Estimated Changes in Life Expectancy and Adult Mortality Resulting from Declining PM$_{2.5}$ Exposures in the Contiguous United States: 1980–2010

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Background: PM$_{2.5}$ precursor emissions have declined over the course of several decades, following the implementation of local, state, and federal air quality policies. Estimating the corresponding change in population exposure and PM$_{2.5}$-attributable risk of death prior to the year 2000 is made difficult by the lack of PM$_{2.5}$ monitoring data.

Objectives: We used a new technique to estimate historical PM$_{2.5}$ concentrations, and estimated the effects of changes in PM$_{2.5}$ population exposures on mortality in adults (age ≥ 30 y), and on life expectancy at birth, in the contiguous United States during 1980–2010.

Methods: We estimated annual mean county-level PM$_{2.5}$ concentrations in 1980, 1990, 2000, and 2010 using universal kriging incorporating geographic variables. County-level death rates and national life tables for each year were obtained from the U.S. Census and Centers for Disease Control and Prevention. We used log-linear and nonlinear concentration–response coefficients from previous studies to estimate changes in the numbers of deaths and in life years and life expectancy at birth, attributable to changes in PM$_{2.5}$.

Results: Between 1980 and 2010, population-weighted PM$_{2.5}$ exposures fell by about half, and the estimated number of excess deaths declined by about a third. The States of California, Virginia, New Jersey, and Georgia had some of the largest estimated reductions in PM$_{2.5}$-attributable deaths. Relative to a counterfactual population with exposures held constant at 1980 levels, we estimated that people born in 2050 would experience a 1–2% increase in life expectancy at birth, and that there would be a cumulative gain of 4.4 million life years among adults ≥ 30 y of age.

Conclusions: Our estimates suggest that declines in PM$_{2.5}$ exposures between 1980 and 2010 have benefitted public health. https://doi.org/10.1289/EHP507

Introduction

Implementing the Clean Air Act has markedly improved outdoor air quality in the United States (U.S. EPA 2011a, 1997). The National Ambient Air Quality Standards for common air pollutants, first promulgated in the 1970s, set health-based ambient standards for six criteria pollutants: particulate matter, ground-level ozone, carbon monoxide, sulfur dioxide (SO$_2$), nitrogen dioxide (NO$_2$), and lead (Bachmann 2007). Emissions of these pollutants have declined by about 60% since 1980 (U.S. EPA 2016). While ambient data for fine particle matter concentrations (those 2.5 μm and smaller: PM$_{2.5}$) were not regularly collected on an extensive spatial scale until the late 1990s (U.S. EPA 2016), concentrations of monitored pollutants that are precursors to PM$_{2.5}$, including SO$_2$ in particular, have declined by approximately 80% during the period from 1980 to 2010 (U.S. EPA 2016).

Fine particles are of particular interest to health scientists and policy makers because of evidence from controlled human exposure, toxicological, and epidemiological studies that acute PM$_{2.5}$ exposure (over hours or days) is associated with adverse health outcomes, including aggravated asthma, hospital and emergency department visits, and premature death (Brook et al. 2010; U.S. EPA 2009; WHO 2013). In addition, epidemiologic studies have consistently reported that chronic (i.e., years-long) exposure to fine particles is associated with an increased risk of premature death (U.S. EPA 2009; WHO 2013). Systematic reviews by the U.S. Environmental Protection Agency (U.S. EPA) and the World Health Organization have concluded that there is a causal relationship between short- and long-term exposure to fine particles and the risk of premature death (U.S. EPA 2009; WHO 2013).

Air pollution human health impact assessments have used findings from epidemiological studies to estimate the human health impacts of air quality policies. These assessments model PM$_{2.5}$ emissions, air quality, and exposure to estimate counts and rates of adverse outcomes (Anenberg et al. 2011; Caiazzo et al. 2013; Fann et al. 2011a, 2011b, 2013; Lim et al. 2012; U.S. EPA 2009). Results of these analyses can help inform a chain of accountability that describes the events linking air quality policies to human health outcomes. As described by the Health Effects Institute, this chain includes five stages: a) regulatory action; b) changes in emissions; c) changes in ambient air quality; d) changes in exposure/dose; and e) human health responses (Health Effects Institute 2003). However, incomplete data on emissions, monitoring, personal exposure, and health outcomes have made it difficult to elucidate the links in this chain.

Photochemical transport models, such as the Community Multi-scale Air Quality Model, use archival emissions and meteorology data to predict pollutant concentrations (Byun and Schere 2006; U.S. EPA 2012a). The resolution of emissions inventories across sources and locations has improved since 2000, making it possible to model changes in air quality with greater confidence. However, prior to the advent of the extensive national regulatory PM$_{2.5}$ monitoring network in late 1990s, it was difficult to characterize changes in ambient PM$_{2.5}$ exposures.
over time and space, or estimate the subsequent effects of these changes on public health.

The Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air) developed a statistical model to estimate annual average PM$_{2.5}$ concentrations during 1980–2010 at locations throughout the continental United States (Kim et al. 2017). This national point prediction model does not rely on inventoried emissions data, and can be used to estimate long-term average PM$_{2.5}$ concentrations during 1980–2010 at any locations throughout the continental United States (Kim et al. 2017). This national point prediction model can be applied to estimate area-level long-term average PM$_{2.5}$ concentrations that are representative of population exposures during 1980, 1990, 2000, and 2010. For the present analysis, we used this prediction model to estimate decadal trends in population-representative annual mean PM$_{2.5}$ exposures to residents of the contiguous United States, and performed two analyses to estimate the influence of these multidecade changes in PM$_{2.5}$ exposure on health. We used the first analysis to estimate the number of deaths that were avoided due to falling PM$_{2.5}$ exposures during 1980–2010. Because air pollution health impact assessments report the number of individuals who die prematurely, but not the degree to which exposure to PM$_{2.5}$ shortens their life spans, we also used a life table approach to estimate changes in population longevity through the year 2050. By estimating the health effects of changes in PM$_{2.5}$ exposures, these two assessments inform the final stage of the chain of accountability (HEI 2003).

**Methods**

**Estimating Annual Mean PM$_{2.5}$ Concentrations in 1980, 1990, 2000, and 2010**

We estimated county average PM$_{2.5}$ concentrations using a previously developed point-wise spatiotemporal prediction model of PM$_{2.5}$ annual average concentrations in the continental United States for 1980–2010, as described elsewhere (Kim et al. 2017). This spatiotemporal prediction model for 1980–2010 was developed based on PM$_{2.5}$ annual average concentration data from 1999 through 2010 obtained from the U.S. EPA Federal Reference Method and the Interagency Monitoring of Protected Visual Environments (IMPROVE) networks (Hand et al. 2011; U.S. EPA 2009) and backward projection of pollutant trends. The model included terms for a spatially varying long-term mean and a time trend, as well as spatial smoothing of available data from nearby monitors (Kim et al. 2017). The overall time trend was estimated using monitoring data for 1999–2010, which was back-extrapolated to estimate the temporal trend before 1999 based on extensive sensitivity analyses of trends for sulfate and visibility. The long-term mean and time trends were characterized in a universal kriging framework with geographic variables, and spatial smoothing was modeled by using an exponential covariance function. External validation of the model using other sources of PM$_{2.5}$ data showed good model performance (Kim et al. 2017). For example, $R^2$ values were >0.7 based on the data for 1990–1999 from IMPROVE and the Children’s Health Study (Peters et al. 1999; Sisler and Malm 2000). Incorporating emission and meteorological data did not significantly improve the performance of the model (Kim et al. 2017).

Using the spatiotemporal prediction model, we predicted annual average concentrations for 1980, 1990, 2000, and 2010 at about 70,000 census tract centroids from the 2010 census, which we assumed were representative of residence locations. These census tract centroids were created by using the 2010 census boundary maps downloaded from the National Historical Geographic Information System website (https://www.nhgis.org/). We next computed population-weighted county-level averages of predictions across census tract centroids within a county for each year, where the population weight was the census tract population divided by the total county population.

**Estimating the Number of PM$_{2.5}$-Attributable Deaths**

Similar to previous studies (Hubbell et al. 2009a; Levy et al. 2002; Voorhees et al. 2011), we estimated the number of PM$_{2.5}$-related deaths using a health impact function. The function combines relative risk estimates from epidemiological studies (i.e., concentration–response functions representing the association between PM$_{2.5}$ exposure and mortality) with estimated annual mean PM$_{2.5}$ concentrations, population data, and baseline death rates for each county in each year. We derived these estimates using the Benefits Mapping and Analysis Program—Community Edition (BenMAP-CE, version 1.1, U.S. EPA) tool as described below.

We estimated the number of PM$_{2.5}$-related total deaths ($y_{ij}$) during each year $i$ ($i = 1980, 1990, 2000, 2010$) among adults aged 30 and above in each county $j$ ($j = 1, \ldots, J$ where $J$ is the total number of counties) as

$$y_{ij} = \sum_{m=1}^{M} y_{ijm}$$

where $\beta$ is the risk coefficient for all-cause mortality for adults in association with PM$_{2.5}$ exposure, $m_{ij} = \beta_{ij}$ is the baseline all-cause mortality rate for adults aged $a = 30–99$ in county $j$ in year $i$ stratified in 10-y age bins, $C_{ij}$ is annual mean PM$_{2.5}$ concentration in county $j$ in year $i$, and $P_{ij}$ is the number of county adult residents aged $a = 30–99$ in county $j$ in year $i$ stratified into 5-y age bins.

U.S. Census data for age-stratified population counts and county boundaries were stored within the BenMAP-CE tool for 3,109 contiguous U.S. counties in 2000 and 2010 (http://www.factfinder.census.gov); and corresponding data for 3,109 and 3,111 counties in 1980 and 1990, respectively, were obtained from the U.S. Census (NHGIS Database). County boundaries change over time, and so we assigned each set of death rates to the appropriate county boundary file for each year (NHGIS Database).

For baseline rates of death for adults aged 30–99, we selected county-level age-stratified all-cause death rates from the Centers for Disease Control (WONDER) database for each of the four years (CDC Wonder Database). In each of the four decadal periods in each county, we matched the age-stratified rate of death with the population count in that age range.

We used risk coefficients ($\beta$) drawn from a broadly cited long-term air pollution study of the extended American Cancer Society cohort (American Cancer Society Cancer Prevention Study II, CPS-II) (Krewski et al. 2009) and an impact assessment study of the same cohort (Nasari et al. 2016) to estimate the temporal trend before 1999 based on extensive sensitivity analyses of trends for sulfate and visibility. The long-term mean and time trends were characterized in a universal kriging framework with geographic variables, and spatial smoothing was modeled by using an exponential covariance function. External validation of the model using other sources of PM$_{2.5}$ data showed good model performance (Kim et al. 2017). For example, $R^2$ values were >0.7 based on the data for 1990–1999 from IMPROVE and the Children’s Health Study (Peters et al. 1999; Sisler and Malm 2000). Incorporating emission and meteorological data did not significantly improve the performance of the model (Kim et al. 2017).

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such that there is no threshold concentration below which PM$_{2.5}$-attributable mortality falls to zero. This assumption is consistent with findings in previous studies, which reported no evidence of a population-level threshold in the relationship between long-term exposure to PM$_{2.5}$ and mortality, and so we elected not to apply one in this health impact function (Crouse et al. 2012; Schwartz et al. 2008; U.S. EPA 2009).

There is some evidence suggesting that the assumption of a log-linear association between PM$_{2.5}$ and mortality may not hold at high PM$_{2.5}$ concentrations (Burnett et al. 2014; Pope et al. 2009b). In addition, the maximum PM$_{2.5}$ concentration predicted for counties in the present analysis (about 26 mg/m$^3$) is higher than the maximum exposure in the CPS-II population from which the Krewski et al. (2009) risk coefficient was derived (about 22 mg/m$^3$). Therefore, we derived a second set of attributable mortality estimates using a nonlinear form of the concentration–response relationship reported by Nasari et al. (2016) that was also based on the extended CPS-II study population.

This nonlinear risk function allows the risk of mortality to vary at different PM$_{2.5}$ concentrations, conditional on the values of two parameters, $\mu$ and $\tau$. Nasari et al. (2016) used CPS-II population data to derive a concentration–response curve and 95% CIs based on an ensemble analysis of models with different values of $\mu$ and $\tau$, with likelihood-based weights used to summarize the results. We used the same parameters [six paired values of $\mu$ and $\tau$ that contributed to the models in Nasari et al. (2016); these covered all weights greater than 0.001] to estimate county-specific numbers of PM$_{2.5}$-related deaths.

We performed a Monte Carlo simulation to construct an error distribution of estimated PM$_{2.5}$-related death counts using the standard error of risk coefficients reported in Krewski et al. (2009) and Nasari et al. (2016), respectively. We estimated total numbers of deaths in the continental United States for each year by summing the county-specific estimates, and reported the sums of the 2.5th and 97.5th percentiles of the Monte Carlo distributions as 95% CIs.

**Estimating the Fraction of Deaths Attributable to PM$_{2.5}$**

We calculated the fraction of all-cause deaths attributable to PM$_{2.5}$ deaths in each county and year using the following function:

$$AF_{ij} = \frac{y_{ij}}{\sum_i m_{bij} \times P_{ij}}$$

where $y_{ij}$ is the estimated number of PM$_{2.5}$-related all-cause deaths, $m_{bij}$ is the age-stratified baseline death rate, and $P_{ij}$ is the age-stratified population, respectively, in county $j$ in year $i$. Death rates for certain age strata were unavailable from the U.S. Census between 7 and 318 counties in 1980, either because data were missing or because the number of deaths were too small to report (e.g., in rural counties with small populations). For these counties, we imputed county-specific values using the median age-specific death rates for the United States as a whole. These imputed values represent about 3.2% of the total number of age-stratified county-level death rates. State-level estimates were derived by summing county-specific estimates.

**Estimating Gains in Life Years and Life Expectancy at Birth Attributable to Declines in PM$_{2.5}$**

We used a life table approach (Miller and Hurley 2003) to estimate national-level impacts on the number of years of life expected to remain at each age, and on life expectancy at birth, from 1980 to 2050. To estimate changes in life years and life expectancy values, we used the PopSim life table model, in the software BenMAP-CE (version 1.1; U.S. Environmental Protection Agency) as in a previous U.S. EPA analysis of changes in longevity associated with the provisions of the Clean Air Act (U.S. EPA 2011a). PopSim starts with a table reflecting the age-specific risk of death for a given starting year (i.e., 1980 here) and then adjusts the baseline hazard to account for the decrease (or increase) in air pollution-attributable risk of death, using national population-weighted changes in PM$_{2.5}$ over time (Table 2) and a risk coefficient (Krewski et al. 2009).

In brief, the model uses a Mortality Hazard Adjustment Factor (MHAF) to adjust gender-specific life table estimates of age-specific risks of death for a given baseline year (1980 in the present analysis) to account for the decrease (or increase) in air pollution-attributable risk of death. For the present analysis, the MHAF was derived for year $i$ ($i = 1980$, 1981, 1982 through 2050) as

$$MHAF_i = 1 + \sum_{i'}^{2050} \left( e^{\beta \times \Delta C_{i'} / \gamma} - 1 \right)$$

where $\beta$ is the fully adjusted risk coefficient for all-cause mortality in adults ($\geq 30$ y) in association with a 10 mg/m$^3$ increase in long-term PM$_{2.5}$ exposure from Krewski et al. (2009), and $\Delta C_{i'}$ is the national-scale population-weighted annual mean difference in PM$_{2.5}$ concentration (mg/m$^3$) in year $i'$ (the start year to the year $i$) from the previous year.

We calculated the population-weighted annual mean concentration for all counties combined ($C_i$) in year $i$ as

**Table 1. Risk coefficients of all-cause mortality for PM$_{2.5}$ concentrations applied to the health impact function.**

<table>
<thead>
<tr>
<th>Study</th>
<th>Study population</th>
<th>$\beta(\sigma)$</th>
<th>Likelihood weight$^1$</th>
<th>$\mu \text{g/m}^3$ (percentile)$^2$</th>
<th>$\tau^3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Krewski et al. (2009)$^b$</td>
<td>American Cancer Society Population ages $\geq$ 30 y</td>
<td>0.005826 (0.000962)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Nasari et al. (2016)$^b$</td>
<td>American Cancer Society Cancer Prevention Study II $\geq$ Population ages $\geq$ 30 y</td>
<td>0.0930 (0.0084)</td>
<td>0.036</td>
<td>–5.43 (−5%)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.0802 (0.00843)</td>
<td>0.080</td>
<td>1.38 (0%)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.0433 (0.00446)</td>
<td>0.460</td>
<td>8.19 (5%)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.0398 (0.00412)</td>
<td>0.334</td>
<td>9.04 (10%)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.0351 (0.00369)</td>
<td>0.056</td>
<td>10.55 (25%)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.0666 (0.00704)</td>
<td>0.044</td>
<td>1.38 (0%)</td>
<td>0.2</td>
</tr>
</tbody>
</table>

$^1$Long-term hazard ratio for all-cause PM$_{2.5}$-related mortality reported in the most recent extended analysis of the American Cancer Society Cancer Prevention Study II (ages 30 and older) [hazard ratio 1.06; 95% confidence interval (CI): 1.04, 1.08 per 10 mg/m$^3$ increase in average PM$_{2.5}$ concentrations in 1999-2000, adjusted for all individual-level and ecologic covariates].

$^2$This is the effect coefficient (per 1 mg/m$^3$) and standard error for each of the six log-linear concentration–response functions within a specific concentration range. Adjusted for individual-level and ecologic covariates.

$^3$We weighted the average of the six results using these likelihood weights.

$^4$This term determines the air quality level at which the c–r function curve.

$^5$Parameter $\tau$ controls the curvature of the weighting function, with larger values yielding shapes with less curvature.

$^6$This function would be specified in the BenMAP-CE tool as: $(1-(1/\text{EXP}((\text{Beta} \times \text{LOG}(Q_1))/(1+\text{EXP}(-(Q_1-(-5.43))/2.66)))) - \text{LOG}(Q_0)/(1+\text{EXP}(-(Q_0-(-5.43))/2.66))))$ × Incidence × POP.
In 1980, the fraction of PM$_{2.5}$-attributable mortality, was highest in the eastern United States and California, and in clusters of counties in western states, including Colorado, North Dakota, and Oregon (Figure 1). Statewide estimates of the PM$_{2.5}$-attributable mortality all indicated declines between 1980 and 2010, though the extent of the estimated decline varied among the states (Figure 2), and in a very small number of individual counties, PM$_{2.5}$-attributable mortality estimates increased over time (Figure S2). The average estimated reduction in PM$_{2.5}$-attributable mortality among states in the lowest quintile of PM$_{2.5}$-attributable mortality in 1980 (≤5.0%) was low compared with the estimated reduction among the states in the highest quintile in 1980 (≥10.1%) (Table S1).

We estimate that the U.S. population will experience an increase in life expectancy due to reductions in annual mean PM$_{2.5}$ concentrations from 1980–2010 (Table 4). Specifically, we estimate that because of declining PM$_{2.5}$ concentrations between 1980 and 2010 (and resulting reductions in PM$_{2.5}$-attributable mortality), the life expectancy of U.S. residents born in 2050 will be almost 1 y longer than it would have been if PM$_{2.5}$ had stayed constant at 1980 levels (0.94 and 0.87 additional y for men and women, respectively). In addition, we estimate that, as a group, U.S. adults ≥30 who are born in 2050 will live 4.4 million more years than they would have lived if they had been exposed to PM$_{2.5}$ at 1980 levels.

Discussion

To our knowledge, this is the first analysis to estimate national and local changes in ambient PM$_{2.5}$ concentrations from 1980 to 2010, and subsequent changes in adult mortality, in the contiguous United States. We applied a new approach to estimate historical PM$_{2.5}$ concentrations, including concentrations before the

### Table 2. Summary statistics of county-specific PM$_{2.5}$ annual average predictions and nationwide population-weighted PM$_{2.5}$ annual average predictions (in μg/m$^3$) in 1980, 1990, 2000, and 2010.

<table>
<thead>
<tr>
<th>Year</th>
<th>n</th>
<th>Min</th>
<th>10%</th>
<th>25%</th>
<th>50%</th>
<th>75%</th>
<th>90%</th>
<th>Max</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>3,111</td>
<td>2.95</td>
<td>6.93</td>
<td>9.72</td>
<td>12.43</td>
<td>14.30</td>
<td>15.81</td>
<td>21.80</td>
<td>11.92</td>
<td>3.34</td>
</tr>
</tbody>
</table>

Note: 10%, 25%, 50%, 75%, and 90% are percentiles. SD, standard deviation.

### Table 3. Estimated numbers and fractions of PM$_{2.5}$-attributable deaths in adults in the continental United States in 1980, 1990, 2000, and 2010.

<table>
<thead>
<tr>
<th>Year</th>
<th>Estimated numbers of PM$_{2.5}$-attributable deaths (95% confidence interval)$^a$</th>
<th>Estimated percentage of total deaths attributable to PM$_{2.5}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980</td>
<td>190,000 (130,000–240,000)</td>
<td>8.63%</td>
</tr>
<tr>
<td>1990</td>
<td>170,000 (110,000–220,000)</td>
<td>8.39%</td>
</tr>
<tr>
<td>2000</td>
<td>140,000 (98,000–190,000)</td>
<td>6.40%</td>
</tr>
<tr>
<td>2010</td>
<td>120,000 (83,000–160,000)</td>
<td>5.62%</td>
</tr>
</tbody>
</table>

$^a$Estimates rounded to two significant figures.

$^b$Counts of premature deaths estimated using a risk coefficient reported in the Krewski et al. (2009) long-term cohort study.

$^c$Counts of premature deaths estimated using risk coefficients reported in the Nasari et al. (2016) long-term cohort study.
spatially extensive PM$_{2.5}$ monitoring network was fully implemented in 1999. Using this approach, we estimated that annual average population-weighted PM$_{2.5}$ exposures for the United States as a whole dropped by nearly half between 1980–2010, while the U.S. population grew from 227 million to 309 million people (U.S. Census Bureau 2010).

Our estimates indicate that the human health burden of PM$_{2.5}$ declined as exposures decreased between 1980 and 2010, with a 30–40% reduction in the estimated number of PM$_{2.5}$-attributable deaths depending on the mortality risk coefficient applied (Krewski et al. 2009 or Nasari et al. 2016). Our estimates of the number of PM$_{2.5}$-attributable deaths in 2000 and 2010 (140,000 and 120,000, respectively) are generally consistent with estimates reported by others (Anenberg et al. 2010; Caiazzo et al. 2013; Fann et al. 2011a, 2013). For example, Anenberg et al. (2010) estimated that PM$_{2.5}$ exposures were responsible for approximately 124,000 cardiopulmonary deaths and 17,000 lung cancer deaths the United States and Canada in 2000, while Fann et al. (2011) estimated that PM$_{2.5}$ exposures caused approximately 120,000 premature deaths in the United States in 2005.

Analyses using the pooled Nasari et al. (2016) all-cause risk coefficients yielded consistently larger estimates of PM$_{2.5}$-related deaths than analyses that used the all-cause risk coefficient from Krewski et al. (2009)—generally by a margin of about 90,000 deaths—and 95% CIs were much wider as well. However, regardless of the risk coefficient used, estimated numbers of premature deaths decreased between 1980 and 2010. Although we had anticipated that mortality estimates based on the Nasari et al. (2016) risk coefficients would decline at an increasing rate over time (given the slight sigmoidal shape of the concentration–response relationship), this did not occur, probably because the slope of the concentration–response curve was roughly constant over the range of estimated annual mean population-weighted PM$_{2.5}$ concentrations during 1980–2010 (Table 2).

Air pollution health impact assessments for PM$_{2.5}$ often report estimated numbers of individuals who die prematurely due to PM$_{2.5}$ exposures, but not the degree to which exposure shortened their life spans. For this reason, we also estimated the additional life years gained and life expectancy at birth. Declining PM$_{2.5}$ concentrations over the three-decade period were associated with a cumulative increase in the estimated number of life years gained over time, such that falling PM$_{2.5}$ concentrations between 1980 and 1990 were estimated to result in thousands of additional life years by 1990, hundreds of thousands of additional life years by 2010, and millions of additional life years by 2050 (Table 3). Our estimates assumed that PM$_{2.5}$ concentrations will remain constant from 2010–2050, but this may be a conservative assumption given the likelihood of further reductions in PM$_{2.5}$ exposures due to local, state, and federal air quality policies implemented and promulgated after 2010 (U.S. EPA, 2010, 2011a, 2011b, 2011c, 2012a, 2012b, 2014a, 2014b).

The number of PM$_{2.5}$-related deaths increased by approximately 120,000 (95% CIs of 110,000 to 131,000) from 1980 to 1990, with a cumulative increase in the estimated number of life years gained and an additional 0.2 years of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame. For example, using different methods than those employed here, Pope et al. (2009a) estimated an average additional 0.4 y of life expectancy at birth for U.S. residents born in 2010 (relative to a counterfactual 2010 birth cohort with PM$_{2.5}$ exposures fixed at 1980 levels) is smaller than previous estimates based on measured PM$_{2.5}$ concentrations during a similar time frame.
are likely to have contributed to the decline in PM$_{2.5}$ concentrations from 1980–2010, federal air quality policies are likely to have played an important role (Chestnut and Mills 2005; U.S. EPA 2011a). The Acid Rain program and the Clean Air Interstate and Cross-State Air Pollution rules each contributed to reductions in SO$_2$ concentrations, which is a key precursor to forming PM$_{2.5}$ (Hubbell et al. 2009b). Other rules contributed to reductions in precursor emissions from light duty, heavy duty, and nonroad vehicles (Fann et al. 2012; U.S. EPA 2014a).

Our estimates of PM-related mortality were calculated relative to a baseline rate of death in each decade. There is ample evidence that the life expectancy of the U.S. population has been increasing over time (Bell and Miller 2005; CDC 2011), and the Social Security Administration reported a particularly sharp decline in the number of cardiovascular deaths between 1981 and 2001. Other things being equal, a decline in the death rate will yield smaller estimates of air pollution related deaths in a health impact assessment, suggesting that some fraction of the reduced PM$_{2.5}$ related deaths is partly a result of the population becoming healthier, more resilient, and less susceptible to the risk of PM$_{2.5}$-related death. On the other hand, previous studies using county-level data have reported that reductions in PM$_{2.5}$ concentrations were associated with lower death rates, even after changes in demographic and socioeconomic characteristics were accounted for (Correia et al. 2013; Pope et al. 2009a). Our estimates are subject to important uncertainties and limitations. We derived CIs for our estimates of PM$_{2.5}$-related mortality using an approach that incorporated uncertainty in the concentration–response estimates from previous studies due to random error, but we did not account for uncertainty due to other sources of error. For example, the model used to estimate historical levels of PM$_{2.5}$ before the PM$_{2.5}$ monitoring network was deployed had $R^2$s of 0.11–0.40 in 1980 and 1981, indicating poor predictive performance. Monitoring data from the Inhalable Particulate Network (IPN), which were used as the observed comparison data for validating our model predictions, were limited to data from

![Figure 2. Estimated reduction in PM$_{2.5}$-attributable Deaths in 48 U.S. states among adults Aged 30 and older between 1980 and 2010. Note: AL = Alabama; AR = Arkansas; AZ = Arizona; CA = California; CO = Colorado; CT = Connecticut; DE = Delaware; FL = Florida; GA = Georgia; IA = IOWA; ID = Idaho; IL = Illinois; IN = Indiana; KS = Kansas; KY = Kentucky; LA = Louisiana; MA = Massachusetts; MD = Maryland; ME = Maine; MI = Michigan; MN = Minnesota; MO = Missouri; MS = Mississippi; MT = Montana; NC = North Carolina; ND = North Dakota; NE = Nebraska; NH = New Hampshire; NJ = New Jersey; NM = New Mexico; NY = New York; OH = Ohio; OK = Oklahoma; OR = Oregon; PA = Pennsylvania; RI = Rhode Island; SC = South Carolina; SD = South Dakota; TN = Tennessee; TX = Texas; UT = Utah; VA = Virginia; VT = Vermont; WA = Washington; WI = Wisconsin; WV = West Virginia; WY = Wyoming. The region of the United States corresponds to seven clusters of states that are proximate to one another. The right-hand end of each arrow denotes the fraction of deaths due to PM$_{2.5}$ in 1980, while the left-hand side of each arrow shows the fraction of deaths due to PM$_{2.5}$ in 2010.](image-url)
Table 4. Estimated increase in life expectancy at birth and number of life years lived attributed to PM$_{2.5}$ concentration reductions between 1980 and 2010 for individuals born in 1990, 2000, 2010, and 2050.

<table>
<thead>
<tr>
<th>Year of birth</th>
<th>Men</th>
<th>Women</th>
<th>Cumulative number of life years gained$^{a,b,c}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>0.01</td>
<td>0.01</td>
<td>7,800</td>
</tr>
<tr>
<td>2000</td>
<td>0.19</td>
<td>0.18</td>
<td>140,000</td>
</tr>
<tr>
<td>2010</td>
<td>0.43</td>
<td>0.61</td>
<td>640,000</td>
</tr>
<tr>
<td>2050</td>
<td>0.94</td>
<td>0.87</td>
<td>4,400,000</td>
</tr>
</tbody>
</table>

$^{a}$Estimates rounded to two significant figures.

$^{b}$The all-cause risk coefficient from Krewski et al. (2009) was used to estimate changes in life expectancy at birth and life years, under the assumption that PM$_{2.5}$ has no effect on mortality prior to the age of 30.

$^{c}$Estimated increase in life expectancy at birth due to declines in PM$_{2.5}$ from 1980–2010, relative to the estimated life expectancy for the same birth cohort if PM$_{2.5}$ concentrations were fixed at 1980 concentrations.

$^{d}$Estimated cumulative increase in life years among adults ≥30 y of age in each birth cohort due to declines in PM$_{2.5}$ concentrations from 1980–2010 compared with the estimated life years for the same birth cohort if PM$_{2.5}$ concentrations were fixed at 1980 concentrations.

Conclusions

While subject to important limitations and uncertainties, our estimates suggest that the U.S. population has experienced significantly improved health, with fewer deaths and prolonged life expectancy at birth, as a result of declining exposures to fine particles in ambient air pollution between 1980 and 2010. In nearly all counties, estimated numbers of excess deaths and proportions of total deaths attributed to fine particles declined, consistent with a reduced public health burden. Future research might extend these findings by addressing whether the benefits of declining PM$_{2.5}$ levels vary among different population groups, and by projecting exposure estimates further into the future.

References


