SPECIAL ARTICLE

Fine-Particulate Air Pollution and Life Expectancy in the United States

C. Arden Pope III, Ph.D., Majid Ezzati, Ph.D., and Douglas W. Dockery, Sc.D.

ABSTRACT

BACKGROUND

Exposure to fine-particulate air pollution has been associated with increased morbidity and mortality, suggesting that sustained reductions in pollution exposure should result in improved life expectancy. This study directly evaluated the changes in life expectancy associated with differential changes in fine particulate air pollution that occurred in the United States during the 1980s and 1990s.

METHODS

We compiled data on life expectancy, socioeconomic status, and demographic characteristics for 211 county units in the 51 U.S. metropolitan areas with matching data on fine-particulate air pollution for the late 1970s and early 1980s and the late 1990s and early 2000s. Regression models were used to estimate the association between reductions in pollution and changes in life expectancy, with adjustment for changes in socioeconomic and demographic variables and in proxy indicators for the prevalence of cigarette smoking.

RESULTS

A decrease of 10 μg per cubic meter in the concentration of fine particulate matter was associated with an estimated increase in mean (±SE) life expectancy of 0.61±0.20 year (P=0.004). The estimated effect of reduced exposure to pollution on life expectancy was not highly sensitive to adjustment for changes in socioeconomic, demographic, or proxy variables for the prevalence of smoking or to the restriction of observations to relatively large counties. Reductions in air pollution accounted for as much as 15% of the overall increase in life expectancy in the study areas.

CONCLUSIONS

A reduction in exposure to ambient fine-particulate air pollution contributed to significant and measurable improvements in life expectancy in the United States.

From the Department of Economics, Brigham Young University, Provo, UT (C.A.P.); the Harvard School of Public Health, Boston (M.E., D.W.D.); and the Harvard Initiative for Global Health, Cambridge, MA (M.E.). Address reprint requests to Dr. Pope at 142 FOB, Brigham Young University, Provo, UT 84602-2363, or at cap3@byu.edu.

N Engl J Med 2009;360:376-86.

SINCE THE 1970S, THE UNITED STATES HAS made substantial efforts and investments to improve air quality. As these efforts continue, a fundamental question remains: Do improvements in air quality result in measurable improvements in human health and longevity? Associations between long-term exposure to fine-particulate air pollution and mortality have been observed in population-based studies[1-3] and, more recently, in cohort-based studies.[4-11] Daily time-series and related studies,[12-15] natural intervention studies,[16-18] and cohort studies[10,19] all support the view that relatively prompt and sustained health benefits are derived from improved air quality.

We directly assessed associations between life expectancy and fine-particulate air pollution in 51 U.S. metropolitan areas, comparing data for the period from the late 1970s to the early 1980s with matched data for the period from the late 1990s to the early 2000s. We hypothesized that temporal changes in fine-particulate air pollution between 1980 and 2000 would be associated with changes in life expectancy. Specifically, we hypothesized that metropolitan areas with the largest declines in fine-particulate pollution would have the largest increases in life expectancy, even after adjustment for changes in various socioeconomic and demographic characteristics and proxy variables for status with regard to smoking.

METHODS

DATA COLLECTION AND STUDY AREAS

For the years 1979 through 1983, the U.S. Environmental Protection Agency (EPA) maintained the Inhalable Particle Monitoring Network for research purposes. The network sampled particulate matter in the air using dichotomous samplers with 15-μm and 2.5-μm cutoff points. On the basis of these data, from 1979 through 1983, mean concentrations of particulate matter with an aerodynamic diameter less than or equal to 2.5 μm ($PM_{2.5}$) were calculated for 61 U.S. metropolitan areas and used in the reanalysis and extended analyses of the American Cancer Society prospective cohort study.[6,7] (Metropolitan-area–specific means are presented in Appendix D of the American Cancer Society reanalysis report.[6]) After 1983, no broad-based monitoring network systematically and routinely collected $PM_{2.5}$ data until the promulgation of the National Ambient Air Quality Standard for $PM_{2.5}$ in 1997.[20] As required by the new $PM_{2.5}$ standard, many sites began measuring $PM_{2.5}$ in 1999. Daily $PM_{2.5}$ data were extracted from the EPA's Aerometric Information Retrieval System database for 1999 and the first three quarters of 2000. Data for the four quarters were averaged when more than 50% of the samples and 45 or more total sampling days were available for at least one of the two corresponding quarters in each year. Measurements were averaged first by monitoring site and then by metropolitan area. These calculated mean concentrations of $PM_{2.5}$ were available for 116 U.S. metropolitan areas and were used as part of the extended analysis of the American Cancer Society study.[7] There were 51 metropolitan areas with matching $PM_{2.5}$ data for the early 1980s and the late 1990s.

As part of a nationwide analysis of disparities in mortality across the counties, standard life-table techniques[21] were used to estimate annual life expectancies for more than 2000 individual or merged county units, on the basis of individual death records from national mortality statistics and population data from the U.S. Census, as described in more detail elsewhere.[22] For the purposes of this study, life expectancy for the 215 county units that were part of the 51 metropolitan areas with matching $PM_{2.5}$ data were included. The metropolitan areas were distributed throughout the United States (Fig. 1). For each county unit, life expectancy was calculated with the use of pooled death and population data for the 5-year periods 1978 through 1982 and 1997 through 2001. Because borough-specific death statistics were unavailable for the five boroughs of New York for the earlier period, the boroughs were treated as a single unit, resulting in 211 distinct county-level observations. As described elsewhere,[22] U.S. Census data were used to collect information on county-level socioeconomic and demographic variables, including population, income, and proportions of persons in the population who were high-school graduates, had urban residences, had not lived in their current county of residence 5 years before the census (5-year immigration), and reported that they were white, black, or Hispanic. Income was adjusted for inflation (base year, 2000).

In accordance with previous analyses,[23,24] age-standardized death rates for lung cancer and chronic obstructive pulmonary disease (COPD) were used as indicators of accumulated exposure

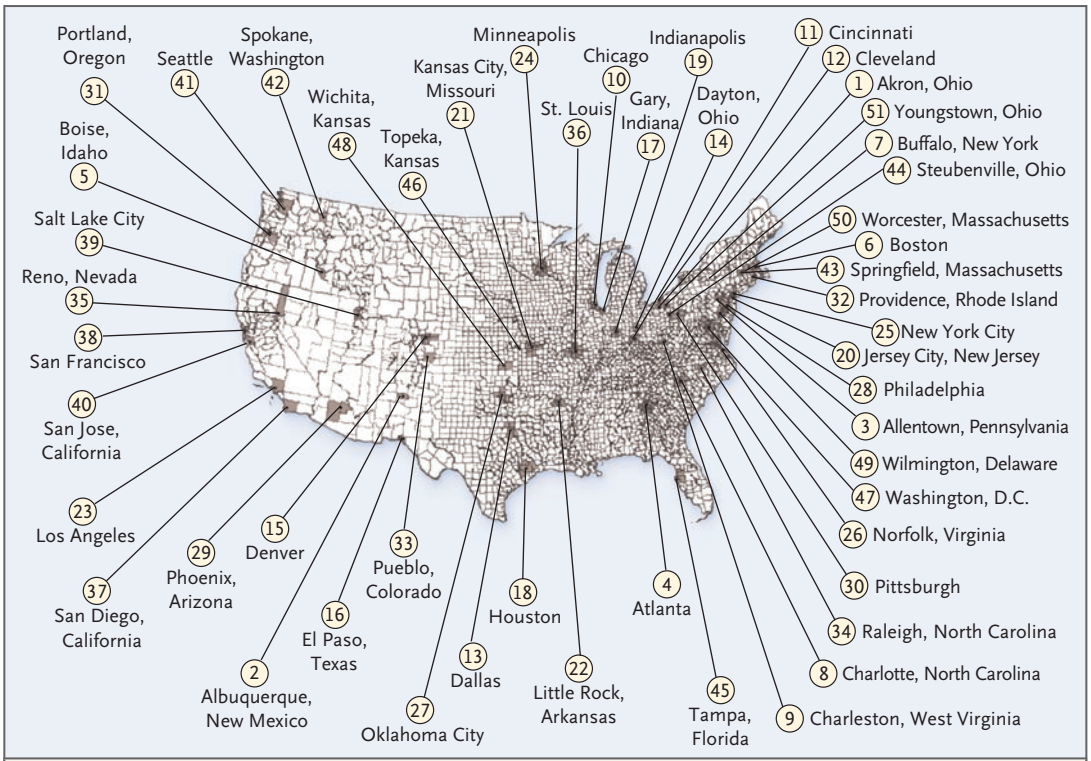

Figure 1. Distribution of Study Areas.

The locations of the counties included in the study are shown in gray, and the dots represent the approximate locations of the 51 metropolitan areas in the study. The metropolitan areas are coded by number as follows: 1 — Akron, Ohio; 2 — Albuquerque, New Mexico; 3 — Allentown, Pennsylvania; 4 — Atlanta; 5 — Boise, Idaho; 6 — Boston; 7 — Buffalo, New York; 8 — Charlotte, North Carolina; 9 — Charleston, West Virginia; 10 — Chicago; 11 — Cincinnati; 12 — Cleveland; 13 — Dallas; 14 — Dayton, Ohio; 15 — Denver; 16 — El Paso, Texas; 17 — Gary, Indiana; 18 — Houston; 19 — Indianapolis; 20 — Jersey City, New Jersey; 21 — Kansas City, Missouri; 22 — Little Rock, Arkansas; 23 — Los Angeles; 24 — Minneapolis; 25 — New York City; 26 — Norfolk, Virginia; 27 — Oklahoma City; 28 — Philadelphia; 29 — Phoenix, Arizona; 30 — Pittsburgh; 31 — Portland, Oregon; 32 — Providence, Rhode Island; 33 — Pueblo, Colorado; 34 — Raleigh, North Carolina; 35 — Reno, Nevada; 36 — St. Louis; 37 — San Diego, California; 38 — San Francisco; 39 — Salt Lake City; 40 — San Jose, California; 41 — Seattle; 42 — Spokane, Washington; 43 — Springfield, Massachusetts; 44 — Steubenville, Ohio; 45 — Tampa, Florida; 46 — Topeka, Kansas; 47 — Washington, D.C.; 48 — Wichita, Kansas; 49 — Wilmington, Delaware; 50 — Worcester, Massachusetts; 51 — Youngstown, Ohio.

to smoking. There were two reasons for using these indirect indicators of smoking. First, for most study areas, data on the prevalence of smoking are not available for the late 1970s and early 1980s, and second, the measures for lung cancer and COPD indicate the population's cumulative exposure to smoking. The *International Classification of Diseases, 10th Revision* (ICD-10) was used to calculate death rates for lung cancer (ICD-10 codes C33-C34 and D02.1-D02.2) and COPD (ICD-10 code J40-J44). The death rates were based on the underlying cause of death in individual death records from national mortality statistics and population data from the U.S. Census, pooled for the same 5-year periods as life expectancy. Death rates were calculated in 5-year age groups, and were age-standardized for the 2000 U.S. population of adults 45 years of age or older (rates of death from these diseases are unstable among younger adults because there is such a small number of cases). Additional estimates of changes in the prevalence of cigarette smoking were obtained from health surveys for use in sensitivity analyses of a subgroup of the metropolitan areas with data in both periods. The prevalence of smoking among adults in metropolitan areas for the years 1998 through 2002 could be estimated for 50 of the 51 metropolitan study areas from the Behavioral Risk Factor Surveillance System (www.cdc.gov/brfss/technical_infodata/surveydata.htm);

the prevalence for the years 1978 through 1980 could be estimated for 24 of the metropolitan study areas with data from the National Health Interview Survey (www.cdc.gov/nchs/nhis.htm). The change in the prevalence of smoking was estimated for each of 24 metropolitan areas on the basis of data from these sources for both periods.

STATISTICAL ANALYSIS

For both 5-year periods, life expectancies were plotted against $PM_{2.5}$ concentrations, and increases in life expectancy from the first period to the second were plotted against reductions in the $PM_{2.5}$ concentration. Cross-sectional regression models were estimated for both time periods, and first-difference regression models were estimated by regressing increases in life expectancy against reductions in monitored $PM_{2.5}$ concentrations. The sensitivity of the estimates on the pollution-related effect was explored with the use of five different approaches: including combinations of demographic, socioeconomic, and proxy variables for prevalence of smoking in the models, restricting the analysis to counties that had a population of 100,000 or more in 1986 or to the 51 largest counties in each metropolitan area, estimating population-weighted regression models, stratifying the analysis according to the pollution levels for 1979 through 1983 (in order to evaluate the influence of baseline pollution levels), and including direct measures of change in the prevalence of smoking for the subgroup of study areas with adequate survey data on smoking. Because of the potential for lack of statistical independence between counties in the same metropolitan area, clustered standard errors that were robust with regard to within-cluster correlation[25,26] (clustered by the 51 metropolitan areas) were estimated for all models except for the analysis that included only the 51 largest counties in each metropolitan area. Models were estimated with the use of PROC REG and PROC SURVEYREG in SAS, version 9.2 (SAS Institute).

RESULTS

Summary statistics for key study variables are listed in Table 1. In Figures 2 and 3, cross-sectional life expectancies are plotted against air-pollution data for the earlier and later time periods, respectively. At least five observations can be made on the basis of the data presented in these two

Table 1. Summary Characteristics of the 217 Counties Analyzed.*

Variable	Mean Value
Life expectancy (yr)	
1978–1982	74.32±1.52
1997–2001	77.04±1.82
Change	2.72±0.93
$PM_{2.5}$ ($\mu g/m^3$)	
1979–1983	20.61±4.36
1999–2000	14.10±2.86
Reduction	6.52±2.94
Per capita income (in thousands of $)	
1979	15.18±2.64
1999	23.67±5.05
Change	8.49±3.16
Population (in hundreds of thousands)	
1980	3.83±8.47
2000	4.82±10.13
Change	0.99±2.26
5-Year in-migration (proportion of population)†‡	
1980	0.25±0.10
2000	0.24±0.08
Change	–0.01±0.06
Urban residence (proportion of population)†	
1980	0.58±0.33
2000	0.78±0.22
Change	0.20±0.18
High-school graduates (proportion of population)†	
1980	0.68±0.11
2000	0.87±0.05
Change	0.19±0.15
Black population (proportion of population)†§	
1980	0.097±0.12
2000	0.115±0.13
Change	0.018±0.06
Hispanic population (proportion of population)†§	
1980	0.035±0.072
2000	0.068±0.093
Change	0.033±0.043
Deaths from lung cancer (no./10,000 population)	
1979–1983	14.38±2.95
1997–2001	16.73±3.27
Change	2.35±2.77
Deaths from COPD (no./10,000 population)	
1979–1983	7.92±1.85
1997–2001	12.37±2.71
Change	4.45±2.43

* Plus–minus values are means ±SD. COPD denotes chronic obstructive pulmonary disease, and $PM_{2.5}$ particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.
† Proportions of the population are based on U.S. Census data.
‡ Five-year in-migration refers to the proportion of the population who did not reside in the county 5 years earlier.
§ Data on race and ethnic group were self-reported.

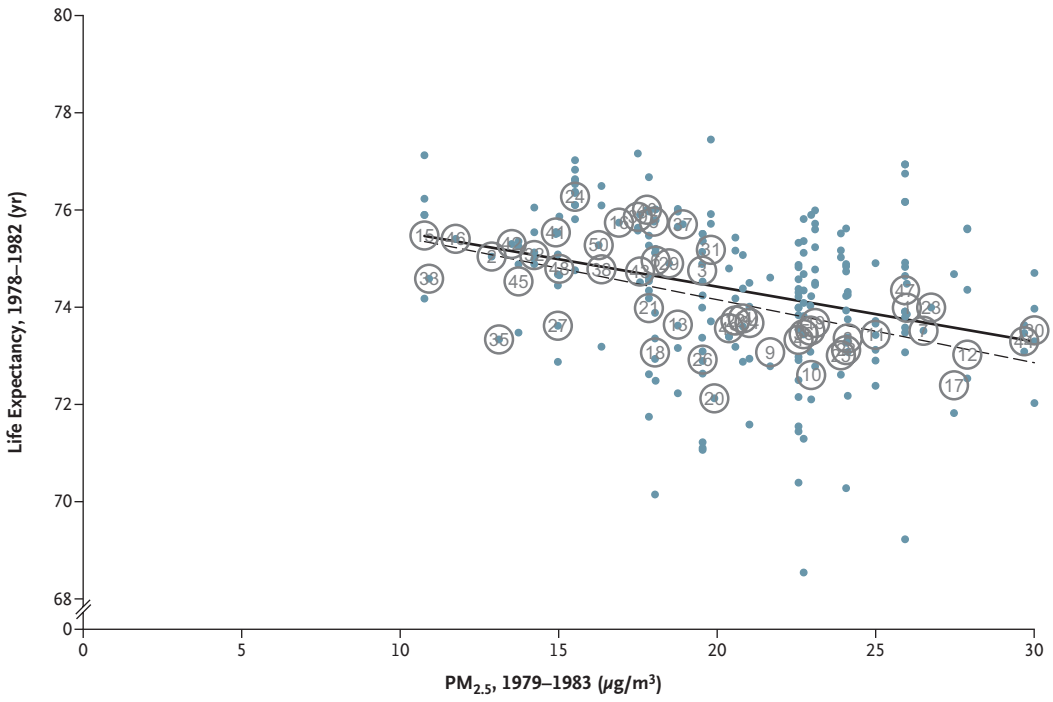

Figure 2. Cross-Sectional Life Expectancies for 1978–1982, Plotted against $PM_{2.5}$ Concentrations for 1979–1983.

Dots and circles labeled with numbers represent population-weighted mean life expectancies at the county level and the metropolitan-area level, respectively. The solid and broken lines represent regression lines with the use of county-level and metropolitan-area–level observations, respectively. The metropolitan areas are coded by number as follows: 1 — Akron, Ohio; 2 — Albuquerque, New Mexico; 3 — Allentown, Pennsylvania; 4 — Atlanta; 5 — Boise, Idaho; 6 — Boston; 7 — Buffalo, New York; 8 — Charlotte, North Carolina; 9 — Charleston, West Virginia; 10 — Chicago; 11 — Cincinnati; 12 — Cleveland; 13 — Dallas; 14 — Dayton, Ohio; 15 — Denver; 16 — El Paso, Texas; 17 — Gary, Indiana; 18 — Houston; 19 — Indianapolis; 20 — Jersey City, New Jersey; 21 — Kansas City, Missouri; 22 — Little Rock, Arkansas; 23 — Los Angeles; 24 — Minneapolis; 25 — New York City; 26 — Norfolk, Virginia; 27 — Oklahoma City; 28 — Philadelphia; 29 — Phoenix, Arizona; 30 — Pittsburgh; 31 — Portland, Oregon; 32 — Providence, Rhode Island; 33 — Pueblo, Colorado; 34 — Raleigh, North Carolina; 35 — Reno, Nevada; 36 — St. Louis; 37 — San Diego, California; 38 — San Francisco; 39 — Salt Lake City; 40 — San Jose, California; 41 — Seattle; 42 — Spokane, Washington; 43 — Springfield, Massachusetts; 44 — Steubenville, Ohio; 45 — Tampa, Florida; 46 — Topeka, Kansas; 47 — Washington, D.C.; 48 — Wichita, Kansas; 49 — Wilmington, Delaware; 50 — Worcester, Massachusetts; 51 — Youngstown, Ohio. $PM_{2.5}$ denotes particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.

An interactive version of this figure is available at NEJM.org

figures: $PM_{2.5}$ concentrations generally declined during the 1980s and 1990s; life expectancies increased between the two periods; in both periods there were cross-sectional negative associations between life expectancies and pollution levels; similar negative associations were observed when analyses were performed with the use of county-level or metropolitan-area–level observations; and there was substantial variation, or scatter, around the regression line, indicating that the association with air pollution explains only part of the cross-sectional variation — clearly, other important factors influence life expectancy.

Estimates of the associations between $PM_{2.5}$ and life expectancies with the use of cross-sectional regression models were sensitive to the inclusion of socioeconomic and demographic variables and proxy variables for the prevalence of cigarette smoking and especially the proportion of high-school graduates, which was highly correlated with per capita income. For example, the association between $PM_{2.5}$ concentrations and life expectancy was stronger in the period with less pollution, without adjustment for any covariates. On the basis of regression models without any covariates, an increase in the $PM_{2.5}$ concen-

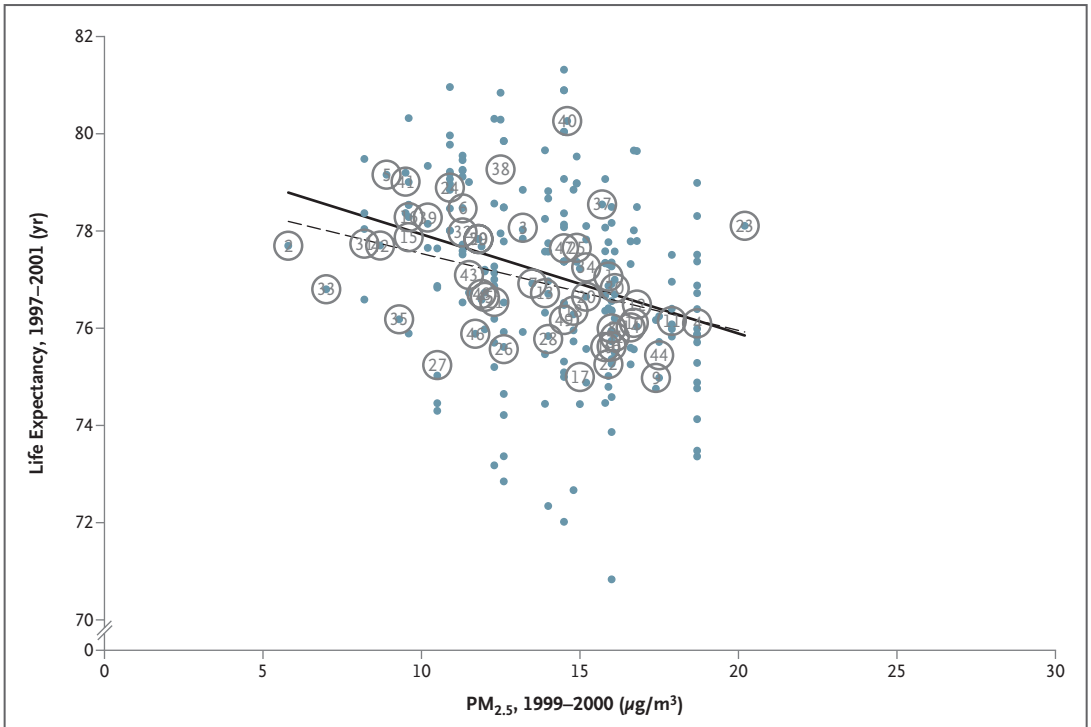

Figure 3. Cross-Sectional Life Expectancies for 1997–2001, Plotted against $PM_{2.5}$ Concentrations for 1999–2000.
Dots and circles labeled with numbers represent population-weighted mean life expectancies at the county level and the metropolitan-area level, respectively. The solid and broken lines represent regression lines with the use of county-level and metropolitan-area–level observations, respectively. The metropolitan areas are coded by number as follows: 1 — Akron, Ohio; 2 — Albuquerque, New Mexico; 3 — Allentown, Pennsylvania; 4 — Atlanta; 5 — Boise, Idaho; 6 — Boston; 7 — Buffalo, New York; 8 — Charlotte, North Carolina; 9 — Charleston, West Virginia; 10 — Chicago; 11 — Cincinnati; 12 — Cleveland; 13 — Dallas; 14 — Dayton, Ohio; 15 — Denver; 16 — El Paso, Texas; 17 — Gary, Indiana; 18 — Houston; 19 — Indianapolis; 20 — Jersey City, New Jersey; 21 — Kansas City, Missouri; 22 — Little Rock, Arkansas; 23 — Los Angeles; 24 — Minneapolis; 25 — New York City; 26 — Norfolk, Virginia; 27 — Oklahoma City; 28 — Philadelphia; 29 — Phoenix, Arizona; 30 — Pittsburgh; 31 — Portland, Oregon; 32 — Providence, Rhode Island; 33 — Pueblo, Colorado; 34 — Raleigh, North Carolina; 35 — Reno, Nevada; 36 — St. Louis; 37 — San Diego, California; 38 — San Francisco; 39 — Salt Lake City; 40 — San Jose, California; 41 — Seattle; 42 — Spokane, Washington; 43 — Springfield, Massachusetts; 44 — Steubenville, Ohio; 45 — Tampa, Florida; 46 — Topeka, Kansas; 47 — Washington, D.C.; 48 — Wichita, Kansas; 49 — Wilmington, Delaware; 50 — Worcester, Massachusetts; 51 — Youngstown, Ohio. $PM_{2.5}$ denotes particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.

An interactive version of this figure is available at NEJM.org

tration of 10 μg per cubic meter was associated with mean (±SE) reductions in life expectancy of 1.19±0.27 years from 1978 to 1982 and 2.02±0.50 years from 1997 to 2001 (P<0.001 for both comparisons). However, models that controlled for income, population, cross-county migration, and the proportion of the population that was black or Hispanic or had an urban residence and that also included proxy variables for the prevalence of smoking showed smaller associations, especially in the second period. An increase of 10 μg per cubic meter in the $PM_{2.5}$ concentration was associated with a reduction in life expectancy of 0.46±0.22 year (P=0.039) from 1978 to 1982 and 0.37±0.20 year (P=0.091) from 1997 to 2001.

In Figure 4, increases in life expectancies are plotted against reductions in $PM_{2.5}$ concentrations from approximately 1980 to 2000. Several additional important observations can be made on the basis of these data: on average, life expectancy increased more in areas with larger reductions in air pollution; similar positive associations were observed between gains in life expectancy and reductions in $PM_{2.5}$ concentrations at the county level and the metropolitan-area level; and there was substantial variation, or scatter, around the

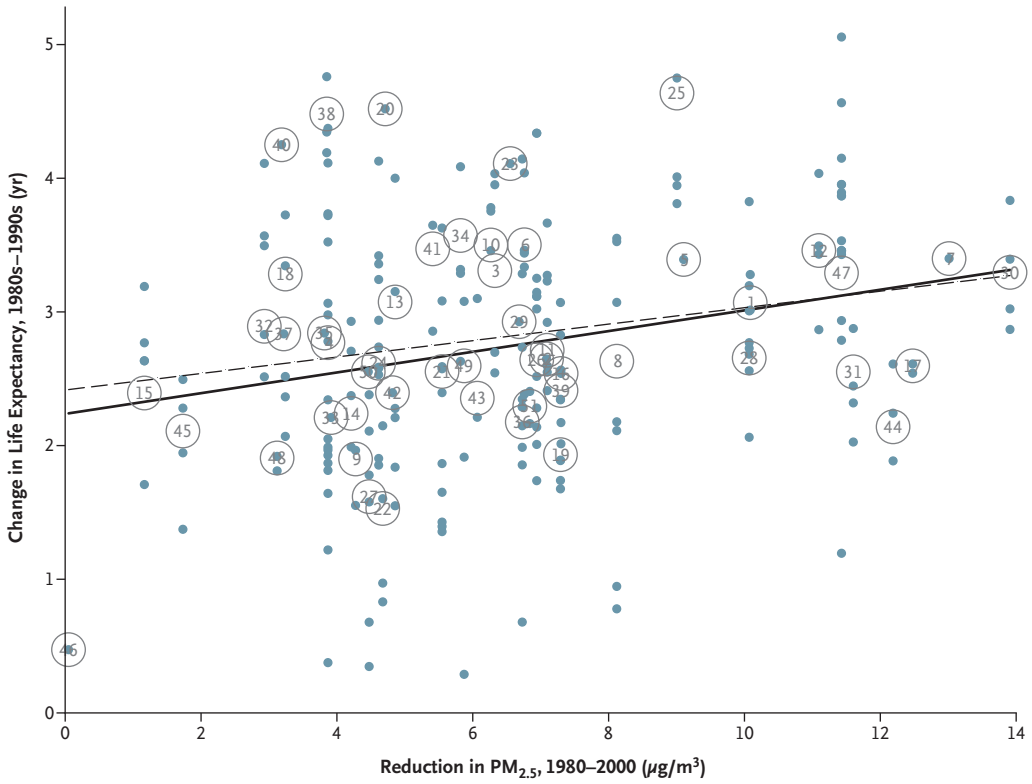

Figure 4. Changes in Life Expectancy for the 1980s–1990s, Plotted against Reductions in $PM_{2.5}$ Concentrations for 1980–2000.

Dots and circles labeled with numbers represent changes in population-weighted mean life expectancies at the county level and metropolitan-area level, respectively. The solid and broken lines represent regression lines with the use of county-level and metropolitan-area–level observations, respectively. The metropolitan areas are coded by number as follows: 1 — Akron, Ohio; 2 — Albuquerque, New Mexico; 3 — Allentown, Pennsylvania; 4 — Atlanta; 5 — Boise, Idaho; 6 — Boston; 7 — Buffalo, New York; 8 — Charlotte, North Carolina; 9 — Charleston, West Virginia; 10 — Chicago; 11 — Cincinnati; 12 — Cleveland; 13 — Dallas; 14 — Dayton, Ohio; 15 — Denver; 16 — El Paso, Texas; 17 — Gary, Indiana; 18 — Houston; 19 — Indianapolis; 20 — Jersey City, New Jersey; 21 — Kansas City, Missouri; 22 — Little Rock, Arkansas; 23 — Los Angeles; 24 — Minneapolis; 25 — New York City; 26 — Norfolk, Virginia; 27 — Oklahoma City; 28 — Philadelphia; 29 — Phoenix, Arizona; 30 — Pittsburgh; 31 — Portland, Oregon; 32 — Providence, Rhode Island; 33 — Pueblo, Colorado; 34 — Raleigh, North Carolina; 35 — Reno, Nevada; 36 — St. Louis; 37 — San Diego, California; 38 — San Francisco; 39 — Salt Lake City; 40 — San Jose, California; 41 — Seattle; 42 — Spokane, Washington; 43 — Springfield, Massachusetts; 44 — Steubenville, Ohio; 45 — Tampa, Florida; 46 — Topeka, Kansas; 47 — Washington, D.C.; 48 — Wichita, Kansas; 49 — Wilmington, Delaware; 50 — Worcester, Massachusetts; 51 — Youngstown, Ohio. $PM_{2.5}$ denotes particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.

An interactive version of this figure is available at NEJM.org

regression line, indicating that factors other than changes in air pollution were influencing the changes in life expectancy.

Table 2 shows regression coefficients for the association between increases in life expectancy and reductions in $PM_{2.5}$ for models with various combinations of socioeconomic and demographic variables and proxy variables for the prevalence of smoking. Table 2 includes models that are restricted to counties with a population of 100,000 or more in 1986 or to the 51 largest counties in each metropolitan area. In all models, increased life expectancies were significantly associated with decreases in $PM_{2.5}$. According to model 4, a decrease of 10 μg per cubic meter in $PM_{2.5}$ was associated with an adjusted increase in life expectancy equal to 0.61±0.20 year. The estimated effect of reduced $PM_{2.5}$ on life expectancy was not highly sensitive after adjustment for changes in socioeconomic and demographic variables and

Table 2. Results of Selected Regression Models, Including Estimates of the Increase in Life Expectancy Associated with a Reduction in $PM_{2.5}$ of 10 μg per Cubic Meter, Adjusted for Socioeconomic, Demographic, and Proxy Indicators for Prevalence of Smoking.*

Variable	Model 1	Model 2	Model 3	Model 4	Model 5†	Model 6‡	Model 7‡
				years			
Intercept	2.25±0.21§	0.80±0.19§	1.78±0.27§	1.75±0.27§	2.02±0.34§	1.71±0.51§	2.09±0.36§
Reduction in $PM_{2.5}$ (10 μg/m³)	0.72±0.29¶	0.83±0.20§	0.60±0.20§	0.61±0.20§	0.55±0.24¶	1.01±0.25§	0.95±0.23§
Change in income (in thousands of $)	—	0.17±0.02§	0.13±0.02§	0.13±0.01§	0.11±0.02§	0.15±0.04§	0.11±0.02§
Change in population (in hundreds of thousands)	—	0.08±0.02§	0.05±0.02§	0.06±0.02§	0.05±0.02§	0.04±0.02	0.05±0.02¶
Change in 5-yr in-migration (proportion of population)‖**	—	0.19±0.79	1.28±0.80	—	—	−0.02±1.83	—
Change in high-school graduates (proportion of population)‖	—	0.17±0.56	−0.11±0.53	—	—	−0.90±0.86	—
Change in urban residence (proportion of population)‖	—	−0.76±0.32¶	−0.40±0.25	—	—	0.03±1.88	—
Change in black population (proportion of population)‖††	—	−1.94±0.58§	−2.74±0.58§	−2.70±0.64§	−2.95±0.78§	−5.06±2.12§	−5.98±1.99§
Change in Hispanic population (proportion of population)‖††	—	1.46±1.23	1.33±1.10	—	—	2.44±2.22	—
Change in lung-cancer mortality rate (no./10,000 population)	—	—	−0.07±0.02§	−0.06±0.02§	−0.07±0.03¶	0.01±0.03	0.02±0.03
Change in COPD mortality rate (no./10,000 population)	—	—	−0.07±0.02§	−0.08±0.02§	−0.09±0.03§	−0.15±0.06§	−0.19±0.05§
No. of county units	211	211	211	211	127	51	51
R^2‡‡	0.05	0.47	0.55	0.53	0.60	0.76	0.74

* Plus–minus values are regression coefficients ±SE. COPD denotes chronic obstructive pulmonary disease, and $PM_{2.5}$ particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.
† This model included only counties with populations of 100,000 or more in 1986.
‡ This model included only counties with the largest 1986 population in the statistical metropolitan area.
§ For these values, P<0.01.
¶ For these values, P<0.05.
‖ Proportions of the population are based on U.S. Census data.
** Five-year in-migration refers to the proportion of the population who did not reside in the county 5 years earlier.
†† Data on race and ethnic group were self-reported.
‡‡ R^2 denotes the coefficient of determination.

proxy variables for the prevalence of smoking or to data restricted to large counties.

In a variety of related sensitivity analyses, the effect estimate for a change in $PM_{2.5}$ was quite robust. In stepwise regressions, a reduction in $PM_{2.5}$ generally entered the model, after changes in per capita income and proxy indicators for prevalence of smoking were introduced; the effect estimate was stable with the inclusion of other variables. When models 4 and 7 in Table 2 were reestimated with the use of weighted regression (weighting by the square root of the average population for the two periods), similar results were observed, with a decrease of 10 μg per cubic meter in $PM_{2.5}$ associated with an estimated increase in life expectancy equal to 0.58±0.20 year for model 4 and 0.86±0.24 year for model 7. Stratified estimates of model 4 in Table 2 were calculated for the 44 counties in the 15 least-polluted metropolitan areas in the earlier period ($PM_{2.5}$, <17 μg per cubic meter) (Fig. 2) as compared with all the other, more-polluted areas. A reduction of 10 μg per cubic meter in $PM_{2.5}$ was associated with an increased life expectancy of 0.95±0.57 for the least-polluted areas and 0.57±0.26 year for other areas; there was no significant difference in the pollution effect for areas that initially had relatively low or high levels of pollution (P≥0.15).

Similarly, the effect estimate for the change in $PM_{2.5}$ was not highly sensitive to the inclusion of survey-based estimates of metropolitan-area–level changes in the prevalence of cigarette smoking. For example, when model 4 in Table 2 was reestimated with the use of data from the 136 counties in the 24 metropolitan areas with matching survey data for the prevalence of smoking, a reduction in $PM_{2.5}$ of 10 μg per cubic meter was associated with an estimated increase in life expectancy of 0.61±0.22 year without inclusion of the change in the variable for smoking prevalence (P=0.011) and 0.64±0.22 year with its inclusion (P=0.007). When model 7 in Table 2 was reestimated with data restricted to the 24 largest counties in the 24 metropolitan areas with matching survey data for the prevalence of smoking, a reduction of 10 μg per cubic meter in $PM_{2.5}$ was associated with an estimated increase in life expectancy of 0.94±0.32 year without inclusion of the change in the variable for smoking prevalence (P=0.007) and 1.00±0.34 years with its inclusion (P=0.008). When added to these models, the change in the prevalence of smoking was not significant (P>0.15), and the estimated effect of a change in the rate of death from COPD was largely unaffected. These results indicate that county-level changes in the rate of death from COPD were more robustly associated with county-level changes in life expectancy than metropolitan-area–level estimates of changes in the prevalence of smoking based on limited survey data.

DISCUSSION

Improvements in life expectancy during the 1980s and 1990s were associated with reductions in fine-particulate pollution across the study areas, even after adjustment for various socioeconomic, demographic, and proxy variables for prevalence of smoking that are associated with health through a range of mechanisms. Indirect calculations point to an approximate loss of 0.7 to 1.6 years of life expectancy that can be attributed to long-term exposure to fine-particulate matter at a concentration of 10 μg per cubic meter, with the use of life tables from the Netherlands and the United States and risk estimates from the prospective cohort studies.[27,28] In the present analysis, a decrease of 10 μg per cubic meter in the fine-particulate concentration was associated with an estimated increase in life expectancy of approximately 0.61±0.20 year — an estimate that is nearly as large as these indirect estimates.

For the approximate period of 1980 through 2000, the average increase in life expectancy was 2.72 years for the counties in this analysis. Reduced air pollution was only one factor contributing to increased life expectancies, with its effects overlapping with those of other factors. On the basis of the average reduction in the $PM_{2.5}$ concentration (6.52 μg per cubic meter) in the metropolitan areas included in this analysis and the effect estimate from model 4 in Table 2, the average increase in life expectancy attributable to the reduced levels of air pollution was approximately 0.4 year (6.52 × 0.061). Multicausality and competing risk issues make it difficult to quantify changes in life expectancy attributable to single risk factors, but these results suggest that the individual effect of reductions in air pollution on life expectancy was as much as 15% of the overall increase. In metropolitan areas where reductions in $PM_{2.5}$ were 13 to 14 μg per cubic meter, the contribution of improvements in air

quality to increases in life expectancy may have been as much as 0.82 year (13.5 × 0.061).

In previous cross-sectional analyses, investigators have observed associations between mortality rates and particulate-air pollution,[1-3] but the size of these associations was sensitive to efforts to control the analyses for potential confounders. Our analysis showed similar sensitivity for the strictly cross-sectional associations with life expectancy. The primary strength of this analysis, however, is the additional use of temporal variations. The availability of data on changes in pollution exposure across metropolitan areas from 1980 to 2000 provides the opportunity for an assessment that is similar to a natural experiment. Cross-sectional characteristics that do not change over time are controlled as if by design. Characteristics that affect life expectancy and that change over time — but not in correlation with changes in pollution — are unlikely to confound the results. Even with underlying spatial correlations, if the temporal changes in these characteristics are relatively less correlated, adjusted effect estimates from temporal regression models are likely to be more robust. In this analysis of differences in temporal changes, the estimated effects of reduced $PM_{2.5}$ exposure on increases in life expectancy were robust in analyses adjusted for socioeconomic, demographic, and proxy variables for the prevalence of smoking, as well as in an analysis restricted to large counties.

From an analytic perspective, it would have been informative if pollution had actually increased in some of the areas that were initially less polluted. However, pollution did not increase in any of the metropolitan areas, and the potential for reducing pollution was greater in the areas that were more polluted initially than in those that were less polluted. Stratified analyses showed no significant differences in pollution effects for the areas that initially had low or high pollution, which is consistent with previous findings on the effects of $PM_{2.5}$ even at relatively low concentrations.[7,10,11,15,19]

An appealing aspect of this analysis is that it is a simple, direct, and transparent exploration of the association between life expectancy and air pollution, with the use of available monitored data on $PM_{2.5}$ for both the first and second time periods. However, limited monitoring of data on $PM_{2.5}$ air pollution, especially for the period from 1979 through 1983, reduced both the number of metropolitan areas that could be included in the analysis and our ability to evaluate spatial and temporal associations with more specificity. Furthermore, because the analysis was population-based, we were limited in our ability to control for additional potential confounders, especially various individual and community risk factors that may have been affected by policies that were broadly related to environmental regulation.

For example, the three variables in the analysis that were most strongly associated with changes in life expectancy are all proxy variables. Increases in per capita income probably serve as a proxy variable for, or are highly correlated with, such factors as access to medical care, higher-quality diets, and healthier lifestyles. The use of rates of death from lung cancer and COPD as proxy variables was necessitated by the lack of reliable data on smoking, especially for the period from 1978 through 1982, yet these rates reflect the cumulative effects of smoking, which may similarly affect life expectancy. Although the large majority of deaths from lung cancer and COPD are attributable to smoking,[23] pollution may also have an effect (albeit much smaller) on these health outcomes,[7,8] potentially leading to conservative estimates of the effects of pollution when such proxies are used. The $PM_{2.5}$ variable may serve, in part, as a proxy variable for copollutants, and changes in $PM_{2.5}$ may represent estimates of changes in area-wide ambient concentrations based on fixed-site monitoring during the two time periods instead of being a direct measure of changes in personal exposures. Nevertheless, U.S. air-quality standards and related public policies are designed to restrict ambient pollutant concentrations in an effort to protect human health.[20] Previous prospective cohort studies, using measures of ambient concentrations of pollutants and controlling for smoking and other individual risk factors, have suggested similar improvements in survival and life expectancy, on the basis of indirect estimates.[4-11] The results of our population-based analysis, which showed similar improvements in life expectancy associated with public-policy–related reductions in ambient pollutant concentrations, corroborate these previous findings.

In conclusion, the results of this analysis are generally good news. Although multiple factors affect life expectancy, our findings provide evidence that improvements in air quality have con-

tributed to measurable improvements in human health and life expectancy in the United States.

Supported by a cooperative agreement with the Centers for Disease Control and Prevention; by grants from the Association of Schools of Public Health (U36/CCU300430-23), the Harvard Environmental Protection Agency (EPA) Particulate Matter Center (EPA RD832416), and the National Institute of Environmental Health Sciences (ES0002); and funds from the Mary Lou Fulton Professorship, Brigham Young University.

Dr. Dockery reports receiving grant support from the Health Effects Institute. No other potential conflict of interest relevant to this article was reported.

The contents of this article are solely the responsibility of the authors and do not necessarily represent the official views of the Centers for Disease Control and Prevention or the Association of Schools of Public Health. Although funded in part by the EPA, this study has not been subjected to the agency's peer- and policy-review process and therefore does not necessarily reflect the views of the agency, and no official endorsement should be inferred.

REFERENCES

1. Lave LB, Seskin EP. Air pollution and human health. Science 1970;169:723-33.
2. Evans JS, Tosteson T, Kinney PL. Cross-sectional mortality studies and air pollution risk assessment. Environ Int 1984;10:55-83.
3. Özkaynak H, Thurston GD. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. Risk Anal 1987;7:449-61.
4. 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.
5. Pope CA III, Thun MJ, Namboodiri MM, et al. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 1995;151:669-74.
6. Krewski D, Burnett RT, Goldberg MS, et al. Reanalysis of the Harvard Six Cities Study and the American Cancer Society study of particulate air pollution and mortality: special report. Cambridge, MA: Health Effects Institute, 2000.
7. Pope CA III, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002;287:1132-41.
8. Pope CA III, Burnett RT, Thurston GD, et al. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 2004;109:71-7.
9. Jerrett M, Burnett RT, Ma R, et al. Spatial analysis of air pollution and mortality in Los Angeles. Epidemiology 2005;16:727-36.
10. Laden F, Schwartz J, Speizer FE, Dockery DW. 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.
11. Miller KA, Siscovick DS, Sheppard L, et al. Long-term exposure to air pollution and incidence of cardiovascular events in women. N Engl J Med 2007;356:447-58.
12. Burnett RT, Goldberg MS. Size-fractionated particulate mass and daily mortality in eight Canadian cities. In: Revised analyses of time-series studies of air pollution and health: special report. Boston: Health Effects Institute, 2003:85-90.
13. Peng RD, Dominici F, Pastor-Barriuso R, Zeger SL, Samet JM. Seasonal analyses of air pollution and mortality in 100 U.S. cities. Am J Epidemiol 2005;161:585-94.
14. Anderson HR, Atkinson RW, Peacock JL, Sweeting MJ, Marston L. Ambient particulate matter and health effects: publication bias in studies of short-term associations. Epidemiology 2005;16:155-63.
15. Pope CA III, Dockery DW. Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc 2006;56:709-42.
16. Clancy L, Goodman P, Sinclair H, Dockery DW. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. Lancet 2002;360:1210-4.
17. Pope CA III, Rodermund DL, Gee MM. Mortality effects of a copper smelter strike and reduced ambient sulfate particulate matter air pollution. Environ Health Perspect 2007;115:679-83.
18. Hedley AJ, Wong CM, Thach TQ, Ma S, Lam TH, Anderson HR. Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study. Lancet 2002;360:1646-52.
19. Schwartz J, Coull B, Laden F, Ryan L. The effect of dose and timing of dose on the association between airbourne particles and survival. Environ Health Perspect 2008;116:64-9.
20. Environmental Protection Agency. Revised requirements for the designation of reference and equivalent methods for $PM_{2.5}$ and ambient air quality surveillance for particulate matter: final rule. Fed Regist 1997;62:5725-6.
21. Preston SH, Heuveline P, Guillot M. Demography. Malden, MA: Blackwell, 2001.
22. Ezzati M, Friedman AB, Kulkarni SC, Murray CJ. The reversal of fortunes: trends in county mortality and cross-county mortality disparities in the United States. PLoS Med 2008;5(4):e66.
23. Peto R, Lopez AD, Boreham J, Thun M, Heath C Jr. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. Lancet 1992;339:1268-78.
24. Eftim SE, Samet JM, Janes H, McDermott A, Dominici F. Fine particulate matter and mortality: a comparison of the Six Cities and American Cancer Society cohorts with a Medicare cohort. Epidemiology 2008;19:209-16.
25. White H. A heteroskedasticity-consistent covariance matrix estimator and a direct test for heteroskedasticity. Econometrika 1980;48:817-38.
26. Wooldridge JM. Econometric analysis of cross section and panel data. Cambridge, MA: MIT Press, 2002.
27. Brunekreef B. Air pollution and life expectancy: is there a relation? Occup Environ Med 1997;54:781-4.
28. Pope CA III. Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? Environ Health Perspect 2000;108:Suppl 4:713-23.