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Chemical Composition of Fine Particulate Matter and Life Expectancy:

In 95 US Counties Between 2002 and 2007

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Abstract

Background—In a previous study, we provided evidence that a decline in fine particulate matter (PM_{2.5}) air pollution during the period between 2000 and 2007 was associated with increased life expectancy in 545 counties in the United States. In this article, we investigated which chemical constituents of PM_{2.5} were the main drivers of the observed association.

Methods—We estimated associations between temporal changes in seven major components of PM_{2.5} (ammonium, sulfate, nitrate, elemental carbon matter, organic carbon matter, sodium, and silicon) and temporal changes in life expectancy in 95 counties between 2002 and 2007. We included US counties that had adequate chemical components of PM_{2.5} mass data across all seasons. We fitted single pollutant and multiple pollutant linear models, controlling for available socioeconomic, demographic, and smoking variables and stratifying by urban and nonurban counties.

Results—In multiple pollutant models, we found that: (1) a reduction in sulfate was associated with an increase in life expectancy; and (2) reductions in ammonium and sodium ion were associated with increases in life expectancy in nonurban counties only.

Conclusions—Our findings suggest that recent reductions in long-term exposure to sulfate, ammonium, and sodium ion between 2002 and 2007 are associated with improved public health.

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Since the mid-1990s, air quality across metropolitan areas in the United States has improved substantially. Much of this progress has been driven by the Clean Air Act of 1970.¹ In 2009, Pope et al.² compiled data on life expectancy, socioeconomic status, and demographic characteristics for 211 counties in the 51 US metropolitan areas with matching data on fine particulate air pollution (PM_{2.5}) 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. These authors found that a reduction of 10 µg/m³ of PM_{2.5} over this period was associated with an increase of 0.61 ± 0.2 years in life expectancy.

Correia et al.³ extended the Pope et al.² analysis using data for the period between 2000 and 2007 and for 545 US counties. Using the same statistical methods as Pope et al.,² Correia et al.³ investigated whether more recent and smaller reductions in PM_{2.5} continued to be associated with increases in life expectancy. They found that a reduction of 10 µg/m³ of PM_{2.5} over the period between 2000 and 2007 was associated with an increase of 0.35 ± 0.16 years in life expectancy.

Although these previous findings provide evidence that past and more recent declines in ambient levels of PM_{2.5} are associated with an increased in life expectancy, they did not identify which PM_{2.5} components are most responsible for the observed associations. To address this gap in knowledge, we extended the previous study by Correia et al.³ on the association between county-specific changes in PM_{2.5} total mass (between 2000 and 2007) and county-specific change in life expectancy (between 2007 and 2000) to the chemical components of PM_{2.5}. Specifically, we employed the same analytic methods used by Pope et al.² and Correia et al.³ to estimate the association between county-specific temporal changes (between 2002 and 2007) of the major PM_{2.5} components and county-specific temporal changes (between 2007 and 2002) in life expectancy. We estimated these associations for all counties combined and separately for the urban and nonurban counties.

METHODS

Data Source

We used county-level data, which includes life expectancy, PM_{2.5}, and potential county-specific confounders, from our previous studies.^{2,3} We restricted this analysis to the 95 counties that had at least six daily observations for each season in the years 2002 and 2007 for PM_{2.5} mass and for each of its seven chemical components. These 95 counties are located in 75 Metropolitan Statistical Area (MSA) areas and were all included in Correia et al.³ Because of the small number of observed data on the chemical components in the years 2000 and 2001, we restricted our analysis to the period between 2002 and 2007 rather than between 2000 and 2007 as in Correia et al.³

Outcome

Our primary outcome was the county-specific change in life expectancy, calculated as the difference between the values in 2007 and the values in 2002. County and yearly specific

life expectancies were calculated by fitting a mixed effects spatial Poisson model to the National Center for Health Statistics data and US Census population data. From this model, we obtained robust estimates of the number of deaths in each county (by age, race groups, and year) and then calculated life expectancies for each county. This method was also used in our previous analysis.^{3,4} Detailed explanation of the county-level life expectancy calculation is included in Section A in the Online Supplementary Appendix (<http://links.lww.com/EDE/A907>).

PM_{2.5} Total Mass, Components, and Potential Confounders

PM_{2.5} total mass data and the chemical components of PM_{2.5} mass were extracted from Air Explorer provided by the US Environmental Protection Agency.⁵ We calculated the county-level PM_{2.5} measurements by first averaging daily measurements across monitors within counties, then averaging across days to calculate yearly averages.⁶ The seven chemical components of PM_{2.5}, including sulfate, nitrate, ammonium, elemental carbon, organic carbon, silicon, and sodium ion, were chosen based on the findings of Bell et al.,⁷ as they are the only components that each contributed greater than 1% of PM_{2.5} mass for either seasonal or yearly averages, and in total make up at least 79–85% of PM_{2.5} mass. The county-specific changes in PM_{2.5} mass and its components were defined as the differences between 2002 and 2007 annual values. The unit of these changes is $\mu\text{g}/\text{m}^3$.

For each county, we also obtained county-specific demographics, socioeconomic, population, and smoking information as they could be potential confounders. In particular, we calculated differences between 2007 and 2002 of the following county-specific variables: total population, proportion of population that was black, proportion of population that was Hispanic, proportion of population that graduated from high school, all were compiled from US Census and American Community Survey data, and per capita income that was compiled from data available through the Bureau of Economic Analysis.⁸ The income data was standardized using the Consumer Price Index with 2000 as the base year as provided by the US Bureau of Labor Statistics.⁹ To adjust for smoking, we used age-standardized lung cancer and chronic obstructive pulmonary disease (COPD) death rates as proxy variables for smoking prevalence. These death rates were calculated according to World Health Organization guidelines for age-standardized death rates, using 2000 and 2005 National Center for Health Statistics death rates.^{3,10} In addition, we obtained county-specific current smoking rates from the Center for Disease Control's Behavioral Risk Factor Surveillance System (BRFSS). Among 95 counties, 87 had current smoking information based on the BRFSS data. Using the urbanicity index,¹¹ we categorized 95 counties into "Urban" counties ($n = 52$) that have more than 90% of population in urban and "nonurban" counties ($n = 43$), if otherwise.³

Statistical Analysis

We fitted linear regression models to estimate the association between temporal changes in each of the seven chemical components of PM_{2.5} (between 2002 and 2007) and changes in life expectancy (between 2007 and 2002). In regression models, we included temporal changes in socioeconomic and demographic variables and in prevalence of cigarette smoking. We fitted these regression models to all 95 counties combined and for the urban

and nonurban counties, separately. Standard errors of the estimated regression coefficients were adjusted for the clustering of the counties within MSAs ($n = 75$).² The estimated regression coefficients were scaled to represent the association between change in life expectancy and one interquartile range (IQR) change in the corresponding chemical component.

We fitted three regression models: (1) unadjusted single pollutant models that included only one dependent variable, defined as the temporal change in one of the seven chemical components at the time; (2) adjusted single pollutant models, same as model 1 but adjusted for the changes in demographics, socioeconomic, population characteristics, lung cancer death rates, and COPD death rates; and (3) adjusted multiple pollutant models, same as model 2, but adjusted for the temporal changes in all seven components. As a sensitivity analysis, we also fitted models that adjust for county-specific changes in current smoking rates in addition to the changes in lung cancer and COPD death rates. This sensitivity analysis was conducted for the 87 counties with BRFSS's current smoking data available. All analyses were conducted using SAS version 9.3 (SAS Institute Inc, Cary, NC).

RESULTS

Study Sample

In 2007, the population from the 95 counties was approximately 70 million, 23% of total population in the United States. Figure S1 in the eAppendix (<http://links.lww.com/EDE/A907>) shows the distributions of the county-specific averages of PM_{2.5} total mass, across counties by season, and separately for the years 2002 and 2007.

Change in Life Expectancy

Figure 1 plots county-specific values of life expectancy in 2007 (y axis) versus county-specific values of life expectancy in 2002 (x axis). Approximately 95% of the 95 counties had an increase in life expectancy during this period. This increase was more pronounced in urban than nonurban counties.

Changes in PM_{2.5} Mass, Components, and Potential Confounders

Table 1 reports the summary characteristics of 95 counties, overall and by urban and nonurban counties. Table S1 in the eAppendix (<http://links.lww.com/EDE/A907>) summarizes the mean, median, standard deviation, and IQR across the 95 counties of the difference between county-specific yearly averages in 2002 minus the county-specific yearly averages in 2007 of PM_{2.5} and its components.

Figure 2 shows scatter plots of county-specific averages of PM_{2.5} total mass and its seven components in 2002 (x axis) versus 2007 (y axis). Between 2002 and 2007, we observed notable declines in PM_{2.5} total mass and its seven components, except elemental carbon, which on average across counties has increased. These declines varied by regions. The West region had the largest declines in PM_{2.5} total mass (1.00 $\mu\text{g}/\text{m}^3$), nitrate (0.50 $\mu\text{g}/\text{m}^3$), ammonium (0.22 $\mu\text{g}/\text{m}^3$), organic carbon (1.61 $\mu\text{g}/\text{m}^3$), and sodium ion (0.14 $\mu\text{g}/\text{m}^3$), the Midwest region had the largest decline in sulfate (0.32 $\mu\text{g}/\text{m}^3$), and the Northeast region had

the largest decline in silicon ($0.03 \mu\text{g}/\text{m}^3$) and the smallest increase in elemental carbon ($0.13 \mu\text{g}/\text{m}^3$). In anticipation of multiple pollutant analysis, Table 2 summarizes correlations between the changes (between 2002 and 2007) for each pair of chemical components across all 95 counties. Changes in ammonium and nitrate are the most highly correlated ($R = 0.81$).

Association Between Changes in $\text{PM}_{2.5}$ Components and Changes in Life Expectancy

Figure S2 in the eAppendix (<http://links.lww.com/EDE/A907>) shows scatter plots of county-specific changes in $\text{PM}_{2.5}$ mass and its seven chemical components (between 2002 and 2007, x axis) versus county-specific changes in life expectancy (between 2007 and 2002, y axis). Figure 3A–C shows the point estimates and 95% confidence intervals (CIs) of the association between temporal changes in life expectancy and temporal changes in each of the seven components of $\text{PM}_{2.5}$ and $\text{PM}_{2.5}$ total mass across all counties combined and by urban and nonurban counties. These estimates were based on the unadjusted single pollutant model (Figure 3A), adjusted single pollutant (Figure 3B), and adjusted multiple pollutant models (Figure 3C). Regression models for Figure 3B, C included changes in per capita income, total population, proportion of population that was black, proportion of population that was Hispanic, proportion of population that graduated from high school, age-standardized death rates for lung cancer, and age-standardized death rates for COPD. Estimates and corresponding 95% CIs of all the regression coefficients from regression models are listed in Tables S2–S4 in the eAppendix (<http://links.lww.com/EDE/A907>). Figure S3A–C in the eAppendix (<http://links.lww.com/EDE/A907>) summarizes the results of the sensitivity analysis for the unadjusted single, adjusted single, and multiple pollutant models that adjusted for the change in current smoking rates obtained from BRFSS. Only 87 counties that had current smoking information from BRFSS were included in the sensitivity analysis.

$\text{PM}_{2.5}$ Mass

Under the single adjusted pollutant model (Figure 3B), we did not find evidence that a decrease of an IQR ($2.20 \mu\text{g}/\text{m}^3$) of $\text{PM}_{2.5}$ mass was associated with an increase in life expectancy.

Sulfate

Under a single adjusted pollutant model (Figure 3B), we found that a decrease of an IQR ($0.32 \mu\text{g}/\text{m}^3$) of sulfate was weakly associated with an increase in life expectancy. This association became stronger under a multiple adjusted pollutant model (0.12 years [0.02 to 0.22]; Figure 3C). This association is stronger in nonurban counties compared with urban counties.

Ammonium

Under a multiple pollutant model, we found that a decrease of an IQR of ammonium was associated with an increase in life expectancy in the 43 nonurban counties (0.14 years [95% CI = 0.01, 0.27]; Figure 3C).

Sodium Ion

Under a multiple adjusted pollutant model (Figure 3C), we found that a decrease of an IQR ($0.04 \mu\text{g}/\text{m}^3$) of sodium ion was associated with an increase in life expectancy in the 43 nonurban counties (0.10 years [95% CI = 0.06, 0.14]).

We did not find evidence of an association between temporal changes in the chemical components and temporal changes in life expectancy for any of these chemical components: elemental carbon, nitrate, organic carbon, or silicon.

The sensitivity analysis shows that all these results, except ammonium, were robust to the adjustment for the temporal change in current smoking rates between 2002 and 2007 (Figure S3A–C in the eAppendix; <http://links.lww.com/EDE/A907>). Specifically, we found that the association between a reduction in ammonium and an increase in life expectancy for nonurban counties was lost when adjusted for smoking rates. However, this might be due to the smaller number of nonurban counties included in the sensitivity analysis ($n = 35$) compared with the already small number of nonurban counties included in the main analysis ($n = 43$). We also found that the change in current smoking rates was not associated with the change in life expectancy under either a single adjusted pollutant model or a multiple adjusted pollutant model.

DISCUSSION

This study estimates long-term effects of $\text{PM}_{2.5}$ chemical components on life expectancy in both urban and nonurban counties. We found that a decrease in sulfate was associated with increases in life expectancy in all 95 counties and decreases in ammonium and sodium ion were associated with increases in life expectancy in the nonurban counties. Our findings were based on a multiple pollutant model in which each individual pollutant also was adjusted for the other six constituents.

The identification of which chemical components of particulate air pollution are primarily responsible for various observed adverse health effects are complicated and clearly not fully resolved. Overall the literature seems to suggest that various complex mixtures of fine particles including metals, elemental and organic carbon, ammonium, sulfate, nitrate, and related pollutants can contribute to adverse health effects.^{7,12–24}

Our result on the adverse effects of sulfate on life expectancy is consistent with the results by Lepeule et al.,²⁵ Pope et al.,²⁶ and Kravchenko et al.²⁷ These authors also found that a decrease in sulfate leads to a decrease in mortality.

Restricted by available chemical components of $\text{PM}_{2.5}$ data, we did not find that decreases in elemental carbon, nitrate, silicon, and organic carbon were associated with an increase in life expectancy. However, recent time series studies have reported that these components might be associated with mortality. For example, in a recent study of acute health effects associated with short-term exposure to chemical components of $\text{PM}_{2.5}$, Krall et al.²² fitted single pollutant models to multiple site time series data and estimated short-term associations between nonaccidental mortality and $\text{PM}_{2.5}$ components across 72 urban

counties from 2000 to 2005. They found that daily changes in silicon, organic carbon, and elemental carbon were associated with daily change in mortality. Dai et al.²⁸ fitted a city-season-specific Poisson regression model to estimate PM_{2.5} effects on approximately 4.5 million deaths for all-cause, cardiovascular diseases, respiratory diseases, myocardial infarction, and stroke in 75 US cities from 2000 to 2006. They found that silicon, calcium, and sulfur were associated with all-cause mortality and sulfur was also related to more respiratory deaths. Our analysis focused on long-term exposure to chemical components and mortality, whereas all the time series analyses focused on short-term exposure.

Epidemiologic studies of multiple pollutants could report findings that are unexpected and hard to interpret because of the many methodological challenges.²⁹ These challenges are: (1) the correlation among pollutants could hamper the possibility of isolating the effect of one pollutant from the others; (2) unmeasured confounding and/or the misspecification of the statistical model could bias the results; (3) exposure measurement error, also considering that the different components have different degree of spatial variability.^{30,31}

Our study has limitations. First, we only had data on 95 counties and 75 MSAs, a much smaller number than the 545 counties included in Correia et al.³ This is mainly due to the fact that the number of monitors that measure the components of PM_{2.5} is much smaller than the number of monitors that measure PM_{2.5} total mass. In 2007, among 259 monitors that measure PM_{2.5} mass across 95 counties, only 107 (41.3%) were available for one or more of the seven components of PM_{2.5} mass.

Second, although our statistical models included many potential confounders such as temporal differences in socioeconomic, demographic, and smoking variables, we recognize that it is plausible that temporal changes in lifestyles over the past decade could have been affecting life expectancy. These changes, including the decrease in the size of the smoking population,³² the decline in the consumption of red meat,³³ and the improvement in diabetic control,³⁴ could all contribute to increase in life expectancy. Moreover, the improvements in treatments, quality of care, access to care, decline in hospitalizations in acute cardiovascular disease nationwide can also result to increase in life expectancy.^{35–38} However, all these factors are likely to confound the reported associations only if: (1) their temporal changes within each county are also correlated with the temporal changes in the PM_{2.5} components within the same county, and (2) their county-specific temporal changes are not captured by the county-specific measured confounders that were included in the statistical model.

Third, in this analysis, we regressed temporal differences in exposure to air pollution versus temporal differences in life expectancy across counties, and therefore county-specific factors that are correlated with both exposure to air pollution and life expectancy but that do not vary temporally are not confounders.

Another potential limitation is that exposure measurement error for the chemical components could be larger in nonurban counties than urban counties. This could be due to two reasons. First, nonurban counties usually are larger than urban counties, and yet the number of monitors in non-urban counties is smaller than the number of monitors in urban counties. In our study, the numbers of monitors for PM_{2.5} mass were 79 and 180 in

nonurban and urban counties, respectively. Second, the between-monitor correlation among chemical components is likely to decrease as the distance between monitors increases and these correlations are different across the chemical components. Bell et al.³⁰ studied the correlations of daily pollutant's data for monitor pairs for each of the seven chemical components and found that correlations decline with increasing distance between two monitors. Correlations of ammonium, nitrate, and sulfate seem less sensitive to the change in distance than the correlation of elemental carbon.³⁰

Finally, we only focused on seven components that contribute more than 1% to the overall PM_{2.5} total mass.⁷ Our analysis did not include components that constitute less than 1% of PM_{2.5} total mass but that are harmful.

Despite these limitations, our findings provide evidence that county-specific reductions in long-term exposure to sulfate emissions, which are usually generated from automobile traffic, power generation, industry and agriculture, were associated with an increase in life expectancy. Also we found that county-specific reductions in ammonium emissions, which are more common in nonurban areas and usually generated by the use of fertilizers and waste disposal sites, and county-specific reductions in sodium ion emissions, which are usually generated from industry and agriculture (e.g., treatment of biological wastewater), were associated with an increase in life expectancy in the non-urban counties. Identifying these drivers could potentially allow for more targeted air quality regulation and further inform understanding of the mechanisms by which fine-particulate air pollution affects public health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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References

1. Samet JM. The clean air act and health—a clearer view from 2011. *New Engl J Med*. 2011; 365:198–201. [PubMed: 21732828]
2. Pope CA, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. *New E J Med*. 2009; 360:376–386.
3. Correia AW, Pope CA III, Dockery DW, Wang Y, Ezzati M, Dominici F. Effect of air pollution control on life expectancy in the United States: an analysis of 545 U.S. counties for the period from 2000 to 2007. *Epidemiology*. 2013; 24:23–31. [PubMed: 23211349]
4. Kulkarni SC, Levin-Rector A, Ezzati M, Murray CJ. Falling behind: life expectancy in US counties from 2000 to 2007 in an international context. *Popul Health Metr*. 2011; 9:1–12. [PubMed: 21219615]
5. US EPA. AirData website download data page. Available at: <http://www.epa.gov/airdata/addata.html>. Accessed October 17, 2013
6. Peng, RD.; Dominici, F. *Statistical Methods for Environmental Epidemiology in R: A Case Study in Air Pollution and Health*. New York, NY: Springer; 2008.

7. Bell ML, Dominici F, Ebisu K, Zeger SL, Samet JM. Spatial and temporal variation in PM_{2.5} chemical composition in the United States for health effects studies. *Environ Health Perspect.* 2007; 115:989–995. [PubMed: 17637911]
8. US Department of Commerce B. E. A. Bureau of economic analysis. Available at: <http://www.bea.gov/regional/downloadzip.cfm>. Accessed October 17, 2013
9. US Department of Labor. Consumer price index research series using current methods. 2011. Available at: <http://www.bls.gov/cpi/cpirsdc.htm>. Accessed January 10, 2013
10. Ahmad, OB.; Boschi-Pinto, C.; Lopez, AD., et al. Age standardization of rates: a new WHO standard. World Health Organization; Geneva: Available at: <http://www.who.int/healthinfo/paper31.pdf>. Accessed October 17, 2013
11. Allen, W. PhD thesis, Dissertation. The University of Texas; Austin: 1976. The conceptualization and measurement of urbanization.
12. Janes H, Dominici F, Zeger SL. Trends in particulate matter and mortality in 113 U.S. counties, 2000–2002: evidence on the long term effects of air pollution. *Epidemiology.* 2007; 18:416–423. (with discussion). [PubMed: 17568215]
13. Dominici F, Peng D, Bell M, et al. Fine particulates air pollution and hospital admission for cardiovascular and respiratory diseases. *J Am Med Assoc.* 2006; 295:1127–1135.
14. Peng R, Chang H, Bell M, et al. Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among medicare patients. *J Am Med Assoc.* 2008; 299:2172–2179.
15. Zeger SL, Dominici F, McDermott A, Samet JM. Mortality in the medicare population and chronic exposure to fine particulate matter. *Environ Health Perspect.* 2008; 116:1614–1619. [PubMed: 19079710]
16. Bell M, Ebisu K, Peng R, Samet JM, Dominici F. Hospital admissions and chemical composition of fine particle air pollution. *Am J Respir Crit Care Med.* 2009; 179:1–6. [PubMed: 19098155]
17. Dockery DW. Health effects of particulate air pollution. *Ann Epidemiol.* 2009; 19:257–263. [PubMed: 19344865]
18. Dominici F, Peng RD, Barr CD, Bell ML. Protecting human health from air pollution: shifting from a single-pollutant to a multipollutant approach. *Epidemiology.* 2010; 21:187–194. [PubMed: 20160561]
19. Pope CA 3rd, Ezzati M, Dockery DW. Fine particulate air pollution and life expectancies in the United States: the role of influential observations. *J Air Waste Manag Assoc.* 2013; 63:129–132. [PubMed: 23472296]
20. Ito K, Mathes R, Ross Z, Nádas A, Thurston G, Matte T. Fine particulate matter constituents associated with cardiovascular hospitalizations and mortality in New York City. *Environ Health Perspect.* 2011; 119:467–473. [PubMed: 21463978]
21. Ostro BD, Feng WY, Broadwin R, Malig BJ, Green RS, Lipsett MJ. The impact of components of fine particulate matter on cardiovascular mortality in susceptible subpopulations. *Occup Environ Med.* 2008; 65:750–756. [PubMed: 18417555]
22. Krall JR, Anderson GB, Dominici F, Bell ML, Peng RD. Short-term exposure to particulate matter constituents and mortality in a national study of U.S. urban communities. *Environ Health Perspect.* 2013; 121:1148–1153. [PubMed: 23912641]
23. Peng RD, Bell ML, Geyh AS, et al. Emergency admissions for cardiovascular and respiratory diseases and the chemical composition of fine particle air pollution. *Environ Health Perspect.* 2009; 117:957–963. [PubMed: 19590690]
24. Bell M, Ebisu K, Peng R, Samet J, Dominici F. Hospital admissions and chemical composition of fine particulate air pollution. *Am J Respir Crit Care Med.* 2009; 12:1115–1120. [PubMed: 19299499]
25. Lepeule J, Laden F, Dockery DW, Schwartz J. Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities study from 1974 to 2009. *Environ Health Perspect.* 2012; 120:965–970. [PubMed: 22456598]
26. 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; 115:679–683. [PubMed: 17520052]

27. Kravchenko J, Akushevich I, Abernethy A, Holman S, Ross W, Lysterly K. Long-term dynamics of death rates of emphysema, asthma, and pneumonia and improving air quality. *Int J Chron Obstruct Pulmon Dis*. 2014; 9:613–627. [PubMed: 25018627]
28. Dai L, Zanobetti A, Koutrakis P, Schwartz JD. Associations of fine particulate matter species with mortality in the United States: a multicity time-series analysis. *Environ Health Perspect*. 2014; 122:837–842. [PubMed: 24800826]
29. Dominici F, Levy JI, Louis TA. Methodological challenges and contributions in disaster epidemiology. *Epidemiol Rev*. 2005; 27:9–12. [PubMed: 15958422]
30. Bell ML, Ebisu K, Peng RD. Community-level spatial heterogeneity of chemical constituent levels of fine particulates and implications for epidemiological research. *J Expo Sci Environ Epidemiol*. 2011; 21:372–384. [PubMed: 20664652]
31. Baxter L, Duvall R, Sacks J. Examining the effects of air pollution composition on within region differences in PM_{2.5} mortality risk estimates. *J Expo Sci Environ Epidemiol*. 2013; 23:457–465. [PubMed: 23250195]
32. Levy DT, Nikolayev L, Mumford E. Recent trends in smoking and the role of public policies: results from the SimSmoke tobacco control policy simulation model. *Addiction*. 2005; 100:1526–1536. [PubMed: 16185214]
33. Daniel CR, Cross AJ, Koebnick C, Sinha R. Trends in meat consumption in the United States. *Public Health Nutr*. 2011; 14:575–583. [PubMed: 21070685]
34. Looker HC, Krakoff J, Andre V, et al. Secular trends in treatment and control of type 2 diabetes in an American Indian population: a 30-year longitudinal study. *Diabetes Care*. 2010; 33:2383–2389. [PubMed: 20855550]
35. Yeh RW, Normand SLT, Wang Y, Barr CD, Dominici F. Geographic disparities in the incidence and outcomes of hospitalized myocardial infarction does a rising tide lift all boats? *Circ Cardiovasc Qual Outcomes*. 2012; 5:197–204. [PubMed: 22354937]
36. Barr CD, Diez DM, Wang Y, Dominici F, Samet JM. Comprehensive smoking bans and acute myocardial infarction among Medicare enrollees in 387 U.S. counties: 1999 to 2008. *Am J Epidemiol*. 2012; 176:642–648. [PubMed: 22986145]
37. Chen J, Normand SL, Wang Y, Krumholz HM. National and regional trends in heart failure hospitalization and mortality rates for Medicare beneficiaries, 1998–2008. *JAMA*. 2011; 306:1669–1678. [PubMed: 22009099]
38. Chen J, Normand SL, Wang Y, Drye EE, Schreiner GC, Krumholz HM. Recent declines in hospitalizations for acute myocardial infarction for Medicare fee-for-service beneficiaries: progress and continuing challenges. *Circulation*. 2010; 121:1322–1328. [PubMed: 20212281]

FIGURE 1.
Changes in life expectancy in 95 US counties (2007 vs. 2002).

Circle, square, diamond, and triangle denote west, midwest, northeast, and south Census regions, respectively. Filled and unfilled symbols denote urban and nonurban counties, respectively.

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FIGURE 2.

County-specific yearly average of PM_{2.5} total mass and its seven components in 2002 versus 2007.

FIGURE 3.

A, Point estimates and 95% confidence intervals of the association across counties between a decrease (between 2002 and 2007) in an IQR ($\mu\text{g}/\text{m}^3$) of the $\text{PM}_{2.5}$ total mass or in each of the seven chemical components and an increase (between 2007 and 2002) in life expectancy (years). Results are reported for all the 95 counties (*left panel*), 43 nonurban counties (*middle panel*), and 52 urban counties (*right panel*). These estimates were obtained under an “unadjusted single pollutant model” that includes a single exposure predictor of the change in $\text{PM}_{2.5}$ total mass or the change in one of the seven components. B, Point estimates and 95% confidence intervals of the association across counties between a decrease (between 2002 and 2007) in an IQR $\mu\text{g}/\text{m}^3$ of the $\text{PM}_{2.5}$ total mass or in each of the seven chemical components and an increase (between 2007 and 2002) in life expectancy (years). Results are reported for all the 95 counties (*left panel*), 43 nonurban counties (*middle panel*), and 52

urban counties (*right panel*). These estimates were obtained under an “adjusted single pollutant model” that includes a single exposure predictor (change in PM_{2.5} total mass or the change in one of the seven components) and the additional confounders that represent changes in demographics, socioeconomic, population, lung cancer, and COPD variables (surrogate information on smoking population). C, Point estimates and 95% confidence intervals of the association across counties between a decrease (between 2002 and 2007) in an IQR $\mu\text{g}/\text{m}^3$ of the PM_{2.5} total mass or in each of the seven chemical components and an increase (between 2007 and 2002) in life expectancy (years). Results are reported for all the 95 counties (*left panel*), 43 nonurban counties (*middle panel*), and 52 urban counties (*right panel*). These estimates were obtained under an “adjusted multiple pollutant model” that includes changes in all the seven components as exposure predictors and the additional confounders that represent changes in demographics, socioeconomic, population, lung cancer, and COPD variables (surrogate information on smoking population).

TABLE 1

Summary Characteristics of 95 Counties for the Years 2002 and 2007: Average Value Across Counties

Measure	All Counties (n = 95)		Urban Counties (n = 52)		Nonurban Counties (n = 43)	
	2002	2007	2002	2007	2002	2007
Life expectancy (years)	76.6 (1.8)	77.5 (1.9)	76.6 (1.8)	77.7 (1.8)	76.6 (1.7)	77.3 (2.0)
PM _{2.5} mass (µg/m ³ [SD])	13.9 (3.1)	13.7 (3.1)	14.2 (2.9)	13.9 (3.0)	13.6 (3.3)	13.5 (3.4)
Sulfate (µg/m ³ [SD])	3.5 (1.4)	3.2 (1.4)	3.5 (1.4)	3.2 (1.4)	3.5 (1.5)	3.2 (1.5)
Nitrate (µg/m ³ [SD])	2.0 (1.3)	1.7 (1.0)	2.2 (1.4)	1.8 (1.0)	1.7 (1.1)	1.5 (1.0)
Ammonium (µg/m ³ [SD])	1.6 (0.7)	1.5 (0.6)	1.7 (0.7)	1.6 (0.6)	1.5 (0.6)	1.4 (0.6)
Elemental carbon (µg/m ³ [SD])	0.6 (0.3)	0.7 (0.3)	0.7 (0.3)	0.8 (0.3)	0.5 (0.2)	0.6 (0.3)
Organic carbon (µg/m ³ [SD])	4.5 (1.8)	3.7 (1.3)	4.6 (1.2)	3.7 (0.9)	4.4 (2.2)	3.8 (1.7)
Silicon (µg/m ³ [SD])	0.1 (0.1)	0.1 (0.1)	0.2 (0.1)	0.1 (0.1)	0.1 (0.1)	0.1 (0.1)
Sodium ion (µg/m ³ [SD])	0.2 (0.1)	0.1 (0.1)	0.2 (0.1)	0.1 (0.1)	0.2 (0.1)	0.1 (0.1)
Age-standardized lung cancer mortality (smoking surrogate, no./10,000 population) (SD) ^a	16.5 (3.7)	15.6 (4.0)	15.8 (3.5)	14.8 (3.6)	17.3 (3.8)	16.6 (4.3)
Age-standardized chronic obstructive pulmonary disease mortality (smoking surrogate, no./10,000 population) (SD) ^a	12.5 (3.0)	12.3 (3.4)	11.7 (3.1)	11.2 (3.3)	13.6 (2.5)	13.7 (3.0)
Current smoking (proportion of population) (SD) ^b	0.2 (0.1)	0.2 (<0.1)	0.2 (0.1)	0.2 (<0.1)	0.2 (0.1)	0.2 (0.1)
Per capita income (in thousands of \$) (SD)	41.2 (7.6)	49.5 (9.6)	43.1 (7.1)	51.6 (9.2)	39.1 (7.5)	47.0 (9.1)
Population (in hundreds of thousands) (SD)	6.9 (8.4)	7.2 (0.9)	10.7 (9.7)	11.1 (10.1)	2.3 (1.9)	2.5 (2.1)
High-school graduates (proportion of population) (SD)	0.8 (0.1)	0.9 (0.1)	0.8 (0.1)	0.9 (0.1)	0.8 (0.1)	0.9 (0.1)
Black population (proportion of population) (SD)	0.1 (0.2)	0.2 (0.2)	0.2 (0.2)	0.2 (0.1)	0.1 (0.1)	0.1 (<0.1)
Hispanic population (proportion of population) (SD)	0.1 (0.1)	0.1 (0.1)	0.1 (0.2)	0.2 (0.2)	0.1 (0.1)	0.1 (0.1)

Total population, proportion of population that was black, proportion of population that was Hispanic, and proportion of population that had graduated from high school were all compiled from US Census and American Community Survey data, and per capita income was compiled from data available through the Bureau of Economic Analysis. The income data was standardized using the Consumer Price Index with 2000 as the base year as provided by the US Bureau of Labor Statistics.

^a Calculated based on 2000 and 2005 National Center for Health Statistics death rates.

^b Calculated based on CDC's Behavioral Risk Factor Surveillance System 2002 and 2007 data. Data were available for 87 counties.

SD indicates standard deviation.

TABLE 2

Correlations Between the Changes (Between 2002 and 2007) for Each Pair of Chemical Components Across All 95 Counties^a

Component	PM _{2.5} Mass	Sulfate	Nitrate	Ammonium	Elemental Carbon	Organic Carbon	Silicon	Sodium
PM _{2.5} mass	1.00							
Sulfate	0.42	1.00						
Nitrate	0.53	0.21	1.00					
Ammonium	0.69	0.55	0.81	1.00				
Elemental carbon	0.38	0.22	-0.05	0.12	1.00			
Organic carbon	0.59	0.15	0.38	0.48	0.26	1.00		
Silicon	0.37	0.16	0.14	0.12	0.20	0.11	1.00	
Sodium ion	-0.17	-0.10	-0.07	-0.08	-0.12	0.16	<0.01	1.00

^a First, we calculated the county-specific difference between the 2002 and 2007 value of each component. Second, we calculated the correlations between these county-specific differences for each pair of PM_{2.5} components.