

# Estimating the National Public Health Burden Associated with Exposure to Ambient PM<sub>2.5</sub> and Ozone

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Ground-level ozone (O<sub>3</sub>) and fine particulate matter (PM<sub>2.5</sub>) are associated with increased risk of mortality. We quantify the burden of modeled 2005 concentrations of O<sub>3</sub> and PM<sub>2.5</sub> on health in the United States. We use the photochemical Community Multiscale Air Quality (CMAQ) model in conjunction with ambient monitored data to create fused surfaces of summer season average 8-hour ozone and annual mean PM<sub>2.5</sub> levels at a 12 km grid resolution across the continental United States. Employing spatially resolved demographic and concentration data, we assess the spatial and age distribution of air-pollution-related mortality and morbidity. For both PM<sub>2.5</sub> and O<sub>3</sub> we also estimate: the percentage of total deaths due to each pollutant; the reduction in life years and life expectancy; and the deaths avoided according to hypothetical air quality improvements. Using PM<sub>2.5</sub> and O<sub>3</sub> mortality risk coefficients drawn from the long-term American Cancer Society (ACS) cohort study and National Mortality and Morbidity Air Pollution Study (NMMAPS), respectively, we estimate 130,000 PM<sub>2.5</sub>-related deaths and 4,700 ozone-related deaths to result from 2005 air quality levels. Among populations aged 65–99, we estimate nearly 1.1 million life years lost from PM<sub>2.5</sub> exposure and approximately 36,000 life years lost from ozone exposure. Among the 10 most populous counties, the percentage of deaths attributable to PM<sub>2.5</sub> and ozone ranges from 3.5% in San Jose to 10% in Los Angeles. These results show that despite significant improvements in air quality in recent decades, recent levels of PM<sub>2.5</sub> and ozone still pose a nontrivial risk to public health.

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**KEY WORDS:** air pollution; mortality; ozone; PM<sub>2.5</sub>; public health burden

## 1. INTRODUCTION

Ground-level ozone (O<sub>3</sub>) and fine particulate matter (PM<sub>2.5</sub>) are associated with increased risk of mortality.<sup>(1,2)</sup> While significant progress has been made in reducing ambient concentrations of air pollution in the United States, recent levels of O<sub>3</sub> and PM<sub>2.5</sub> remain elevated from the natural background and are within the range of concentrations found by epidemiology studies to affect health. This article estimates the public health burden attributable to

recent PM<sub>2.5</sub> and ozone air quality levels within the continental United States.

The World Health Organization Global Burden of Disease (GBD) study found that urban PM<sub>2.5</sub> was associated with about 28,000 premature mortalities in the United States, Canada, and Cuba.<sup>(3,4)</sup> Anenberg *et al.*<sup>(5)</sup> used a chemical transport model (resolution 2.8° × 2.8°) to simulate O<sub>3</sub> and PM<sub>2.5</sub> concentrations in both rural and urban areas, finding 35,000 respiratory premature mortalities due to O<sub>3</sub> in North America and 141,000 cardiopulmonary and lung cancer deaths due to PM<sub>2.5</sub> in North America.

Using simulated rather than monitored concentrations allows for full spatial coverage of air pollution impacts, but global chemical transport

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models are generally coarsely resolved and frequently unable to capture fine spatial gradients of population and concentrations, particularly around urban areas. Global health impact assessment is also limited by the coarse resolution of demographic data in many locations, such as population and baseline mortality rates.

Previously, U.S. EPA calculated the public health burden attributable to PM<sub>2.5</sub> in the United States at a 12 km resolution in the east and a 36 km resolution in the west, finding that the percentage of all-cause mortality associated with PM<sub>2.5</sub> exposure was as high as 11% in some counties.<sup>(6)</sup> However, that analysis did not consider O<sub>3</sub>-related or nonmortality impacts.

Here, we aim to quantify the burden of recent concentrations of O<sub>3</sub> and PM<sub>2.5</sub> on mortality in the United States prior to the implementation of several recently promulgated air quality regulations that promise to greatly improve future air quality. We use the photochemical Community Multiscale Air Quality (CMAQ) model<sup>(7)</sup> in conjunction with several years of ambient monitored data to create fused surfaces of summer season average 8-hour ozone and annual mean PM<sub>2.5</sub> levels at a 12 km grid resolution across the continental United States. In contrast to the global analyses above, we employ finely resolved demographic and concentration data to assess fully the spatial and age distribution of air-pollution-related mortality within specific geographic areas. We also utilize the environmental Benefits Mapping and Analysis Program (BenMAP), a software package that contains a library of PM<sub>2.5</sub> and ozone mortality and morbidity concentration-response functions and is able to automate the process of quantifying PM<sub>2.5</sub> and ozone health impacts from a large number of scenarios. In addition, compared to previous work this analysis expands the metrics for assessing the public health burden for PM<sub>2.5</sub> and ozone exposure by estimating the excess mortalities associated with meeting hypothetical air quality improvements nationwide; the percentage of total mortality attributable to these two pollutants; the estimated life years lost; the change in life expectancy; and the estimated PM<sub>2.5</sub> and ozone morbidity impacts including hospitalizations and nonfatal heart attacks.

These methodological refinements enable us to answer three key policy questions: (1) What are the estimated public health impacts of recent PM<sub>2.5</sub> and ozone levels in the United States? (2) How are these impacts distributed by geographic area, age, and pollutant? and (3) What would be the size and spatial

distribution of the health-related benefits of hypothetical air quality improvements?

## 2. METHODS

### 2.1. Overview of the HIA

We estimate the number of adverse health outcomes associated with population exposure to air pollution using a health impact function. The health impact function used in this analysis has four components: the change in air quality, the affected population, the baseline incidence rate, and the effect estimate drawn from the epidemiological studies.<sup>(8,9)</sup> A typical log-linear health impact function might be as follows:

$$\Delta y = y_0(e^{\beta \Delta x} - 1)Pop,$$

where  $y_0$  is the baseline incidence rate for the health endpoint assessed;  $Pop$  is the population affected by the change in air quality;  $\Delta x$  is the change in air quality; and  $\beta$  is the effect coefficient drawn from the epidemiological study.

Here we use BenMAP (version 4.0)<sup>(10)</sup> to systematize the HIA calculation process, drawing upon its library of population data, baseline incidence, and concentration-response functions. We first describe the CMAQ air quality modeling used to simulate PM<sub>2.5</sub> and ozone concentrations. We then detail our selection of population estimates used to calculate exposure, baseline incidence rates used to calculate risk, and the mortality and morbidity concentration-response functions used to assess PM<sub>2.5</sub> and ozone-related health impacts.

### 2.2. PM<sub>2.5</sub> and Ozone Air Quality Modeling

We utilize the CMAQ model<sup>(7)</sup> to estimate annual PM<sub>2.5</sub> and summer season ozone concentrations for the year 2005 for a horizontal grid covering the continental United States at a 12 km resolution. The CMAQ model is a nonproprietary computer model that simulates the formation and fate of photochemical oxidants, including PM<sub>2.5</sub> and ozone, for given input sets of meteorological conditions and emissions. The CMAQ model is a well-established and thoroughly vetted air quality model that has seen use in a number of national and international applications.<sup>(11–13)</sup> We use CMAQ version 4.7<sup>1</sup> and the U.S.

<sup>1</sup> CMAQ version 4.7 was released on December 1, 2008. It is available from the Community Modeling and Analysis System (CMAS) at: <http://www.cmascenter.org>.

EPA 2005 Modeling Platform, with emissions, meteorology, and initial and boundary conditions detailed elsewhere.<sup>(14,15)</sup> A detailed model performance evaluation for ozone, PM<sub>2.5</sub> and its related speciated components was conducted using observed/predicted pairs of daily/monthly/seasonal/annual concentrations.<sup>(14)</sup> Overall, the fractional bias, fractional error, normalized mean bias, and normalized mean error statistics were within the range or close to that found in other recent applications, and determined to be sufficient to provide a scientifically credible approach for this assessment.

We improve the accuracy of the air quality data used in this analysis by combining the CMAQ-modeled PM<sub>2.5</sub> and ozone concentrations with ambient monitored PM<sub>2.5</sub> and ozone measurements to create “fused” spatial surfaces for the domain shown in Figs. 1 and 2. We performed the fusion using the EPA’s Model Attainment Test Software (MATS),<sup>(16)</sup> which employs the Voronoi neighbor averaging interpolation technique.<sup>(17)</sup> Fusing modeled and measured ozone and PM<sub>2.5</sub> concentrations leverages the complete spatial and temporal coverage of modeled concentrations and the accuracy of observed air quality measurements. This technique identifies the set of monitors that are nearest to the center of each grid cell, and then takes an inverse distance squared weighted average of the monitor concentrations. The fused spatial fields are calculated by adjusting the interpolated ambient data (in each grid cell) up or down by a multiplicative factor calculated as the ratio of the modeled concentration at the grid cell divided by the modeled concentration at the nearest neighbor monitor locations (weighted by distance). For PM<sub>2.5</sub>, spatial surfaces were created by fusing all 2005 valid<sup>2</sup> modeled days of PM<sub>2.5</sub> concentrations with validated PM<sub>2.5</sub> data from 2004 to 2006 from Speciated Trends Network, Interagency Monitoring of Protected Visual Environments, and Clean Air Status and Trends Network monitoring sites. For ozone, we only used modeling results from the summer ozone season period between May 1 and September 30, 2005<sup>3</sup> and fused these data with mon-

itored ozone data from 2005 to 2007.<sup>4</sup> By fusing the CMAQ-modeled air quality data with multiple years of ambient measured data, the air quality concentrations should be more reflective of a 3-year average concentration, and less biased by unusual changes in emissions or meteorology that may have occurred during one year but not another (e.g., plant shutdown for maintenance).

We calculate the total public health burden attributable to PM<sub>2.5</sub> and ozone relative to “nonanthropogenic background” concentrations of summer-season ozone and annual mean PM<sub>2.5</sub> concentrations that would occur in the absence of anthropogenic emissions in the United States, Canada, and Mexico.<sup>(18)</sup> We identified two options to specifying these background levels. The first option was to apply PM<sub>2.5</sub> and ozone levels observed from monitors in remote locations. However, even remote monitors may be affected by nonlocal sources of nonbiogenic emissions. Alternatively, chemical transport models allow users to simulate background levels in the absence of anthropogenic emissions. For ozone, we use a median of the 4-hour mean value (13:00–17:00) for the eastern and western United States (22 ppb in the east and 30 ppb in the west) reported by Fiore *et al.*<sup>(19)</sup> In that analysis, the authors applied GEOS-Chem, a global circulation model, to model ozone formation due to emissions originating outside of the United States.<sup>(18)</sup> We then adjusted the ozone value reported in this study to an 8-hour maximum equivalent,<sup>(20)</sup> consistent with the air quality metric used in the concentration-response functions described below.

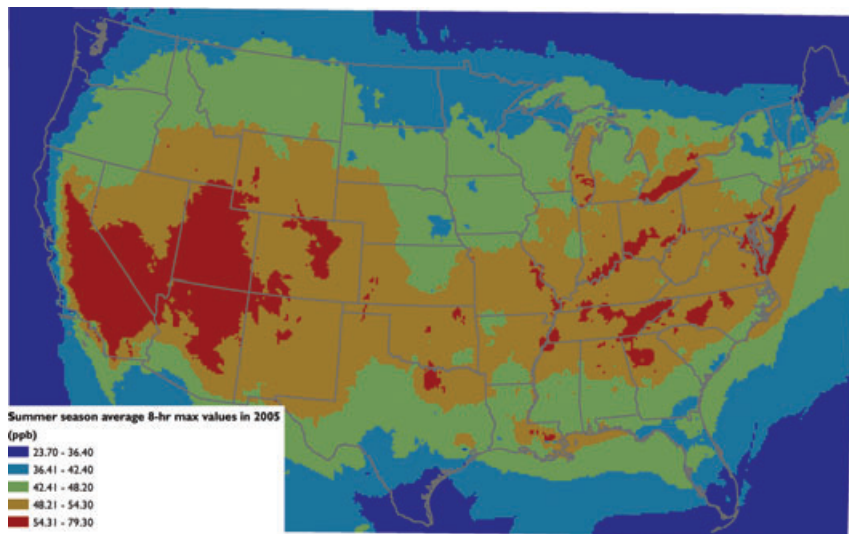
We applied background PM<sub>2.5</sub> levels specified in Table 3–23 of the 2009 EPA Integrated Science Assessment (ISA).<sup>(18)</sup> Within the ISA, average regional nonanthropogenic background concentrations were estimated using CMAQ v 4.7, with boundary conditions from GEOS-Chem and emissions from natural sources everywhere in the world, and anthropogenic sources outside continental North America.<sup>(18)</sup> The CMAQ modeling domain, with 36 km grid spacing, covered the continental United States. A model performance evaluation generally showed

<sup>2</sup> Normally, all 365 model days would have been used in the estimation of PM<sub>2.5</sub> levels; however during the modeling, an error was discovered in the aqueous phase chemistry routines of CMAQ v4.7. This error caused simulated hourly sulfate concentrations to increase sporadically and in an unrealistic manner over a very limited number of grid-cell hours over the RFS2 simulations. These data were removed as described in U.S. EPA, 2010b.

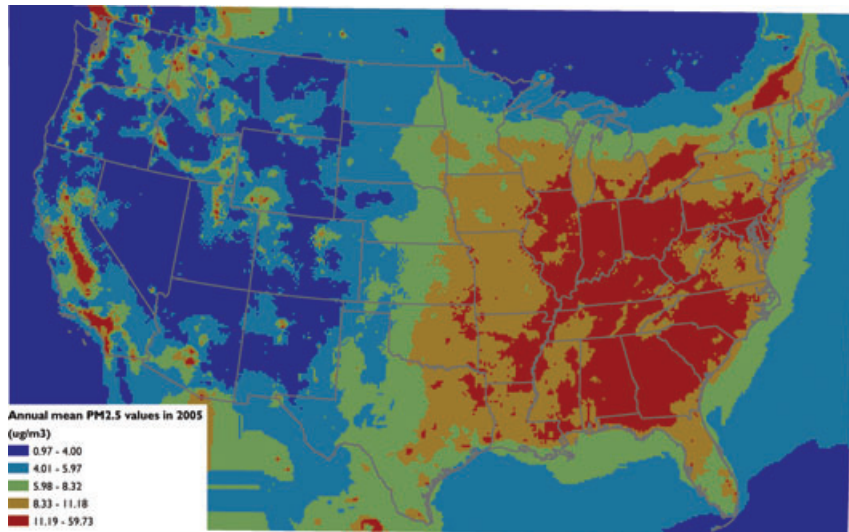
<sup>3</sup> This 153-day period generally conforms to the ozone season across most parts of the United States and contains the major-

ity of days with observed high ozone concentrations in 2005. We acknowledge that the ozone season extends beyond these dates in some urban areas (e.g., Houston, L.A.) and failing to account for the full duration of the season in these areas may introduce a downward bias in our estimate of health impacts.

<sup>4</sup> Normally, the calculation would have used the ambient data from 2004 to 2006. However, because of the abnormally low levels of ozone measured in the continental United States in 2004 as compared to that measured in 2000–2007, we chose to use the ambient data from the years of 2005–2007 instead.



**Fig. 1.** Average daily 8-hour maximum summer-season ozone levels (fused surface of 2005 modeled and 2004–2006 monitored data).



**Fig. 2.** Annual mean PM<sub>2.5</sub> levels (fused surface of 2005 modeled and 2004–2006 monitored data).

good agreement between modeled and monitored values at remote sites.<sup>(18)</sup>

To determine annual regional PM<sub>2.5</sub> background concentrations, the CMAQ domain was divided into seven regions (i.e., Northwest, Southwest, industrial Midwest, upper Midwest, Southwest, Northwest, and southern California). An annual average PM<sub>2.5</sub> concentration was calculated for each CMAQ grid cell and then a regional annual average was calculated from the grid cells within each of the seven regions. These values were 0.74  $\mu\text{g}/\text{m}^3$  for the Northeast, 1.72  $\mu\text{g}/\text{m}^3$  for the Southeast, 0.86  $\mu\text{g}/\text{m}^3$  for the industrial Midwest, 0.84  $\mu\text{g}/\text{m}^3$  for the upper Midwest, 0.62  $\mu\text{g}/\text{m}^3$  for the Southwest, 1.01  $\mu\text{g}/\text{m}^3$  for the

Northwest, and 0.84  $\mu\text{g}/\text{m}^3$  for southern California.<sup>(18)</sup>

### 2.3. Estimation of Air Quality Concentrations Across the Population

We aggregate U.S. Census block-level population data<sup>(21)</sup> to the national 12 km CMAQ modeling domain. We stratify population for the year 2000 by age, sex, race, and ethnicity categories corresponding to the demographic classifications considered in the health impact functions (see later) and project these data to 2005 using an economic forecasting model.<sup>(22)</sup> Modeled PM<sub>2.5</sub> and ozone concentrations

are matched with the population projected in each 12 km grid cell and we assume that the fused air quality value in each cell is the best measure of population exposure.

#### 2.4. Selection of Concentration-Response Relationships and Baseline Incidence Rates

We estimate impacts to several PM<sub>2.5</sub>-related human health endpoints, including premature deaths from long-term exposure, respiratory and cardiovascular-related hospital visits, asthma-related emergency department visits, chronic bronchitis, and nonfatal heart attacks among others. Ozone-related health endpoints include deaths from acute and long-term exposure, respiratory hospital admissions, and asthma-related emergency department visits among others. Table SI specifies each of the endpoints, epidemiological studies, and risk estimates considered in this analysis. We use annual mean PM<sub>2.5</sub> changes as a surrogate for daily changes in PM<sub>2.5</sub> for those functions that quantify short-term impacts; this is unlikely to add appreciable bias to the health impact estimates because the concentration-response functions are approximately linear across the air quality levels experienced by U.S. populations.

We consider several factors in selecting the appropriate epidemiological studies and concentration-response functions for this analysis, including whether the study was peer reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population; this selection procedure is described in detail in previous EPA regulatory analyses.<sup>(23–25)</sup> In general, the studies utilized here are consistent with those applied in recent EPA regulatory analyses.<sup>(24,25)</sup> Because of the significance of mortality as a health endpoint, we describe in detail the selection of the risk coefficients.

To estimate PM<sub>2.5</sub>-related long-term mortality we draw risk estimates from epidemiological studies based on data from two prospective cohort groups, often referred to as the Harvard Six-Cities Study, or “H6C,”<sup>(26,27)</sup> and the American Cancer Society “ACS” study;<sup>(2,28–30)</sup> these studies have found consistent relationships between fine particles and premature death across multiple locations in the United States.

For PM<sub>2.5</sub>, we use from the recent Krewski *et al.* (2009) extended analysis of the ACS cohort the all-cause mortality risk estimate from the random effects

Cox model that controls for 44 individual and seven ecological covariates, based on average exposure levels for 1999–2000 over 116 U.S. cities<sup>(2)</sup> (RR = 1.06, 95% confidence intervals 1.04–1.08 per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub>). We quantify all-cause mortality rather than cardiopulmonary or lung cancer mortality specifically because it is the most comprehensive estimate of PM-related mortality. We also applied an all-cause mortality risk estimate from the Laden *et al.* (2006) reanalysis of the H6C cohort<sup>(27)</sup> (RR = 1.16, 95% confidence intervals 1.07–1.26 per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub>).

There are strengths and weaknesses to each PM<sub>2.5</sub> mortality study that argue for using risk estimates drawn from both analyses. While the ACS-based study includes a much larger population over a broader geographic area than the H6C study, the ACS population is less racially diverse, better educated, and more affluent than the national average. By contrast, the H6C cohort population is more representative of the United States, but estimates PM mortality risk in eastern U.S. cities where PM<sub>2.5</sub> is generally comprised of a larger fraction of sulfate than it is in western cities. There are other differences in population demographics and exposure-related factors that may also contribute to differences in the eastern and western United States. To the extent that PM<sub>2.5</sub>-related mortality is strongly influenced by particle composition, applying a H6C-based risk coefficient nationwide may result in biased estimates of PM<sub>2.5</sub> mortality in the west. Conversely, applying an ACS-based risk estimate nationwide may not characterize well the PM<sub>2.5</sub> mortality impacts in the eastern United States.<sup>(31)</sup>

While we apply both the ACS- and H6C-based risk coefficients, we also include in the supplement to this article additional estimates of PM<sub>2.5</sub>-related premature mortality for the western United States using all-cause mortality risk estimates from the Krewski *et al.* (2009) intra-urban analysis of the Los Angeles region<sup>(2)</sup> (RR = 1.191, 95% confidence intervals 1.06–1.33 per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub>) (Fig. S1). As a means of generating as comprehensive an estimate of mortality possible, to also quantify PM-related infant deaths using a risk estimate from the cohort study by Woodruff *et al.*<sup>(32)</sup> This study found a significant link between PM<sub>10</sub> and infant death between 2 and 12 months of age. (RR = 1.04, 95% confidence intervals 1.02–1.07 per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub>).

For ozone, we estimate the change in ozone-related premature mortality applying both short- and

long-term risk estimates. A number of time-series ozone mortality studies including an analysis by Huang *et al.* (2004) in Los Angeles<sup>(33)</sup> and an analysis by Schwartz (2005)<sup>(34)</sup> in Houston have strengthened the findings of previous studies finding a relationship between short-term ozone exposure and premature mortality. The Bell *et al.* (2004)<sup>(1)</sup> analysis of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data set and the meta-analyses by Bell *et al.* (2005),<sup>(35)</sup> Ito *et al.* (2005),<sup>(36)</sup> and Levy *et al.* (2005)<sup>(37)</sup> sought to resolve the relationship between ozone, PM, weather-related variables, seasonality, and other variables. We apply the Bell *et al.* (2004) ozone nonaccidental mortality relative risk estimate (RR = 1.0052, 95% confidence intervals 1.0027–1.0077 per 10 ppb ozone increase)<sup>(1)</sup> because it is broadly cited. This study is also an NMMAPS-based analysis, which applies a common methodology to all cities, suggesting that it is not subject to the same risk of publication bias as the meta-analyses, which include the results of single-city studies.<sup>5(38)</sup>

Recent evidence also suggests a relationship between long-term exposure to ozone and premature respiratory mortality in the ACS cohort (Jerrett *et al.* 2009).<sup>(39)</sup> Jerrett *et al.* find that long-term exposure to ozone is associated with respiratory premature mortality in a two-pollutant model that controls for PM<sub>2.5</sub>. The Jerrett *et al.* (2009) estimate represents one of the few studies detecting an increase in mortality risk from long-term exposure to ozone. From this study, we apply a long-term respiratory mortality estimate (RR = 1.040, 95% confidence intervals 1.013–1.067 per 10 ppb ozone increase) as a means of capturing the impact of long-term ozone exposure. Until the literature evolves further, it will remain unclear whether the biological mechanisms underlying the ozone-related deaths detected by these short- and long-term studies are similar or different. By extension, the literature may also resolve the degree to which these studies are each detecting the same mortalities, over different periods of time. This uncertainty has implications for our findings, which we discuss below.

<sup>5</sup> Anderson and colleagues cite three reasons that single-city studies may be more prone to publication bias: (1) such studies frequently rely on easily available daily mortality counts, and researchers may be less inclined to publish negative findings; (2) the statistical modeling for time series entail a level of subjectivity and (3) each study can produce a large number of estimates and the researchers may select estimates based on the direction of their effect.

Because epidemiological studies assess changes in risk relative to some baseline rate, we use a baseline incidence rate for each health endpoint in the analysis (Table SII). Ideally, the incidence rate should also be matched to the geographic area of focus so that it describes the health status of the population of interest. In this analysis we apply a three-year average of 2006–2008 cause-specific county-level mortality rates from the CDC-WONDER database as a surrogate for 2005 mortality rates.<sup>(40)</sup> Morbidity rates are either national or regional averages depending on the data source (Table SII).

## 2.5. Calculating Health Impacts

Quantifying the number of PM<sub>2.5</sub> and ozone-related excess mortalities and morbidities involved specifying the health impact function with each of the key data inputs described above. This calculation produces counts of mortality and morbidity impacts. We estimate 95% confidence intervals around each mean health impact estimate using the Monte Carlo method, which samples a distribution based on the standard error reported in each epidemiological study. All estimated PM-related deaths are attributed to exposures to 2005 air quality, which we assume to occur over a 20-year period following this exposure, though recent research suggests that the lag between PM exposure and death is as short as 2 years.<sup>(25,41)</sup> We calculate the percentage of premature PM<sub>2.5</sub> and ozone-related mortality by dividing the number of excess deaths by the total number of cause-specific deaths in each county.

One criticism of the excess- or attributable-mortality calculation is that readers may infer that reductions in air pollution exposure result in deaths avoided altogether, when in fact these deaths are simply deferred into the future.<sup>(42)</sup> For this reason, we use standard life tables available from the Centers for Disease Control,<sup>(43)</sup> we estimate the number of life years and life expectancy lost to air pollution. We calculate the number of life years lost using the following formula:

$$\text{Total Life Years} = \sum_{i=1}^n LE_i \times M_i,$$

where  $LE_i$  is the remaining life expectancy for age interval  $i$ ,  $M_i$  is the change in number of deaths in age interval  $i$ , and  $n$  is the number of age intervals.<sup>(44)</sup> Alternate analyses have employed a cause-modified life

table approach, in which the change in life years and life expectancy is estimated among individuals with preexisting chronic conditions.

For example, the U.S. EPA quantified the change in life years lost due to attainment of alternate ozone National Ambient Air Quality Standards (NAAQS) among both the general population as well as individuals suffering from Chronic Obstructive Pulmonary Disease (COPD). EPA performed this analysis to test the sensitivity of the life year calculation to the assumption that ozone-related mortalities occurred primarily among individuals with preexisting chronic conditions that would increase their susceptibility to ozone mortality. We characterize the sensitivity of our life year and life expectancy estimates to this assumption below.

### 3. RESULTS AND DISCUSSION

#### 3.1. Air Quality Estimates

Figs. 1 and 2 show the geographic distribution of summer-season ozone and annual mean PM<sub>2.5</sub> concentrations across the continental United States for the “fused” spatial surfaces discussed above. The maximum predicted PM<sub>2.5</sub> value within a populated 12 km grid cell in the continental United States is 47.2  $\mu\text{g}/\text{m}^3$ , the mean PM<sub>2.5</sub> value is 7.8  $\mu\text{g}/\text{m}^3$ , the median is 7.48  $\mu\text{g}/\text{m}^3$ , and the 95th percentile value is about 13  $\mu\text{g}/\text{m}^3$ . The maximum predicted summer-season average 8-hour maximum ozone value in a populated 12 km grid cell is 79.3 ppb, the mean value is 47.9 ppb, the median is 48.3 ppb, and the 95th percentile value is 56.1 ppb.<sup>6</sup> In general, the highest PM<sub>2.5</sub> values are in the eastern United States, while the highest ozone values are located in the western United States.

#### 3.2. Estimates of Excess PM<sub>2.5</sub> and Ozone-Related Mortalities Nationwide

We predict over 100,000 PM<sub>2.5</sub>-related premature mortalities and tens of thousands of ozone-related premature mortalities to result from 2005

air quality levels (Table I).<sup>7</sup> We estimate over double the PM<sub>2.5</sub>-related mortalities using a risk estimate drawn the Laden *et al.* (2006)<sup>(27)</sup> H6C-based study as compared to the ACS-based Krewski *et al.* (2009).<sup>(2)</sup> We estimate about four times the number of ozone-related mortalities using the Jerrett *et al.* (2009) long-term respiratory mortality risk estimate as compared to the Bell *et al.* (2004) short-term mortality risk nonaccidental estimate. We also estimate an array of morbidity impacts, including almost 200,000 PM<sub>2.5</sub>-related nonfatal acute myocardial infarctions, tens of thousands of PM<sub>2.5</sub> and ozone-related hospitalizations and emergency department visits, and hundreds of thousands of PM<sub>2.5</sub>-related cases of acute bronchitis.

Because this analysis aims to estimate the total public health burden of air pollution, the estimated impacts reported in this article are significantly larger than those found in previous EPA analyses focused on proposed rules. For the Clean Air Interstate Rule, U.S. EPA estimated that approximately 17,000 PM<sub>2.5</sub>-related premature mortalities would be avoided in 2015 as a result of large-scale air quality improvements in the eastern United States occurring from the implementation of emission controls on electrical generating units (EGUs).<sup>(25,45)</sup> In 2006, U.S. EPA estimated that between 4,400 and 9,000 PM<sub>2.5</sub>-related premature mortalities would be avoided nationwide in 2020 from attaining a new annual PM<sub>2.5</sub> standard of 14  $\mu\text{g}/\text{m}^3$  and a daily standard of 35  $\mu\text{g}/\text{m}^3$  relative to a baseline in which the United States met an annual PM<sub>2.5</sub> standard of 15  $\mu\text{g}/\text{m}^3$  and a daily standard of 65  $\mu\text{g}/\text{m}^3$ .<sup>(46)</sup> U.S. EPA<sup>(23)</sup> estimated that between 450 and 2,100 ozone-related premature mortalities would be avoided in 2020 as a result of meeting a more stringent national ozone standard of 0.065 ppm relative to a baseline in which the United States meets an ozone NAAQS of 0.08 ppm. The ozone and PM<sub>2.5</sub> impacts we estimate for this study of the overall burden of disease are significantly larger than these U.S. EPA estimates because they are projected for a larger change in air quality, assuming the individual rules have not yet gone into effect and for a more

<sup>6</sup> The ozone air quality metric used to estimate health impacts in this analysis is not equivalent to design value metric used to determine attainment with the Ozone NAAQS. The ozone modeling used in this analysis is the summer-season average of the 8-hour daily maximum concentrations. The Ozone NAAQS is the fourth highest daily 8-hour maximum over the summer season. Therefore, these ozone concentrations are not equivalent to ozone concentrations for determining attainment with the Ozone NAAQS.

<sup>7</sup> When quantifying PM-related mortality, EPA generally assumes that reductions in PM-attributable deaths are distributed over a 20-year period, with a larger proportion of deaths occurring in earlier years. Recent research suggests that the lag between PM exposure and death is as short as 2 years (Schwartz *et al.* 2008). While the length and distribution of the PM mortality lag affects the discounting of monetized mortality benefits, it does not affect the overall size of the estimated premature mortalities.

**Table I.** Estimated PM<sub>2.5</sub> and Ozone-Related Health Impacts Due to 2005 Modeled Air Quality (Relative to Nonanthropogenic Background).

Health Effect	Annual Impact Estimates (95% Confidence Interval) <sup>a</sup>
<b>PM-related endpoints</b>	
Premature mortality	
Krewski <i>et al.</i> (2009) (age >29)	130,000 (51,000–200,000)
Laden <i>et al.</i> (2006) (age >24)	320,000 (180,000–440,000)
Infant (< 1 year)	1,800 (–1,500–4,800)
Chronic bronchitis (age > 27)	83,000 (16,000–140,000)
Nonfatal heart attacks (age > 17)	180,000 (70,000–270,000)
Hospital admissions— respiratory (all ages)	30,000 (15,000–45,000)
Hospital admissions— cardiovascular (age > 18)	62,000 (44,000–73,000)
Emergency room visits for asthma (age < 18)	110,000 (68,000–150,000)
Acute bronchitis (ages 8–12)	200,000 (–7,600–350,000)
Lower respiratory symptoms (ages 7–14)	2,400,000 (1,200,000–3,500,000)
Upper respiratory symptoms (asthmatics age 9–18)	2,000,000 (640,000–3,400,000)
Asthma exacerbation (asthmatics 6–18)	2,500,000 (280,000–6,800,000)
Lost work days (ages 18–65)	18,000,000 (15,000,000–20,000,000)
Minor restricted-activity days (ages 18–65)	100,000,000 (87,000,000–120,000,000)
<b>Ozone-related endpoints</b>	
Premature mortality	
Bell <i>et al.</i> (2004) (all ages)	4,700 (1,800–7,500)
Jerrett <i>et al.</i> (2009) (age >29)	19,000 (7,600–29,000)
Hospital admissions— respiratory causes (age > 64)	31,000 (1,200–53,000)
Hospital admissions— respiratory causes (age <2)	27,000 (13,000–39,000)
Emergency room visits for asthma (all ages)	19,000 (–1,200–58,000)
Minor restricted-activity days (ages 18–65)	29,000,000 (14,000,000–44,000,000)
School absence days	11,000,000 (4,500,000–16,000,000)

<sup>a</sup>95% confidence intervals calculated using a Monte Carlo method based on the standard error reported in each epidemiological study. Health impacts attributable to 2005 air quality levels. We assume a time lag between initial exposure to PM<sub>2.5</sub> and death.

comprehensive cross-section of U.S. population. Table SIII summarizes the per-person reduction in PM<sub>2.5</sub> exposure between 2005 and 2014, when a re-

cently proposed rule expected to achieve significant emission reductions from EGUs is scheduled to take effect.

### 3.3. Detailed Estimates of Impacts by Region, Age, and Air Quality Level

While the total estimates of excess mortality and morbidity help characterize the overall national public health burden attributable to recent air quality, they provide limited insight into how these estimated impacts are distributed by geographic location or by age. Below we: (1) consider the spatial distribution of these estimated mortality impacts; (2) characterize the PM<sub>2.5</sub> and ozone-related impacts according to the number of life years lost, change in life expectancy, and the percentage of total mortality attributable to these pollutants; and (3) quantify the reduction in mortality impacts according to hypothetical improvements in air quality. Unless otherwise noted, each of these analyses apply PM<sub>2.5</sub> and ozone mortality risk coefficients from the Krewski *et al.* and Bell *et al.* studies, respectively.<sup>(1,2)</sup>

#### 3.3.1. The Spatial Distribution of PM<sub>2.5</sub> and Ozone-Related Excess Mortalities

To illustrate the spatial distribution of the public health burden, we provide maps of the combined PM<sub>2.5</sub> and ozone-related mortality impacts by county in Fig. 3. These maps also identify the seven geographic regions previously used by the U.S. EPA when performing air quality and health impact analyses.<sup>(6)</sup> In any given location the number of the PM<sub>2.5</sub>-related mortalities will be influenced by the combination of air quality, population density, and baseline health status. On a per-person basis, southern California and the industrial Midwest see the greatest exposure to PM<sub>2.5</sub>. However, the confluence of poor air quality, population size and density, and baseline health status cause the largest number of estimated PM<sub>2.5</sub>-related excess premature mortalities to occur within the Northeast, Southeast, and Midwestern United States. Among urban areas, the largest estimated impacts occur in L.A., Chicago, Detroit, Pittsburgh, Houston, New York, Philadelphia, and Boston. The estimated ozone-related mortality impacts are an order of magnitude smaller than those estimated for PM<sub>2.5</sub>, partly due to the smaller relative risk associated with ozone, and generally distributed among the same counties affected most by PM<sub>2.5</sub> mortality.



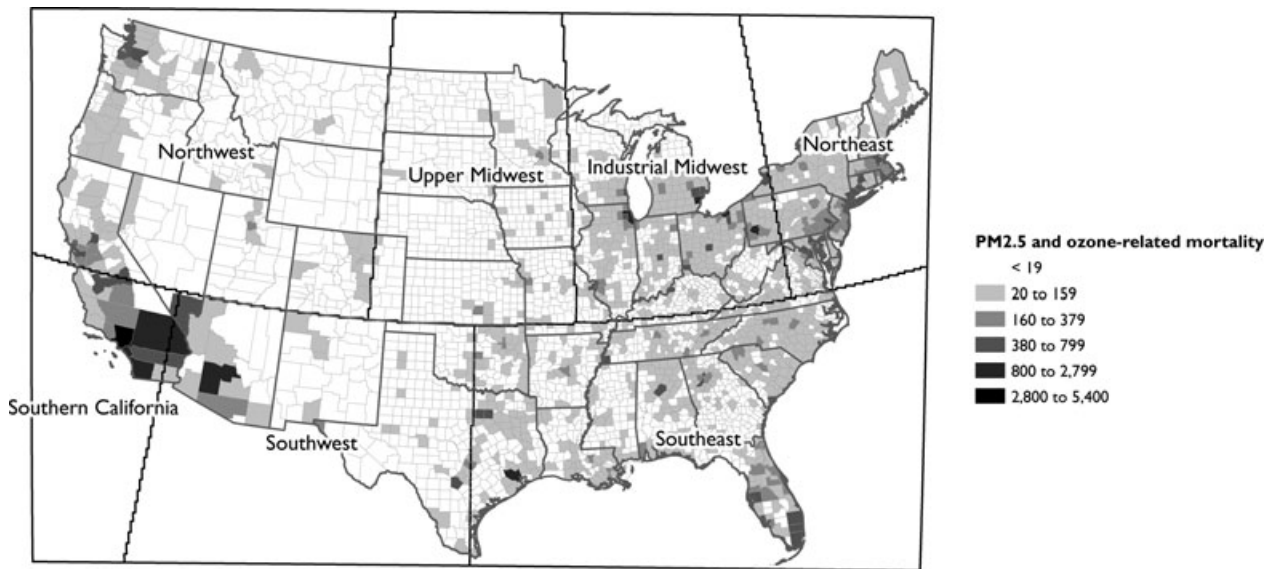


Fig. 3. PM<sub>2.5</sub> and ozone-related excess mortalities at the county level by geographic area.

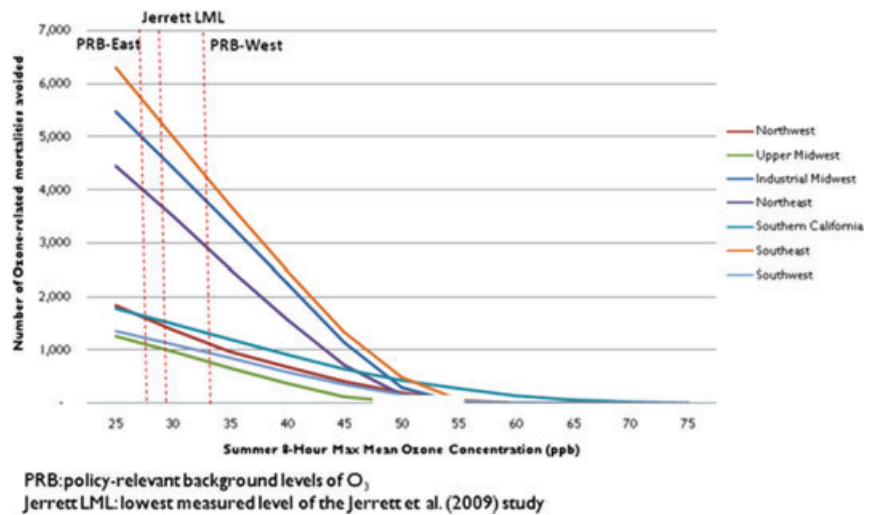
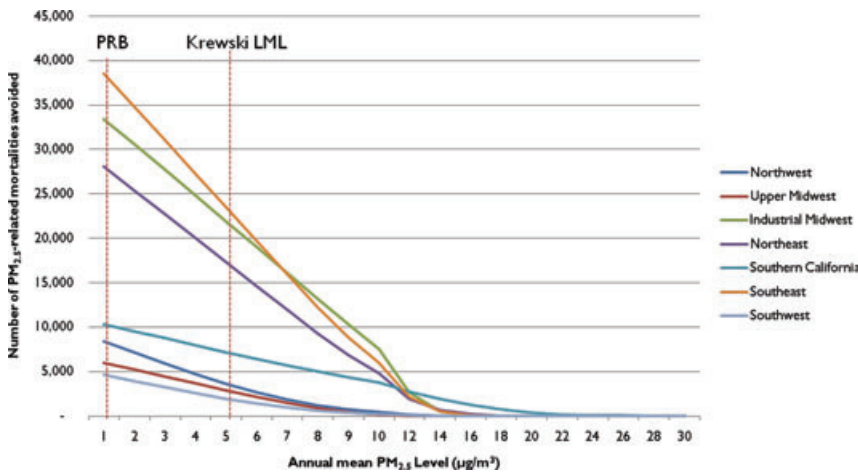


Fig. 4. The number of ozone-related excess mortalities avoided by air quality benchmark and region of the United States (using Jerrett *et al.* 2009 risk estimate).

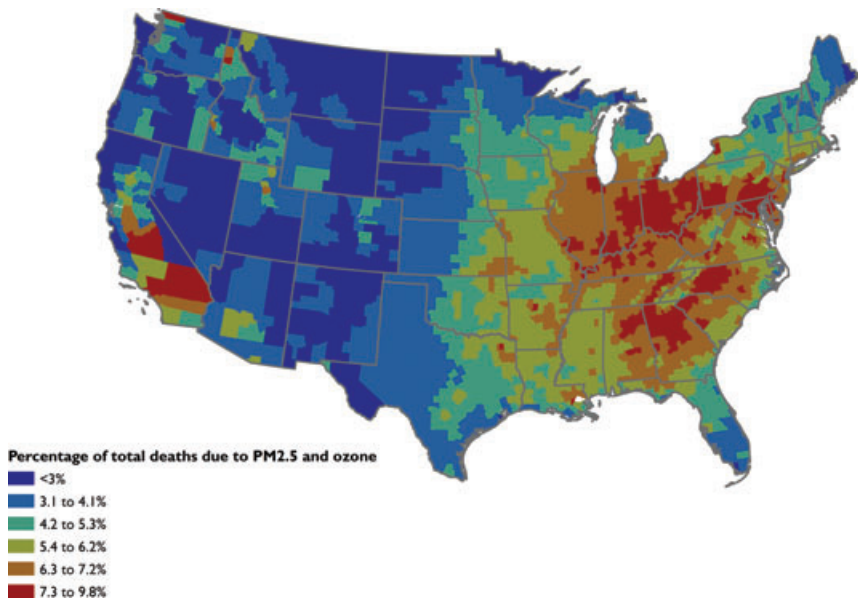
We also consider how the estimated number of avoided PM<sub>2.5</sub> and ozone deaths changes if we assume that air quality incrementally improves (Figs. 4 and 5). For example, we estimate that about 23,000 PM<sub>2.5</sub>-related mortalities would be avoided as a result of lowering 2005 annual mean PM<sub>2.5</sub> levels down to 10  $\mu\text{g}/\text{m}^3$  nationwide. We estimate about 80,000 premature mortalities would be avoided by lowering PM<sub>2.5</sub> levels to 5  $\mu\text{g}/\text{m}^3$  nationwide. We have less confidence in impacts estimated below the lowest measured level of the PM<sub>2.5</sub> mortality studies because we are less certain of the shape of the concentration-response relationship at these levels. However, given that there is little evidence for a

threshold PM<sub>2.5</sub> mortality function, such estimates still give some insight into the fraction of the public health benefits of air quality improvements at lower PM<sub>2.5</sub> levels. The avoided PM<sub>2.5</sub> and ozone-related mortalities appear to increase in an approximately linear fashion as we reduce air quality levels to lower benchmarks. The reduction in O<sub>3</sub> and PM<sub>2.5</sub>-related excess mortalities are not distributed evenly across the United States, and most are concentrated in the Northeast, Southeast, and industrial Midwest.<sup>8</sup>

<sup>8</sup> The ozone air quality benchmarks represent various daily 8-hour maximum levels averaged over the summer season and equivalent with ozone NAAQS levels, which are set according to a



**Fig. 5.** The number of PM<sub>2.5</sub>-related excess deaths avoided by air quality benchmark and region of the United States (using Krewski *et al.* 2009 risk estimate).



**Fig. 6.** Percentage of premature deaths attributable to 2005 PM<sub>2.5</sub> and ozone air quality by U.S. county.

3.3.2. *Estimating the Percentage of All Deaths Attributable to PM<sub>2.5</sub> and Ozone and the Change in Life Years and Life Expectancy*

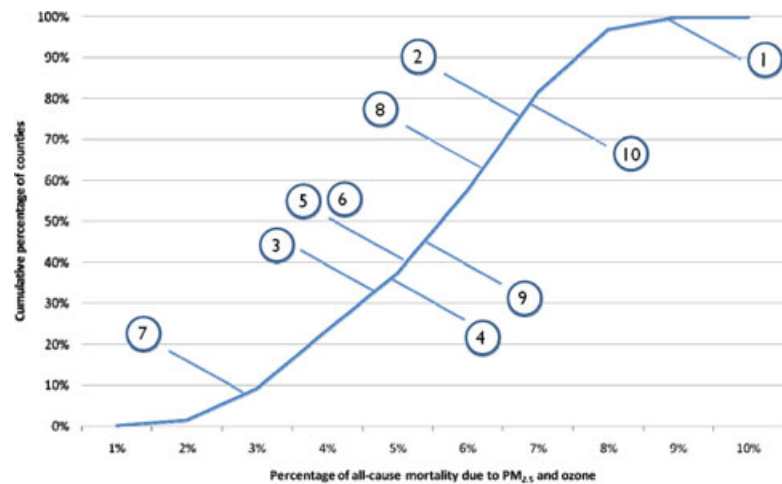
The counties with the largest estimated percentage of mortality due to PM<sub>2.5</sub> and ozone tend to be in the northeastern United States, the industrial Midwest, and southern California (Fig. 6). The cumulative distribution of the percentage of mortality attributable to PM<sub>2.5</sub> and ozone indicates that among the most populous counties, the proportion of total deaths attributable to PM<sub>2.5</sub> and ozone ranges from

a low of 3.5% in San Jose to as high as 10% in Los Angeles (Fig. 7). Nationwide, the percentage ranges from less than 1% to about 10% in southern California. We also estimate that about 19% of all ischemic-heart-disease-related deaths are attributable to PM<sub>2.5</sub> nationwide (Table SIV).

The overall percentage of all deaths due to PM<sub>2.5</sub> is much higher than it is for ozone (Figs. S2 and S3). While the percentage of all deaths attributable to PM<sub>2.5</sub> exposure is significantly higher in southern California than other regions, the percentage of total mortality from ozone exposure is roughly equal for southern California, the industrial Midwest, and to a lesser extent the Northeast and Southeast, and significantly lower in the Pacific Northwest. The

fourth highest daily design value. As such, health impacts at each ozone benchmark level do not reflect the health benefits of meeting different NAAQS levels.

**Fig. 7.** The cumulative distribution of the percentage of all-cause mortality attributable to PM<sub>2.5</sub> and ozone among the 10 most populous U. S. counties.



Counties are coded as follows: 1—Los Angeles; 2—Cook; 3—Harris; 4—Maricopa; 5—San Diego; 6—Dallas; 7—San Jose; 8—New York; 9—Bexar; 10—Philadelphia

spatial distribution of this metric and modeled ozone levels are fairly consistent across the United States. For both ozone and PM<sub>2.5</sub>, the percentage appears to decline modestly for older populations, suggesting that older populations may live in areas with lower modeled concentrations.

We estimate a large number of life years lost to PM<sub>2.5</sub> and ozone and this number varies by region of the country and by age (Figs. S4 and S5). For both PM<sub>2.5</sub> and ozone, the Northeast, Southeast, and industrial Midwest show the largest estimated total number of life years lost. Because the estimate of life years lost is influenced in part by the total number of individuals affected, we also estimate the percentage of these PM<sub>2.5</sub> and ozone-related life years lost by age range and region, which controls for differences in the size of the populations within each region (Table SV). Among populations aged 65–99, we estimate nearly 1.1 million life years lost from PM<sub>2.5</sub> exposure and approximately 36,000 life years lost from ozone exposure. The statistical abstract of the U.S. Census<sup>(47)</sup> reported 15 million life years lost among populations aged 65–99 from all causes in 2005, implying that PM<sub>2.5</sub> and ozone-related mortality accounted for approximately 7% of total life years lost among populations ages 65–99 nationwide in 2005.

Finally, using a standard life table, we quantify the change in life expectancy at birth and by 5-year age increment resulting from the elimination of PM<sub>2.5</sub> and ozone-related mortality risk (Table SVI). Among populations at birth, we estimate a change

in life expectancy of 0.7 years, a result that comports well with recent analyses of the effect of air pollution on life expectancy.<sup>(48)</sup>

When calculating changes in life expectancy, we assume that the life expectancy of those dying from air pollution is the same as the general population. It is possible to characterize the sensitivity of this assumption by referring to a 2008 U.S. EPA analysis of life years lost due to ozone exposure.<sup>(44)</sup> That analysis estimated approximately 14–53% fewer life years lost when assuming that populations dying premature from ozone exposure suffered average-to-severe COPD, as compared to the assumption that these populations shared the same life expectancy as the general population.

However, using a standard life table is reasonable when considering that: (1) the vast majority of premature deaths are estimated to occur among populations aged >64, half of whom suffer from one or more chronic illnesses, suggesting that a standard life table captures the change in life expectancy among a substantial number of individuals who suffer such chronic illnesses; and (2) a recent long-term study found that PM<sub>2.5</sub> initiated cardiovascular events among women with no history of cardiovascular disease—underscoring the role of air pollution in both promoting chronic illness and causing premature death.<sup>(49–51)</sup> Moreover, recent evidence available since the publication of that EPA report suggests that ozone-induced deaths do not occur exclusively, or even mostly, among individuals with such preexisting conditions.<sup>(52,53)</sup>

#### 4. DISCUSSION AND CONCLUSIONS

We have estimated the recent burden of PM<sub>2.5</sub> and ozone on human health in the United States, using ambient measurements, 2005 and nonanthropogenic background PM<sub>2.5</sub> and O<sub>3</sub> concentrations simulated by atmospheric chemistry models, and a health impact function. We find that between 130,000 and 340,000 premature deaths are attributable to PM<sub>2.5</sub> and O<sub>3</sub>. We also find that geographic and age distribution of this health risk is not shared equally. Major metropolitan areas including L.A., Houston, Pittsburgh, and New York see the largest number of estimated excess PM<sub>2.5</sub> and ozone-related deaths. Southern California is estimated to experience the largest percentage of total mortality attributable to PM<sub>2.5</sub> across all ages (between 7% and 17% depending on the risk estimate used), while the greatest percentage of mortality attributable to ozone is the highest in the industrial Midwest (between 0.24% and 1%, again depending on the risk estimate used). Conversely, the largest estimated number of PM<sub>2.5</sub> and ozone-related life years lost are in the Southeast. While estimating the contribution of air pollution to total morbidity impacts is difficult due to incomplete data on hospitalizations and other health endpoints, we find that the nonmortality impacts of air pollution are substantial, consistent with previous studies estimating air pollution mortality and morbidity.<sup>(3,4)</sup>

The size of these mortality estimates is comparable to those reported in Anenberg *et al.*<sup>(5)</sup> for North America and larger than those reported by Cohen *et al.*<sup>(3,4)</sup> Although our estimates may be larger than those of Cohen *et al.*,<sup>(3,4)</sup> our analysis includes several factors that may explain these differences. In particular, we estimate impacts relative to natural background and utilize modeled air quality that better represents population exposures in rural and urban areas. This general consistency with prior estimates of PM<sub>2.5</sub> and ozone impacts reaffirms that despite significant improvements in air quality in recent decades, recent levels of ozone and PM<sub>2.5</sub> still pose a public health risk in many regions of the United States. PM<sub>2.5</sub> and ozone impose a nontrivial level of mortality risk, particularly when compared to other causes of death. For example, while this analysis estimates between 130,000 and 340,000 PM<sub>2.5</sub> and ozone-attributable deaths from 2005 air quality, in this same year there were approximately 120,000 deaths due to accidents, 72,000 deaths due to Alzheimer's, and 63,000 deaths due to influenza.<sup>(54)</sup>

It is more challenging to evaluate the contribution of air pollution to total morbidity impacts. For example, incomplete information regarding the total number and spatial distribution of asthma hospitalizations prevent us from calculating the percentage of total asthma hospitalizations attributable to PM<sub>2.5</sub> with confidence. However, analyses including the GBD<sup>(3,4)</sup> provide evidence that the nonmortality impacts of air pollution are substantial—a finding reinforced by these results.

The estimates presented here are subject to a number of important limitations and uncertainties, only some of which we can quantify. Many of these are endemic to health impact assessments (e.g., the transfer of risk estimates from epidemiology studies to other contexts and the selection of epidemiological studies used to quantify impacts) and are described in detail elsewhere.<sup>(25,55)</sup> However, certain sources of uncertainties are likely to influence the analysis greatly and are worth noting here. This health impact analysis relies upon modeled air quality estimates that utilize a national emissions inventory. Previous analyses<sup>(46)</sup> have found that even small errors in emission inventory, when compounded with other uncertainties in the analysis, can have a significant impact on the overall size of the estimated health impacts.

Alternative methods for assessing air pollution health impacts might also have yielded different results. As noted earlier, the estimated life years lost and changes in life expectancy are sensitive to the assumption that populations dying prematurely from air pollution exposure share the same life expectancy as the general population. Assuming that air pollution deaths occur only among populations with preexisting chronic conditions yields significantly different results—though the empirical evidence suggests that premature death does not occur exclusively among such populations.<sup>(53)</sup> As another example of how alternative methods would have affected our results, we might have employed an Institute of Occupational Medicine (IOM)-style life table approach, calculating lifetime air pollution risk among a cohort of individuals. A principal advantage of this technique is that it characterizes changes in risk among a population cohort over time and reduces the chance that the same health impact may be counted twice from one year to the next.

Due in part to the limited availability of air quality modeling estimates, this analysis estimated health impacts and life expectancy changes attributable to air pollution exposure in a single historical year.

Future research might build upon this analysis by employing the IOM life-table tool in conjunction with both historical and projected air quality. Such a method would yield an improved characterization of the public health burden over time after the implementation of national air quality regulations.

Another approach is the comparative risk analysis method applied by the GBD, which aims to estimate air-pollution-related impacts within a risk framework that evaluates air pollution health impacts as one among many sources of public health risks.<sup>(3,4)</sup> One advantage of this type of comparative risk assessment is that by attempting to systematically account for all sources of mortality risk, of which air pollution is one component, it may reduce the potential for attributing an incorrect fraction of total mortality risk to air pollution. The GBD approach also aims to apportion air pollution risk according to indoor and outdoor exposure, which data limitations prevented this analysis from attempting.

Estimating PM<sub>2.5</sub> mortality and long-term ozone mortality impacts down to nonanthropogenic background levels also introduces important uncertainties. A sizable proportion of the total mortality attributed to these two pollutants occurs at air quality levels below the lowest measured level of each study (Figs. 4 and 5). Estimates of mortality impacts at air quality levels below the observable data in the epidemiology study are inherently more uncertain because at these levels we have less confidence in the shape of the concentration-response curve, although there is little evidence to suggest there is a threshold in the concentration-response functions.

We based PM<sub>2.5</sub> and ozone mortality and morbidity estimates on recent air quality concentrations. As such, our results do not reflect the important air quality improvements expected to result from an array of U.S. EPA and state air quality management programs that will be implemented in the near future—including the nonroad diesel rule, Tier-2 vehicle standards, the proposed transport rule, and a several maximum achievable control technology standards, among other rules. U.S. EPA projections of future air quality indicate that overall ambient levels of PM<sub>2.5</sub> and ozone will decline significantly compared to those levels estimated here. We anticipate that these rules will address a large portion of the PM<sub>2.5</sub> and O<sub>3</sub> public health burden identified in this article.

Another key uncertainty is the use of both time-series and cohort studies to quantify mortality impacts and changes in life years and life expectancy.

While we estimate PM<sub>2.5</sub>-related mortality using risk estimates drawn from two long-term cohort studies, we use both a short-term time-series study and a long-term cohort study to quantify ozone impacts. PM<sub>2.5</sub> cohort analyses are generally understood to better characterize the total risk of PM<sub>2.5</sub> exposure over time because they capture the impacts of both long-term and some portion of short-term exposures.<sup>(46,53,56)</sup> However, it is less clear as to whether there is a separate short- and long-term mortality impact related to ozone exposure—or whether the long-term study used in this analysis might be capturing these impacts. For this reason, there is some uncertainty as to whether the ozone mortality impacts estimated using the Levy *et al.* (2005) short-term study and the Jerrett *et al.* (2009) long-term study are additive or overlapping.

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