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Fine Particulate Air Pollution and US County Life Expectancies

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Abstract

Background—Exposure to fine particulate matter air pollution has been associated with increased risk of cardiopulmonary and lung cancer morbidity and mortality, suggesting that sustained reductions in pollution exposure should result in improved life expectancy. This study directly evaluates changes in life expectancy associated with differential changes in fine particulate air pollution that occurred in the U.S. during the 1980s and 1990s.

Methods—Life expectancy, socio-economic, and demographic data were compiled for 217 counties in the 51 U.S. metropolitan areas with matching fine particulate air pollution data for the late 1970s/early 1980s and the late 1990s/early 2000s. Regression models were used to estimate the association between reductions in pollution and changes in life expectancy, controlling for changes in socio-economic and demographic variables and for proxy indicators of cigarette smoking.

Results—A decrease of 10 $\mu g/m^3$ of fine particulate concentration was associated with an estimated increase in life expectancy equal to 0.77 (SE = 0.17) years. The estimated effect of reduced pollution exposure on life expectancy was not highly sensitive to controlling for changes in socio-economic, demographic, and proxy smoking variables or to restricting observations to relatively large counties and central metro counties. The effect of reductions in air pollution on life expectancy in the study areas was as much as 18% of the overall increase.

Conclusion—Reducing exposure to ambient fine particulate matter air pollution contributed to significant and measurable improvements in life expectancy in the U.S.

Keywords

life expectancy; air pollution; particulate matter

INTRODUCTION

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 relatively prompt and sustained health benefits from improved air

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quality. Although these studies suggest that reduced air pollution should result in improved life expectancy, a direct evaluation of life expectancy estimates and changes in air pollution has not been reported, especially for whole populations.

This analysis directly explores associations of life expectancy with fine particulate air pollution across 51 U.S. metropolitan areas with matching data for the late 1970s and early 1980s versus the late 1990s and early 2000s. We hypothesize that temporal changes in fine particulate air pollution between 1980 and 2000 were associated with changes in life expectancy. Specifically, we hypothesize that metropolitan areas with the largest declines in fine particulate pollution had relatively larger increases in life expectancy, even after controlling for changes in various socio-economic, demographic, and proxy smoking variables.

METHODS

Data collection and study areas

For the years 1979–1983 the U.S. Environmental Protection Agency (EPA) maintained for research purposes the Inhalable Particle Monitoring Network, which sampled particulate matter in the air using dichotomous samplers with 15-μm and 2.5-μm cut points. Based on these data, mean concentrations of particles with an aerodynamic diameter less than or equal to a 2.5-μm cut-point ($PM_{2.5}$) from 1979–1983 for 61 U.S. metropolitan areas were calculated and used in the re-analysis and extended analyses of the ACS prospective cohort study.[6,7] (Metro-specific means are presented in ACS reanalysis report,[6] Appendix D.) 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 (AIRS) database from 1999 and the first 3 quarters of 2000. Four quarters were averaged when at least 1 of the 2 corresponding quarters for each year had >50% of samples and at least 45 total sampling days. Measurements were averaged first by 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 ACS 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 nation-wide analysis of cross-county mortality disparities, standard life table techniques[21] were used to estimate annual life expectancies for over 2,000 individual or merged county units using individual death records from vital registration and population data from the US censuses, as described in more detail elsewhere.[22] For the purposes of this study, life expectancy for the 221 counties that were part of the 51 metropolitan areas with matching $PM_{2.5}$ data were included. Study metro areas were distributed throughout the U.S. (Figure 1). For each county unit, life expectancies were calculated using pooled death and population data for the 5-year periods 1978–1982 and 1997–2001. Because borough-specific death statistics were unavailable for the five boroughs of New York for the early time period, they were treated as a single unit—resulting in 217 unique county-level observations. As described elsewhere,[22] county-level socio-economic and demographic variables including population, income, and proportions of the population who were high school graduates, self described as white, black, Hispanic, or had urban residencies were collected from U.S. Census data. Income was adjusted for inflation (base year 2000) and cross-county migration data were collected from IRS External Data Product (http://www.irs.gov/pub/irs-soi/prodserv.pdf).

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

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smoking. There were two reasons for using these indirect indicators of smoking: First, smoking prevalence data are not available for most study areas in the late 1970s and early 1980s and second, the lung cancer and COPD measures indicate the population's cumulative exposure to smoking. Lung cancer (ICD 10 code: C33–C34, D02.1–D02.2) and COPD (J40–J44) death rates were calculated using the underlying cause of death in individual death records from vital registration and population data from the US censuses, pooled for the same 5-year periods as life expectancy. Death rates were calculated in 5-year age groups, and age-standardized to the 2000 US population for adults aged 45+ years (death rates from these diseases are unstable before 45 years of age). Additional estimates of changes in cigarette smoking prevalence from health surveys were used as part of sensitivity analyses for a subset of the metropolitan areas with data in both periods. Metropolitan area adult smoking prevalence estimates for 1998–2002 could be estimated for 50 of the metro study areas using the Behavioral Risk Factor Surveillance System (BRFSS) (http://www.cdc.gov/brfss/technical_infodata/surveydata.htm). Metropolitan area adult smoking prevalence estimates for 1978–1980 could be estimated for 24 of the 51 metro study areas using data from the National Health Information Survey (NHIS) (http://www.cdc.gov/nchs/nhis.htm). Based on these data, metro-level changes in smoking prevalence were estimated for 24 metropolitan areas with data in both periods.

Statistical analysis

For both time periods, life expectancies were plotted over $PM_{2.5}$ concentrations and increases in life expectancies between the two time periods were plotted over reductions in $PM_{2.5}$. Cross-sectional regression models were estimated for both time periods and first-difference regression models were estimated regressing increases in life expectancy on reductions in monitored $PM_{2.5}$ concentrations. The sensitivity of the pollution-related effect estimates was explored by 1) including combinations of socio-economic, demographic, and smoking proxy variables in the models; 2) restricting the analysis to only counties with a 1986 population ≥100,000, or to only the 51 largest counties in each metropolitan area; 3) estimating population weighted regression models; 4) to evaluate the influence of baseline pollution levels, stratifying the analysis on 1979–1983 pollution levels; and 5) including direct measures of change in smoking prevalence for the subset of study areas with adequate smoking survey data. Because of the potential for lack of independence between counties in the same metro area, clustered standard errors that were robust to within-cluster correlation[25,26] (clustered by the 51 metro areas) were estimated for all models except those including only the 51 central counties. Models were estimated using PROC REG, and PROC SURVEYREG in SAS (release 9.13; SAS Institute, Inc., Cary, NC).

RESULTS

Table 1 presents summary statistics for study variables. Figures 2 and 3 present cross-sectional life expectancies plotted over air pollution data for the two time periods. At least five observations can be made based on these two figures: 1) $PM_{2.5}$ concentrations generally declined during the 1980s and 1990s. 2) Life expectancies increased between the two periods. 3) For both periods there were cross-sectional associations between life expectancies and pollution. 4) Similar negative associations were observed when analyses were performed using county or metro level observations individually. 5) There was substantial variability or scatter around the regression line indicating that the association with air pollution explains only part of the cross-sectional variability and there are clearly other important factors that influence life expectancy.

Estimates of the associations between $PM_{2.5}$ and life expectancies using cross-sectional regression models were sensitive to inclusion of socio-economic, demographic, and proxy cigarette smoking variables, especially the proportion of high school graduates which was

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highly correlated with per capita income. For example, the association between $PM_{2.5}$ and life expectancy was stronger in the less polluted time period without controlling for any covariates. Based on regression models without any covariates, 10μg/m^3 higher $PM_{2.5}$ concentrations were associated with 1.17 (SE = 0.27, $p < 0.001$) and 2.05 (SE = 0.48, $p < 0.001$) years lower life expectancy for the 1978–1982 and 1997–2001 periods, respectively. However, models that controlled for income, population, cross-county migration, proportion of the population who were black, Hispanic, or had urban residences, and that included proxy variables for smoking found smaller associations, especially in the second period. An increase of 10μg/m^3 in $PM_{2.5}$ concentrations was associated with a 0.62 (SE = 0.22, $p < 0.001$) and 0.53 (SE = 0.24, $p < 0.05$) years lower life expectancy for the 1978–1982 and 1997–2001 periods, respectively.

Figure 4 presents increases in life expectancies plotted over reductions in $PM_{2.5}$ concentrations between approximately 1980 and 2000. Several additional important observations follow from this Figure: 1) On average, life expectancy increased more in areas with larger reductions in air pollution. 2) Similar positive associations between life expectancy gains and reductions in $PM_{2.5}$ concentrations were observed using both county-level and metro-level observations. 3) There was substantial variability or scatter around the regression line, indicating other unaccounted for factors influencing the changes in life expectancy.

Table 2 presents regression coefficients between changes in life expectancy and reductions in $PM_{2.5}$ for models with various combinations of socio-economic, demographic and smoking proxy variables. Models restricted to only counties with a 1986 population ≥100,000, or to only the 51 largest counties in each metropolitan area also are presented. In all models, increased life expectancies were significantly associated with decreases in $PM_{2.5}$. Based on Model 4, a decrease of 10μg/m^3 $PM_{2.5}$ was associated with an adjusted estimated increase in life expectancy equal to 0.77 (SE = 0.17) years. The estimated effect of reduced $PM_{2.5}$ on life expectancy was not highly sensitive to controlling for changes in the socio-economic, demographic, or proxy smoking variables or to restricting observations to only large counties.

In a variety of related sensitivity analyses, the effect estimate for change in $PM_{2.5}$ was quite robust. In step-wise regressions, change in $PM_{2.5}$ was generally the third variable to enter the model, following change in per capita income and change in COPD, and was stable to the inclusion of other variables. When models 4 and 7 in Table 2 were re-estimated using weighted regression (weighting by the square root of the two-period average population), similar results were observed with a decrease of 10μg/m^3 $PM_{2.5}$ associated with an estimated increase in life expectancy equal to 0.66 (SE = 0.18) and 0.87 (SE = 0.24) years, respectively. Stratified estimates of Model 4 in Table 2 were estimated using the 45 counties in the 15 least polluted metro areas for the early period ($PM_{2.5} < 17$μg/m^3, see Figure 2) versus all other more polluted areas. A reduction of tenμg/m^3 $PM_{2.5}$ was associated with a 0.99 (SE = 0.42, $p < 0.05$) and 0.80 (SE = 0.22, $p < 0.01$) years increase in life expectancy for the least polluted versus other areas, respectively, finding no statistically significant differential pollution effects for the initially low polluted versus high polluted areas.

The effect estimate for change in $PM_{2.5}$ was also not highly sensitive to the inclusion of survey-based estimates of metro-level changes in cigarette smoking. For example, when Model 4 in Table 2 was re-estimated using data from the 140 counties in the 24 metro areas with matching smoking prevalence data, a reduction of ten μg/m^3 $PM_{2.5}$ was associated with an estimated increase in life expectancy equal to 0.80 (SE = 0.19, $p < 0.05$) and 0.83 (SE = 0.20, $p < 0.05$) years without and with the inclusion of the change in smoking prevalence variable, respectively. When model 7 in Table 2 was re-estimated using only data from the

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24 largest counties in the 24 metro areas with matching smoking prevalence data, a reduction of ten $\mu g/m^3$ $PM_{2.5}$ was associated with an estimated increase in life expectancy equal to 1.00 (SE = 0.29, $p < 0.05$) and 1.06 (SE = 0.30, $p < 0.05$) years without and with the inclusion of the change in smoking prevalence variable, respectively. When added to these models, change in smoking prevalence was not statistically significant ($p > 0.15$) and the estimated effect of a change in COPD death rate was largely unaffected. These results indicate that county-level changes in COPD were more robustly associated with county-level changes in life expectancy than metro-level estimates of changes in 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 study areas even after controlling for various socio-economic, demographic, and proxy smoking variables that are associated with health through a range of mechanisms. Indirect calculations have found approximately 0.7 to 1.6 years loss of life expectancy attributable to 10 $\mu g/m^3$ long-term exposure to fine particulate air pollution using life tables from the Netherlands and from the U.S. and risk estimates from the prospective cohort studies.[27,28] In the present analysis, a decrease of 10 $\mu g/m^3$ of fine particulate concentration was associated with an estimated increase in life expectancy equal to approximately 0.77 (SE = 0.17) years—an estimate consistent with these indirect estimates.

For the time period of approximately 1980–2000, the average increase in life expectancy was 2.74 years for the counties in this analysis. Improved air pollution, was only one contributing factor to increased life expectancies, with overlap between effects of other factors. Based on average reductions in $PM_{2.5}$ concentrations (6.56 $\mu g/m^3$) in the metro areas of 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 0.5 years (6.56 × 0.077). Multi-causality and competing risk issues make it difficult to quantify changes in life expectancy attributable to singular risk factors, but these results suggest that the individual effect of reductions in air pollution on life expectancy was as much as 18% of the overall increase. In metropolitan areas where reductions in $PM_{2.5}$ were 13–14 $\mu g/m^3$, the contribution of improvements in air quality to increases in life expectancy may have been as much as one year (13.5 × 0.077).

Previous cross-sectional analyses have observed associations between mortality rates and particulate air pollution,[1–3] but the size of these associations were sensitive to efforts to control for potential confounders. Similar sensitivity was observed for the strictly cross-sectional associations observed with life expectancy in this analysis. The primary strength of this analysis, however, is the additional use of temporal variability. Differential temporal changes in pollution exposures across metropolitan areas between 1980 and 2000 provide a natural experiment-like opportunity. Cross-sectional characteristics that did not change over time are controlled for 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 differential temporal changes, the estimated effects of reduced $PM_{2.5}$ exposure on increased life expectancy were robust to control of socio-economic, demographic, and proxy smoking variables, and to analysis restricted to observations of only large or central counties.

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From an analytical perspective, it would have been informative if pollution had actually increased in some of the cleaner areas. However, no metro area experienced increased pollution and the more polluted areas had greater potential to reduce pollution than the cleaner ones. Stratified analyses found no statistically significant differential pollution effects for the initially low polluted versus high polluted areas, consistent with previous findings of $PM_{2.5}$ effects 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 with life expectancy and air pollution using available monitored $PM_{2.5}$ data for both the early and late time periods. However, limited monitoring of $PM_{2.5}$ air pollution data, especially for the period 1979–1983, reduces the number of metro areas that could be included in the analysis and our ability to evaluate associations with more spatial and temporal resolution. Furthermore, as a population-based analysis, the ability to control for additional potential confounders, especially various individual and community risk factors that may have policy drivers in common with environmental regulation, is limited. For example, the three variables in this analysis most strongly associated with changes in life expectancy, are all proxy variables. Changes in per capita income likely serves a proxy variable for, or is highly correlated with, access to medical care, higher quality diets, healthier lifestyles, etc. The use of lung cancer and COPD mortality rates as proxy variables is necessitated by lack of reliable smoking data, especially for the early period, yet they reflect the cumulative effects of smoking which may similarly affect life expectancy. Although the large majority of deaths due to 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 co-pollutants and changes in $PM_{2.5}$ represent estimates of changes in area-wide ambient concentrations based on fixed site monitoring during the two time periods, versus a direct measure of personal exposures. Nevertheless, U.S. air quality standards and related public policies are designed to restrict ambient pollution concentrations in an effort to protect human health.[20] Previous prospective-cohort studies, using measures of ambient concentrations and controlling for smoking and other individual risk factors, suggest similar improvements in survival and life expectancy, using indirect estimates.[4–11] The fact that this population-based analysis observes comparable improvements in life expectancy associated with public policy related reductions in ambient pollution concentrations provides interesting and valuable corroboration.

In conclusion, the findings of this analysis are generally good news. Although there are multiple factors affecting life expectancies, these results provide evidence that improvements in air quality have contributed to measurable improvements in human health and life expectancy in the United States.

Acknowledgments

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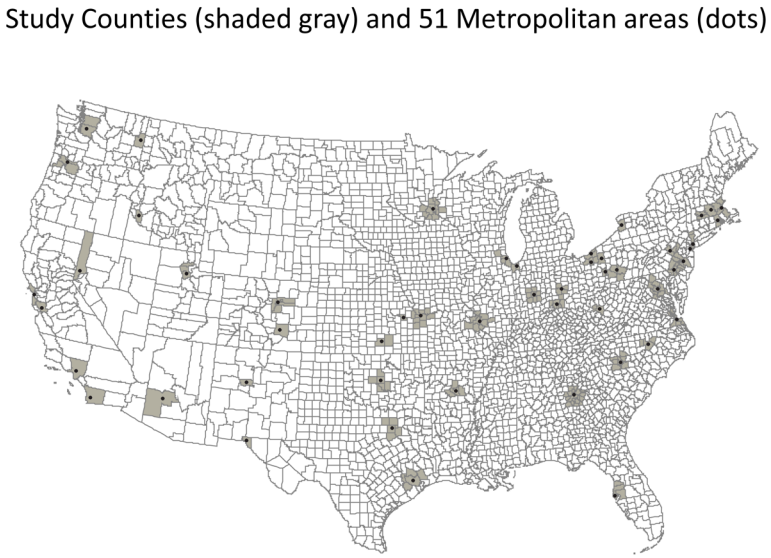

Figure 1.

Location of study areas. Study counties are shaded gray and dots represent approximate location of the following 51 number-coded metropolitan areas: 1. Akron, OH; 2. Albuquerque, NM; 3. Allentown, NY; 4. Atlanta, GA; 5. Boise, ID; 6. Boston, MA; 7. Buffalo, NY; 8. Charlotte, NC; 9. Charlotte, SC; 10. Chicago, IL; 11. Cincinnati, OH; 12. Cleveland, OH; 13. Dallas, TX; 14. Dayton, OH; 15. Denver, CO; 16. El Paso, TX; 17. Gary, IN; 18. Houston, TX; 19. Indianapolis, IN; 20. Jersey City, NJ; 21. Kansas City, MO; 22. Little Rock, AR; 23. Los Angeles, CA; 24. Minneapolis, MN; 25. New York, NY; 26. Norfolk, VA; 27. Oklahoma City, OK; 28. Philadelphia, PA; 29. Phoenix, AZ; 30. Pittsburg, PA; 31. Portland, OR; 32. Providence, RI; 33. Pueblo, CO; 34. Raleigh, NC; 35. Reno, NV; 36. St. Louis, MO; 37. San Diego, CA; 38. San Francisco, CA; 39. Salt Lake City, UT; 40. San Jose, CA; 41. Seattle, WA; 42. Spokane, WA; 43. Springfield, MA; 44. Steubenville, OH; 45. Tampa, FL; 46. Topeka, KS; 47. Washington, DC; 48. Wichita, KS; 49. Wilmington, DE; 50. Worcester, MA; 51. Youngstown, OH.

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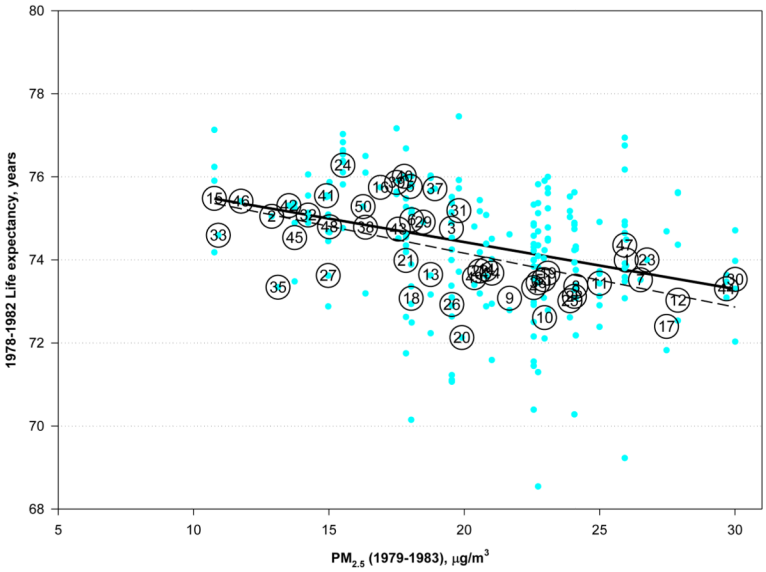

Figure 2.

Cross-sectional life expectancies for 1978–1982 plotted over 1979–1983 $PM_{2.5}$ concentrations. Dots and number-labeled circles represent county level and metro-level population-weighted mean life expectancies, respectively. Metropolitan area locations and number codes are presented in Figure 1. Solid and broken lines represent regression lines using county- and metro-level observations, respectively.

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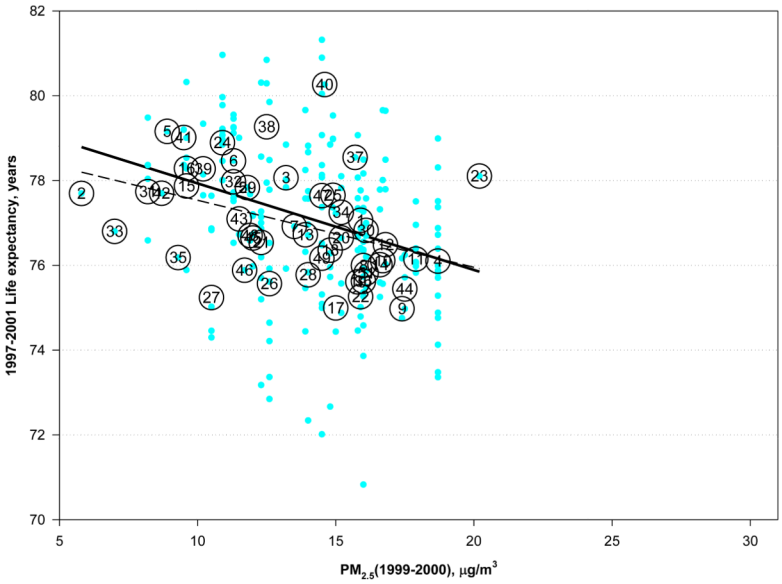

Figure 3.

Cross-sectional life expectancies for 1997–2001 plotted over 1999–2000 $PM_{2.5}$ concentrations. Dots and number-labeled circles represent county level and metro-level and population-weighted mean life expectancies, respectively. Metropolitan area locations and number codes are presented in Figure 1. Solid and broken lines represent regression lines using county- and metro-level observations, respectively.

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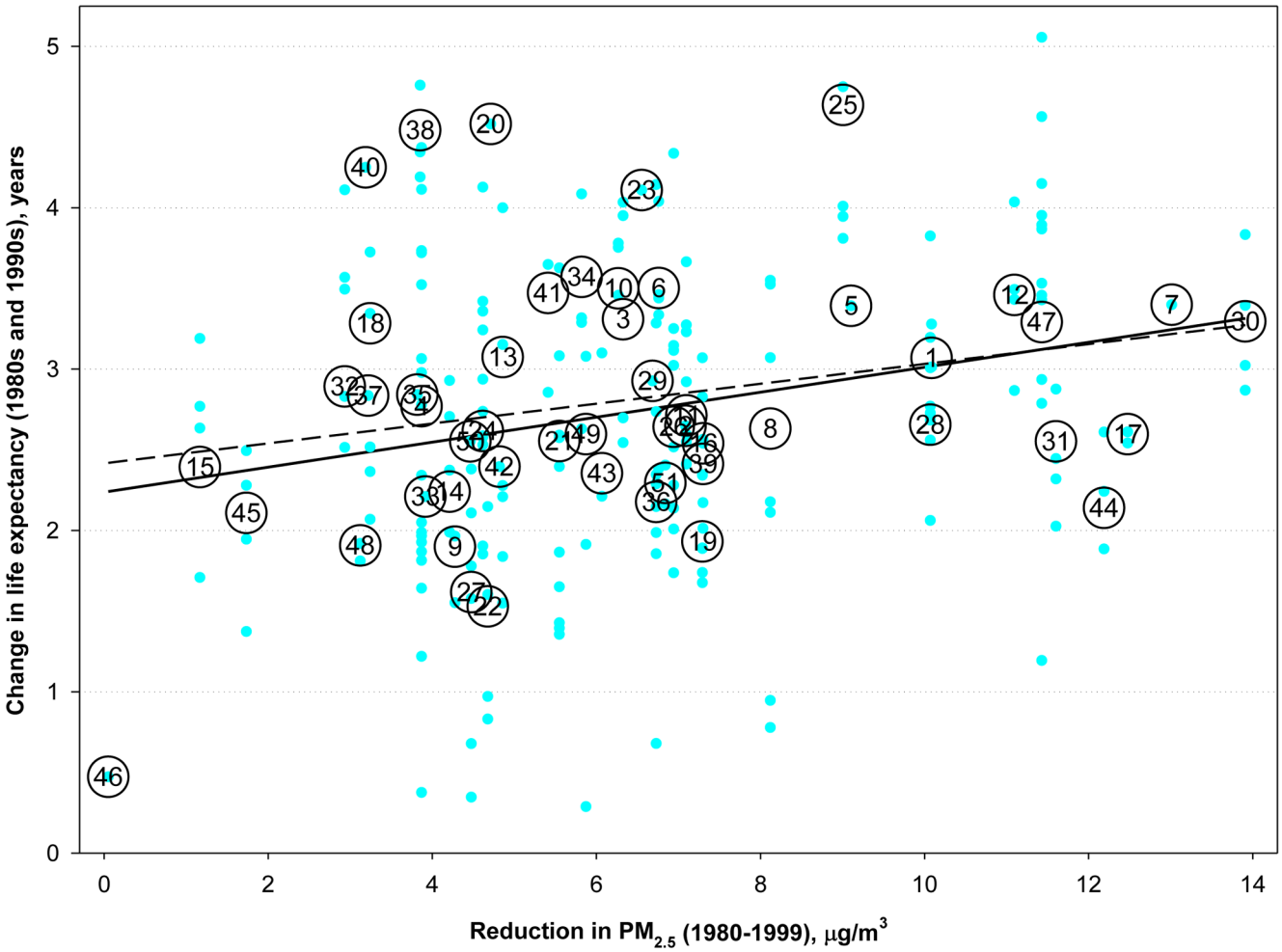

Figure 4.

Changes in life expectancies plotted over reductions in $PM_{2.5}$ concentrations. Dots and number-labeled circles represent county level and metro-level and population-weighted mean life expectancies, respectively. Metropolitan area locations and number codes are presented in Figure 1. Solid and broken lines represent regression lines using county- and metro-level observations, respectively.

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Table 1

Summary statistics of key study variables in the 217 analysis counties

Variable	Unit	Mean	SD
Life expectancy (1978–1982)	years	74.34	1.52
Life expectancy (1997–2001)	years	77.08	1.82
Δ Life expectancy	years	2.74	0.93
$PM_{2.5}$ (1979–1983)	$\mu g/m^3$	20.64	4.40
$PM_{2.5}$ (1999–2000)	$\mu g/m^3$	14.07	2.84
Δ $PM_{2.5}$	$\mu g/m^3$	6.56	2.98
Per capita income (1979)	1000 USD	15.24	2.70
Per capita income (1999)	1000 USD	23.79	5.10
Δ Income	1000 USD	8.56	3.15
Population (1980)	100,000	3.79	8.37
Population (2000)	100,000	4.80	10.02
Δ Population	100,000	1.00	2.24
Cross-county migration (1980)	proportion	0.26	0.11
Cross-county migration (2000)	proportion	0.25	0.09
Δ Migration	proportion	−0.01	0.06
Urban residency (1980)	proportion	0.58	0.33
Urban residency (2000)	proportion	0.78	0.21
Δ Urban	proportion	0.20	0.18
H.S. Graduate (1980)	proportion	0.68	0.11
H.S. Graduate (2000)	proportion	0.87	0.05
Δ H.S. Grad.	proportion	0.19	0.15
Black population (1980)	proportion	0.097	0.117
Black population (2000)	proportion	0.115	0.129
Δ Black	proportion	0.018	0.057
Hispanic population (1980)	proportion	0.037	0.076
Hispanic population (2000)	proportion	0.069	0.093
Δ Hispanic	proportion	0.032	0.047
Lung cancer mortality rates (1979–1983)	per 10,000	14.40	3.15
Lung cancer mortality rates (1997–2001)	per 10,000	16.82	3.51
Δ Lung cancer	per 10,000	2.43	3.60
COPD mortality rates (1979–1983)	per 10,000	7.88	1.93
COPD mortality rates (1997–2001	per 10,000	12.38	2.73
Δ COPD	per 10,000	4.50	2.45

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Table 2

Results of selected regression models including estimates of change in life expectancy (and standard errors) associated with a 10 $\mu g/m^3$ reduction in $PM_{2.5}$ controlling for different combinations of socio-economic, demographic and smoking variables

Var.	Model 1	Model 2	Model 3	Model 4	Model 5[a]	Model 6[b]	Model 7[b]
Int.	2.25 (0.21)*	0.77 (0.18)*	1.37 (0.28)*	1.38 (0.25)*	1.82 (0.31)*	1.71 (0.51)*	2.03 (0.35)*
Δ $PM_{2.5}$ (x10)	0.75 (0.29)*	0.85 (0.19)*	0.76 (0.18)*	0.77 (0.17)*	0.62 (0.21)*	1.01 (0.25)*	0.93 (0.23)*
Δ Income (x1000)	---	0.17 (0.02)*	0.14 (0.02)*	0.14 (0.01)*	0.12 (0.01)*	0.15 (0.04)*	0.11 (0.02)*
Δ Population (x100,000)	---	0.08 (0.02)*	0.07 (0.02)*	0.08 (0.02)*	0.07 (0.02)*	0.04 (0.02)	0.05 (0.02)*
Δ Migration	---	−0.02 (0.81)	0.46 (0.88)	---	---	−0.02 (1.83)	---
Δ H.S. Graduation	---	0.20 (0.56)	0.02 (0.53)	---	---	−0.90 (0.86)	---
Δ Urban	---	−0.66 (0.32)*	−0.29 (0.26)	---	---	0.03 (1.88)	---
Δ Black	---	−1.91 (0.56)*	−2.18 (0.44)*	−2.13 (0.40)*	−2.63 (0.61)*	−5.06 (2.12)*	−5.82 (1.98)*
Δ Hispanic	---	1.16 (1.03)	1.00 (0.98)	---	---	2.44 (2.22)	---
Δ Lung Cancer	---	---	−0.02 (0.02)	---	---	0.01 (0.03)	---
Δ COPD	---	---	−0.07 (0.02)*	−0.09 (0.02)*	−0.12 (0.03)*	−0.15 (0.06)*	−0.16 (0.04)*
N	217	217	217	217	129	51	51
R^2	0.06	0.48	0.52	0.50	0.58	0.76	0.73

[a] Includes only counties with ≥100,000 1986 population.

[b] Includes only counties with largest 1986 population in statistical metro area.

* Indicates statistical significance ($p < 0.05$).