pollution generated by fossil fuel combustion declined quickly across the 1990s in the eastern portion of the country (29). Several studies have addressed the consequences of this decline. Heinrich et al. (30) carried out serial cross-sectional studies among schoolchildren over this time period and found a decreasing prevalence of respiratory symptoms. A study of similar design found that lung function became comparable in schoolchildren in the eastern and western regions of the country over this decade (31). Two recent analyses by Laden et al. (32) and Janes et al. (33) more directly assessed the accountability question: Is the drop in mortality rates largest in cities with the largest reduction in long-term average particulate matter? Laden et al. provided supporting evidence that this is occurring by analyzing follow-up data from the Harvard Six Cities Study (34) for 1980–1998, whereas Janes et al. used data on 113 US counties for 1999–2002 from the National Medicare Cohort Study (33).

Our study does not provide direct evidence concerning whether the decrease in pollution levels has resulted in health benefits. Rather, we focused on the following question: Did the short-term association between PM_{10} mass concentration and mortality weaken during a period of increasingly stringent air quality control programs that changed the particulate matter source mix? We could not address the overall burden of air-pollution-associated mortality because of the adjustment for longer-term trend. We assessed changes in short-term relative risks independently from changes in the level of particulate matter. For example, acute toxicity of particulate matter may have increased consequent to technological changes that could have altered the chemical composition of particulate matter, whereas levels of particulate matter are decreasing over time (35).

There are significant challenges to interpreting trends in the size of the short-term effects of PM_{10} as evidence for the efficacy of regulations. These challenges include 1) limited power to estimate trends in short-term effects and 2) selection of the appropriate geographic level (i.e., national, regional, state, local) to evaluate the potential linkages between regulatory interventions, changes in particulate matter composition, and changes in air quality and health

outcomes. In the Appendix are sample size calculations to project the gain in statistical power for detecting changes in the short-term effects of PM_{10} over time as a function of additional years of data. Nevertheless, any accountability analyses must rely on data and trend analyses such as those presented here. Hence, it is important to continue to update data and methods for quantifying temporal and spatial patterns in the particulate matter effects. For example, using national Medicare data, we found that short-term exposure to $PM_{2.5}$ was associated with increased risk of hospital admissions for cardiovascular and respiratory diseases on average across 204 US counties during 1999–2002 (36). Because the Medicare data will be updated routinely, we will have the opportunity to conduct improved surveillance of air pollution-related health effects.

Further reductions in air pollution to protect the public's health are increasingly expensive. Outdoor levels of key pollutants have declined over time and their chemical composition is likely to have changed, but epidemiologic studies continue to show adverse health effects at these lower levels. Regulators need increasingly refined information about the sources of pollutants contributing most substantially to risk of adverse effects to create cost-efficient and more focused control strategies. Our methodology for accountability research, which is based on tracking short-term effects of particulate matter on health indicators over time, has the potential to inform the regulatory processes at the national, regional, and local levels with regard to the priority placed on control measures for different sources of particulate matter.

ACKNOWLEDGMENTS

Funding for Drs. Dominici, Zeger, and Samet was provided by the US Environmental Protection Agency (RD-83241701). Funding for Drs. Dominici, Zeger, Peng, and Samet was also provided by the National Institute for Environmental Health Sciences (ES012054-03 and ES012054) and by the NIEHS Center in Urban Environmental Health (P30 ES 03819). Funding for Drs. Dominici, Peng, and Zeger and for Mr. White is also provided by the US Environmental Protection Agency (RD-833622010). Although the research described in this article was funded wholly or in part by the US Environmental Protection Agency through grant agreement RD-83241701 to Johns Hopkins University, it was not subjected to the Agency's required peer and policy review and therefore does not necessarily reflect the views of the Agency, and no official endorsement should be inferred.

The authors thank Howard Chang for his help with the sample size calculations. The authors also thank Charlotte Gerczak for editorial input and Rebecca Nachman for research assistance.

Conflict of interest: none declared.

REFERENCES

1. Health Effects Institute. Assessing health impact of air quality regulations: concepts and methods for accountability research.

- Boston, MA: HEI Accountability Working Group, 2003. (HEI communication 11).
2. Chay K, Greenstone M. The impact of air quality on infant mortality: evidence from geographic variation in pollution shocks induced by a recession. *Q J Econ* 2003;118: 1121–67.
 3. Chay K, Dobkin C, Greenstone M. The Clean Air Act of 1970 and adult mortality. *J Risk Uncertainty* 2003;27: 279–300.
 4. Clancy L, Goodman P, Sinclair H, et al. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. *Lancet* 2002;360:1210–14.
 5. Hedley AJ, Wong CM, Thach TQ, et al. Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study. *Lancet* 2002;360: 1646–52.
 6. Friedman MS, Powell KE, Hutwagner L, et al. Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA* 2001;285:897–905.
 7. Samet JM, Zeger S, Dominici F, et al. The National Morbidity, Mortality, and Air Pollution Study (NMMAPS). Part 2. Morbidity and mortality from air pollution in the United States. Boston, MA: Health Effects Institute, 2000. (<http://pubs.healtheffects.org/view.php?id=118>).
 8. Samet JM, Dominici F, Curriero FC, et al. Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *N Engl J Med* 2000;343:1742–9.
 9. Samet JM, Zeger S, Dominici F, et al. The National Morbidity, Mortality, and Air Pollution Study (NMMAPS). Part I. Methods and methodological issues. Boston, MA: Health Effects Institute, 2000. (<http://pubs.healtheffects.org/view.php?id=228>).
 10. Dominici F, Daniels M, Zeger SL, et al. Air pollution and mortality: estimating regional and national dose-response relationships. *J Am Stat Assoc* 2002;97:100–11.
 11. Dominici F, McDermott A, Zeger SL, et al. On the use of generalized additive models in time-series studies of air pollution and health. *Am J Epidemiol* 2002;156: 193–203.
 12. Lindley DV, Smith AFM. Bayes estimates for the linear model (with discussion). *J R Stat Soc (B)* 1972;34:1–41.
 13. Gelman A, Carlin JB, Stern HS, et al. Bayesian data analysis. New York, NY: Chapman and Hall, 2003.
 14. McCullagh P, Nelder JA. Generalized linear models. New York, NY: Chapman and Hall, 1989.
 15. Kelsall JE, Samet JM, Zeger SL, et al. Air pollution and mortality in Philadelphia, 1974–1988. *Am J Epidemiol* 1997; 146:750–62.
 16. Dominici F, McDermott A, Daniels M, et al. Revised analyses of time-series studies of air pollution and health: (1) Mortality among residents of 90 cities. Boston, MA: Health Effects Institute, 2003.
 17. Peng RD, Dominici F, Louis T. Model choice in multi-site time series studies of air pollution and mortality. *J R Stat Soc (A)* 2006;169:179–203.
 18. DuMouchel WH, Harris JE. Bayes methods for combining the results of cancer studies in humans and other species. *J Am Stat Assoc* 1983;78:293–308.
 19. Everson P, Morris C. Inference for multivariate normal hierarchical models. *J R Stat Soc (B)* 2000;62:399–412.
 20. Dominici F, McDermott A, Daniels M, et al. A report to the Health Effects Institute: reanalysis of the NMMAPS database. Boston, MA: Health Effects Institute, 2002. (<http://pubs.healtheffects.org/view.php?id=4>).
 21. Peng RD, Dominici F, Pastor-Barriuso R, et al. Seasonal analyses of air pollution and mortality in 100 US cities. *Am J Epidemiol* 2005;161:585–94.
 22. Chiogna M, Gaetan C. Dynamic generalized linear models with application to environmental epidemiology. *Appl Stat* 2002;51:453–68.
 23. Peng RD, Dominici F, Zeger SL. Reproducible epidemiologic research. *Am J Epidemiol* 2006;163:783–9.
 24. Daniels MJ, Dominici F, Samet JM, et al. Estimating particulate matter-mortality dose-response curves and threshold levels: an analysis of daily time-series for the 20 largest US cities. *Am J Epidemiol* 2000;152:397–406.
 25. US Environmental Protection Agency. Clean Air Act 1990. (<http://www.epa.gov/oar/caa/contents.html>).
 26. US Environmental Protection Agency. The particle pollution report: current understanding of air quality and emissions through 2003. Research Triangle Park, NC: Office of Air Quality Planning and Standards. December 2004. (EPA publication no. EPA454-R-04-002). (<http://www.epa.gov/air/airtrends/aqtrnd04/pm.html>).
 27. Martin AE. Mortality and morbidity statistics and air pollution. *Proc R Soc Med* 1964;57(suppl):969–75.
 28. Lawther PJ, Waller RE, Henderson M. Air pollution and exacerbations of bronchitis. *Thorax* 1970;25:525–39.
 29. Ebel S, Brauer M, Cyrus J, et al. Air quality in postunification Erfurt, East Germany: associating changes in pollutant concentrations with changes in emissions. *Environ Health Perspect* 2001;109:325–33.
 30. Heinrich J, Hoelscher B, Frye C, et al. Improved air quality in reunified Germany and decreases in respiratory symptoms. *Epidemiology* 2002;13:394–401.
 31. Sugiri D, Ranft U, Schikowski T, et al. The influence of large-scale airborne particle decline and traffic-related exposure on children's lung function. *Environ Health Perspect* 2006;114: 282–8.
 32. Laden F, Schwartz J, Speizer FE, et al. Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard Six Cities study. *Am J Respir Crit Care Med* 2006;173:667–72.
 33. Janes H, Dominici F, Zeger S. Trends in air pollution and mortality: an approach to the assessment of unmeasured confounding. *Epidemiology* 2007;18:416–23.
 34. 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.
 35. US Environmental Protection Agency (EPA). Air quality criteria for particulate matter. Research Triangle Park, NC: USEPA, National Center for Environmental Assessment, 2004. (EPA/600/p-99/022aD and bD).
 36. Dominici F, Peng RD, Bell ML, et al. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA* 2006;295:1127–34.

APPENDIX

Sample Size Consideration

For a particular county, the following model is set up:

$$\log \mu_t = \log \mu_0 + \beta_1 PM_{10,t}^* + \beta_2 PM_{10,t}^* \times t, \\ t = 0, \dots, 365 \times T \text{ (day)}$$

$$PM_{10,t}^* = PM_{10,t} - S(PM_{10,t}).$$

$PM_{10,t}^*$ is the detrended time series, with $S(PM_{10,t})$ being the natural cubic spline smooth function with 4 degrees of freedom per year. Calendar day t takes integer values, and T is the total number of years. Also, let $\beta_2 = -\gamma \beta_1 / (365 \times 15)$ so that the effect of $PM_{10,t}^*$ decreases linearly. After 15 years, the effect is $\beta_1 (1 - \gamma)$; therefore, we can rewrite the Poisson model as

$$\log \mu_t = \log \mu_0 + \left[\beta_1 \left(1 - \frac{\gamma}{365 \times 15} t \right) \right] PM_{10,t}^*,$$

$$t = 0, \dots, 365 \times T(\text{day}).$$

The rate of decrease is therefore a function of γ . For various values of μ_0 , β_1 , γ , and T , Chicago, Illinois' daily $PM_{10,t}$ were used to obtain μ_t . The corresponding asymptotic variance for the estimation of β_2 given by $\text{diag}(X'V^{-1}X)^{-1}_{[\beta_2]}$ is summarized below in the form of $\beta_2/\text{SE}(\beta_2)$ (SE, standard error).

Year T	β_1 ($\times 1,000$)	γ	$\beta_2/\text{SE}(\beta_2)$			
			$\mu_0 = 10$	$\mu_0 = 30$	$\mu_0 = 50$	$\mu_0 = 100$
15	0.5	0.75	0.45	0.79	1.01	1.43
15	0.5	1	0.60	1.05	1.35	1.91
15	1	0.75	0.91	1.58	2.04	2.88
15	1	1	1.21	2.10	2.71	3.83
20	0.5	0.75	0.71	1.23	1.58	2.24
20	0.5	1	0.94	1.63	2.11	2.98
20	1	0.75	1.42	2.46	3.18	4.49
20	1	1	1.89	3.28	4.23	5.98
25	0.5	0.75	0.98	1.70	2.19	3.10
25	0.5	1	1.30	2.26	2.91	4.12
25	1	0.75	1.96	3.40	4.39	6.20
25	1	1	2.61	4.52	5.83	8.25
30	0.5	0.75	1.30	2.24	2.90	4.10
30	0.5	1	1.73	2.99	3.86	5.46
30	1	0.75	2.60	4.49	5.80	8.21
30	1	1	3.45	5.98	7.72	10.91