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## The influence of air quality model resolution on health impact assessment for fine particulate matter and its components

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### Abstract

Health impact assessments for fine particulate matter (PM<sub>2.5</sub>) often rely on simulated concentrations generated from air quality models. However, at the global level, these models often run at coarse resolutions, resulting in underestimates of peak concentrations in populated areas. This study aims to quantitatively examine the influence of model resolution on the estimates of mortality attributable to PM<sub>2.5</sub> and its species in the USA. We use GEOS-Chem, a global 3-D model of atmospheric composition, to simulate the 2008 annual average concentrations of PM<sub>2.5</sub> and its six species over North America. The model was run at a fine resolution of  $0.5 \times 0.66^\circ$  and a coarse resolution of  $2 \times 2.5^\circ$ , and mortality was calculated using output at the two resolutions. Using the fine-modeled concentrations, we estimate that 142,000 PM<sub>2.5</sub>-related deaths occurred in the USA in 2008, and the coarse resolution produces a national mortality estimate that is 8 % lower than the fine-model estimate. Our spatial analysis of mortality shows that coarse resolutions tend to substantially underestimate mortality in large urban centers. We also re-grid the fine-modeled concentrations to several coarser resolutions and repeat mortality calculation at these resolutions. We found that model resolution tends to have the greatest influence on mortality estimates associated with primary species and the least impact on dust-related mortality. Our findings provide evidence of possible biases in quantitative PM<sub>2.5</sub> health impact assessments in applications of global atmospheric models at coarse spatial resolutions.

### Keywords

Health impact assessment; PM<sub>2.5</sub>; Species; Grid resolution; Premature mortality

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## Introduction

Epidemiological studies have linked exposure to ambient fine particulate matter (PM<sub>2.5</sub>) with a wide spectrum of adverse health outcomes, from acute respiratory symptoms to premature deaths. These studies have demonstrated significant associations between both short-term and long-term mortality and exposure to PM<sub>2.5</sub> (Beelen et al. 2008; Dockery et al. 1993; Filleul et al. 2005; Krewski et al. 2009; Laden et al. 2006; Ostro 2004; Pope et al. 2002).

An air quality health impact assessment is an integrated procedure that synthesizes information about air pollution exposures, health effects, and population vulnerability (Kheirbek et al. 2013). This approach has been applied in estimating the burden of disease attributed to air pollution at the national and global levels (Anenberg et al. 2010; Cohen et al. 2005; Fann et al. 2013; Li et al. 2010) and potential public health benefits associated with air quality regulations (Li and Crawford-Brown 2011; Wang and Mauzerall 2006).

Health impact assessments commonly rely on pollutant concentrations modeled with air quality models. Models are very useful tools in this setting because they enable examination of alternative scenarios related to factors such as air pollution emissions, climate, and population growth. In addition, since observational data for air pollutants are only available at limited monitoring locations, model-simulated concentrations have advantages in capturing health impacts that are more spatially and temporally complete than monitoring data. However, grid cell resolution can be limited by computational power, particularly on a larger scale such as global atmospheric models. These models often run at resolutions of roughly 100–400 km (Anenberg et al. 2010, 2012; Bey et al. 2001; West et al. 2006). For PM<sub>2.5</sub>, many of the species that make up its total mass concentration are formed in the atmosphere from chemical reactions between precursor species (Thompson et al. 2014). Strong spatial concentration gradients of emissions can influence chemical production, leading to errors at too coarse resolution due to spatial averaging of emissions (Thompson et al. 2014). As a result, coarse resolution may suppress high urban PM<sub>2.5</sub> concentrations. This in turn can lead to biased health impact estimates. Coarse models do not resolve important relationships between gradients in population and gradients in air pollution. When these gradients are positively correlated, as for black carbon (BC) and other primary anthropogenic PM species, a coarse model will tend to underestimate health impacts. When they are anti-correlated, as for natural dust, a coarse model will tend to overestimate health impacts.

While studies have been conducted to investigate the effect of resolution on predicted PM<sub>2.5</sub> concentrations (Arunachalam et al. 2011; De Meij et al. 2007; Mensink et al. 2008), the influence of grid resolution on health impact assessment for PM<sub>2.5</sub> is not yet well understood. Given the importance of grid resolution on both predicted concentrations and population exposure in a health impact assessment for PM<sub>2.5</sub>, some recent studies investigated how estimates of health impacts at coarser resolution differ from those at fine resolution (Punger and West 2013; Thompson et al. 2014). However, these studies have reported conflicting results: one found that coarse grid resolutions produce national

mortality estimates in the USA that are substantially biased low (Punger and West 2013), whereas the other reported that health impacts associated with changes in  $PM_{2.5}$  concentrations were not sensitive to model resolution (Thompson et al. 2014). Furthermore, these studies relied on urban- and regional-scale model simulations, and studies have not been conducted using simulations from global atmospheric models. In addition, most studies focus on total  $PM_{2.5}$  mass. Since coarse model resolution is thought to affect primary particulates much more strongly than secondary particulate matter (EPA 2007; Punger and West 2013), the effects of resolution on different  $PM_{2.5}$  species warrant further investigation.

This study quantitatively examines the influence of model resolution on estimates of premature mortality attributable to  $PM_{2.5}$  and its chemical components in the USA. We use a global atmospheric model to simulate annual average concentrations of six  $PM_{2.5}$  species: black carbon (BC), ammonium ( $NH_4$ ), nitrate (NIT), sulfate ( $SO_4$ ), organic carbon (OC), and dust (DST) at two spatial resolutions. We also re-grid the fine-scale estimate to coarser scales. Overall, our main study purpose is to help understand possible biases in health impact assessment results across a range of PM species due to coarse grid resolutions of global atmospheric models.

The remainder of the paper is organized as follows: the “Methodology” section describes details of modeling annual average concentrations of air pollutants and the methods used to quantify mortality associated with pollutant exposure in the USA. The “Results and discussions” section presents our mortality estimates at various modeled and re-gridded resolutions. For each  $PM_{2.5}$  species, we quantify how mortality estimates differ from the fine-model resolution to the coarse-model resolution, and from the fine-model resolution to the re-scaled coarser resolutions. We compare the difference in mortality estimates at the national level and county level. The “Discussion and conclusions” section summarizes the conclusions of this study and proposes issues to be further investigated in future work.

## Methodology

### Overview of the method

We evaluate the effect of model resolution on health impact assessment by calculating annual mortality attributable to total  $PM_{2.5}$  and six of its principal component species in the continental USA using modeled and scaled pollutant concentrations. We then compare the mortality estimates based on various modeled or scaled model output to explore the bias resulted from coarse resolution.

The health impacts of  $PM_{2.5}$  and its species are calculated based on the following components: simulated air concentrations, the size of the affected population, baseline mortality rates, and concentration–response functions abstracted from epidemiologic literature. These factors are combined in an integrated procedure using a health impact function. A log-linear health impact function to quantify the excess mortality attributed to air pollution is (Anenberg et al. 2012; Li et al. 2010)

$$\Delta y = \left(1 - e^{-\beta \cdot \Delta x}\right) \times y_0 \times Pop, \quad (1)$$

where

$y$	Excess mortality attributable to pollutant exposures
$\beta$	Concentration–response coefficient from epidemiological studies (increase in mortality per unit increase in pollutant concentration)
$x$	Change in air quality, taken here as the modeled pollutant concentrations
$y_0$	Baseline mortality rates
Pop	The affected population, i.e., the number of people exposed to PM <sub>2.5</sub> pollution

Here, we use BenMAP (Environmental Benefits Mapping and Analysis Program, version 4.0.67, Abt Associates 2013) to calculate the health impacts of exposure to BC. In the following sections, we first describe the GEOS-Chem air quality model used to simulate annual average PM<sub>2.5</sub> concentrations across the continental USA. We then discuss how we select the concentration–response coefficient ( $\beta$ ) used to estimate mortality due to PM<sub>2.5</sub> and its species and how we characterize the affected population (Pop) and the baseline incidence rates ( $y_0$ ) in Eq. (1). We also discuss the approach we use to estimate mortality associated with individual species.

### Modeling annual average pollutant concentrations

We use GEOS-Chem ([www.geos-chem.org](http://www.geos-chem.org)) (Bey et al. 2001), a global 3-D model of atmospheric composition, to simulate annual average concentrations of the six PM<sub>2.5</sub> species over North America in 2008. GEOS-Chem is driven by assimilated meteorological observations from the Goddard Earth Observing System (GEOS) of the NASA Global Modeling Assimilation Office. Global simulations are performed at the 4° latitude × 5° longitude degree resolution, with a 6-month spin-up beginning in July 2007, through the end of 2008. Simulated 3-hourly concentrations from this global calculation are used as the boundary conditions to drive higher-resolution nested simulations over North America at the 0.5° latitude by 0.667° longitude resolution (Chen et al. 2009; Wang et al. 2004). We also computed a global-scale simulation at the 2° latitude × 2.5° longitude resolution. In addition, we re-gridded the modeled concentrations from the 0.5° × 0.667° simulation to several coarser resolutions through simple averaging, including 1° latitude × 1.25° longitude, 2° latitude × 2.5° longitude, and 4° latitude × 5° longitude. The standard BenMAP configuration requires the national 12-km Community Multiscale Air Quality (CMAQ) model grid to evaluate air quality model output. Therefore, we convert all model results to the national 12 × 12 km<sup>2</sup> grid using a two-step process. GEOS-Chem binary punch output is first converted to Climate Forecasting compliant NetCDF with PseudoNetCDF ([pseudonetcdf.googlecode.com](http://pseudonetcdf.googlecode.com)) and then re-gridded using a first-order conservative method as implemented in the Climate Data Operators ([code.zmaw.de/projects/cdo](http://code.zmaw.de/projects/cdo)).

The GEOS-Chem aerosol simulation includes treatment of the ammonium–nitrate–sulfate–water particulate equilibrium (Fountoukis and Nenes 2007; Park et al. 2004), elemental and organic carbonaceous aerosol (Park et al. 2003), dust (Duncan Fairlie et al. 2007), and sea salt (Alexander et al. 2005; Jaeglé et al. 2011). In this study, we did not consider secondary organic aerosol, which likely constitutes a significant fraction of PM<sub>2.5</sub> concentrations (Jimenez et al. 2009), as we focus more on species around which current air quality policies are designed (i.e., primary carbonaceous aerosol and the secondary inorganic species

ammonium sulfate and ammonium nitrate). Aerosol dry deposition is calculated following the resistance-in-series scheme of Wesely (1989); wet removal owing to large-scale precipitation and convective scavenging follows Liu et al. (2001).

Emissions of aerosols and aerosol precursors come from several global and regional inventories. Annual emissions from biofuel and fossil fuel are from Bond et al. (2007), replaced by the inventory of Cooke et al. (1999) over North America, with monthly scaling factors applied from Park et al. (2003). Biomass burning emissions are taken from the Global Fire Emissions Database (Giglio et al. 2010; van der Werf et al. 2010).

Anthropogenic emissions of NO<sub>x</sub> and SO<sub>2</sub> are from the global 2000 inventory of EDGAR 3.2 (Olivier et al. 2005) with annual scaling factors for 2005 from van Donkelaar et al. (2008). Emissions of NH<sub>3</sub> are described in Park et al. (2004). Emissions of SO<sub>2</sub> and NO<sub>x</sub> in the USA are overwritten with values from the US National Emission Inventory (NEI) for 2005. Emissions of SO<sub>2</sub>, NO<sub>x</sub>, and NH<sub>3</sub> are also overwritten by national inventories in Canada (CAC), Mexico (BRAVO), Europe (EMEP), and East Asia (Streets et al. 2006).

Simulated values of PM<sub>2.5</sub> and component species in the USA have been extensively compared to measurements from in situ monitoring networks (Heald et al. 2012; Henze et al. 2009; Liao et al. 2007; Mao et al. 2013; van Donkelaar et al. 2010; Walker et al. 2012; Zhang et al. 2012, 2013; Zhu et al. 2013), aircraft campaigns (van Donkelaar et al. 2008; Dunlea et al. 2009; Fisher et al. 2011; Heald et al. 2011), and remote sensing observations (Generoso et al. 2008; Heald et al. 2010; Johnson et al. 2012; Luan and Jaeglé 2013; van Donkelaar et al. 2010). While the model generally well represents the overall magnitude and relative speciation of primary aerosol and secondary inorganic aerosol (Philip et al. 2014), there is a persistent overestimate of aerosol nitrate in the eastern USA (Heald et al. 2012).

### Health impact assessment

Cohort studies on the associations between PM<sub>2.5</sub> exposure and mortality have been used to estimate PM<sub>2.5</sub>-related long-term mortality in health impact assessment. Two prospective cohort studies have been conducted in the USA, often referred to as the Harvard Six-Cities (H6C) study (Dockery et al. 1993; Laden et al. 2006) and the American Cancer Society (ACS) study (Krewski et al. 2009; Pope et al. 1995, 2002). These studies have found rather consistent relationships between PM<sub>2.5</sub> exposure and premature death across various locations in the USA. More recently, a study has developed an integrated exposure-response function (Burnett et al. 2014), which was used in the Global Burden of Diseases, Injuries, and Risk Factors Study 2010 (Lim et al. 2013).

For this study, we select the concentration-response coefficient for all-cause mortality from the recent Krewski et al. (2009) extended analysis of the ACS study, which includes a large cohort of 500,000 adults over 116 US cities. The mortality risk estimates from the ACS study have been used to assess the health burden associated with exposure to ambient PM<sub>2.5</sub> in the USA and globally (Anenberg et al. 2010; Fann et al. 2012; Pungler and West 2013). Using a 1999–2000 random-effects Cox model adjusted for 7 ecological and 44 individual covariates, Krewski et al. (2009) predicted a risk ratio of 1.06 (95 % confidence interval, CI, 1.04–1.08) for a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub> concentration for all-cause mortality. This risk ratio was converted to a concentration–response coefficient value of 0.005827 used in this

study (a normal distribution with a standard deviation of 0.000963). Although some studies have suggested that certain species of PM<sub>2.5</sub>, such as black carbon, may pose a greater mortality risk on human health than other components (Janssen et al. 2011; Smith et al. 2010), current evidence is still insufficient to differentiate the health effects of the various constituents of PM<sub>2.5</sub> (EPA. 2012). Based on this, we do not differentiate the concentration–response functions for various species of PM<sub>2.5</sub> in this study.

Earlier epidemiological studies on health effects of particles found little evidence of a threshold level for an exposure–response relation between PM and health effects (Daniels et al. 2000; Krewski et al. 2009; Pope 2000; Schwartz et al. 2002). In this study, we estimate premature mortality due to PM<sub>2.5</sub> and its species without applying a threshold level below which pollutants are assumed to have no health effect.

We use the 2008 population and baseline mortality rates data that have been built into BenMAP. BenMAP uses Census block data to calculate the population level in the population grid cells (Abt Associates 2013). We use the population at the national 12-km grid resolution, and the modeled pollutant concentrations are matched with the population in each 12-km grid cell. Also, population data in BenMAP are stratified by age. We select population aged over 30 in order to be consistent with the Krewski et al. (2009) study from which we draw the risk estimate. To estimate mortality rates, BenMAP uses individual-level mortality data from 2004 to 2006 for the whole USA from the Centers for Disease Control (CDC) and National Center for Health Statistics (NCHS) (Abt Associates 2013).

We calculate mortality associated with individual PM<sub>2.5</sub> species by directly applying the simulated annual average concentrations of each species. Another approach is to calculate mortality for the total PM<sub>2.5</sub> concentration relative to the total PM<sub>2.5</sub> minus one species, as was used in the study by Pungler and West (2013). The main difference in the two methods lies in the fact that the approach of removing a species from the air would suggest how responsive total mortality might be to an intervention strategy focused on a specific particle type, whereas our approach directly refers to the attributable fractions of PM<sub>2.5</sub> mortality associated with different species. However, since the concentration–response curve is nearly linear within the range of PM<sub>2.5</sub> concentrations we consider (0–22 µg/m<sup>3</sup>), we expect the results produced from these two approaches will not differ considerably. In order to verify this assumption, we conducted a sensitivity analysis by calculating the national mortality attributable to each PM<sub>2.5</sub> species at the modeled 0.5 × 0.66° resolution using the Pungler and West approach, and then compared the mortality estimates produced from the two approaches. The results indicate that our approach produces mortality estimates for the six species that are approximately 5–7 % higher than those based on the Pungler and West approach. The detailed results of this analysis are presented in Table 4 in the Appendix.

Using simulated pollutant concentrations, the selected concentration–response coefficient for all-cause mortality, and the built-in population and baseline mortality rates in BenMAP, we calculated the number of excess deaths in the USA in 2008 related to total PM<sub>2.5</sub> and its species. We repeat the calculation using the concentrations at the two model resolutions and at the three regridded resolutions. In estimating the 95 % CI of national mortality estimates, the only source of uncertainty that is taken into account is the uncertainty in the value of the

concentration-response coefficient ( $\beta$ , with a mean value of 0.005827). The uncertainty analysis was conducted in BenMAP using Monte Carlo simulations with a sample size of 5000. In addition to the national mortality estimates, we also calculated mortality by county to investigate how the influence of model resolution on mortality estimates may differ spatially.

## Results and discussions

### Pollutant concentrations at various modeled and re-gridded resolutions

Panels a and b of Fig. 1 show the GEOS-Chem modeled annual average  $PM_{2.5}$  concentrations in 2008 at the fine ( $0.5 \times 0.66^\circ$ ) and coarse ( $2 \times 2.5^\circ$ ) resolution, respectively. The maximum simulated  $PM_{2.5}$  value in the continental USA is 21 and  $17 \mu\text{g}/\text{m}^3$  at the fine and coarse resolution, respectively. The mean  $PM_{2.5}$  values are both approximately  $6 \mu\text{g}/\text{m}^3$  at the fine and coarse resolutions. In general, the highest  $PM_{2.5}$  values occur in the eastern USA. Figure 8 in the Appendix shows the modeled annual average concentrations of the six species in 2008 at the fine and coarse resolutions. For BC,  $\text{NH}_4$ , and NIT, the highest values are located in the northeastern USA. For  $\text{SO}_4$  and OC, the highest values occur in the eastern USA. For DST, these values occur in the southwest border area.

Table 1 presents the mean and maximum values for concentrations of  $PM_{2.5}$  and its six species at various modeled and re-gridded resolutions. For all the species and total  $PM_{2.5}$ , the mean concentrations at various resolutions are similar. On the other hand, the maximum simulated concentrations decrease when going from the fine-model resolution ( $0.5 \times 0.66^\circ$ ) to the coarse resolution ( $2 \times 2.5^\circ$ ). For total  $PM_{2.5}$ , the maximum concentration decreases 21 %. For different species of  $PM_{2.5}$ , the percentage change in maximum concentration from the fine resolution to the coarse resolution varies. The percentage change in the maximum value for organic carbon (61 %) is the greatest, followed by black carbon (31 %), indicating that coarse-model resolutions are likely to cause peak values of primary particulates (mainly BC and OC) to be underestimated, consistent with the findings of Pungler and West (2013). For re-gridded output, the maximum values also decrease as the resolution becomes coarse. Interestingly, the results at  $2 \times 2.5^\circ$  agree rather closely for both the modeled and re-gridded data, suggesting that most of the bias comes from simple spatial averaging.

### Premature mortality due to $PM_{2.5}$ exposure based on model output

As shown in Table 2, using the GEOS-Chem modeled results at the fine resolution, we estimate that approximately 142,000 (95 % CI, 97,500–186,400) all-cause deaths occurred in the USA in 2008 due to ambient  $PM_{2.5}$  exposure. The coarse-model results produce an estimate of all-cause mortality that is 8 % lower than the estimate based on the fine-model results (approximately 130,000 deaths totally, 95 % CI, 89,000–171,000). Figure 2 shows the annual  $PM_{2.5}$ -related mortality at the 12-km resolution calculated using the fine ( $0.5 \times 0.66^\circ$ ) resolution model results. In general, the greatest densities of deaths occur in populated areas in the eastern USA and west coast. Our estimates of national  $PM_{2.5}$  mortality are similar to a recent estimate of national mortality associated with exposure to ambient  $PM_{2.5}$  (Fann et al. 2012), but greater than those of Pungler and West (2013). Using the photochemical Community Multiscale Air Quality (CMAQ) model results at a 12-km

grid resolution in conjunction with ambient monitored data, and the same all-cause mortality risk estimate from Krewski et al. (2009), Fann et al. (2012) estimated 130,000 (95 % CI, 51,000–200,000) PM<sub>2.5</sub>-related deaths to result from 2005 air quality levels, relative to regional PM<sub>2.5</sub> background concentrations (ranging from 0.62 to 1.72 µg/m<sup>3</sup>, depending on the region). Using the same CMAQ model results at a 12-km resolution in 2005, Pungler and West (2013) report a national mortality estimate of 66,000 (95 % CI, 39,300–84,500) due to PM<sub>2.5</sub> exposure, relative to the low-concentration exposure threshold of 5.8 µg/m<sup>3</sup> (Krewski et al. 2009). Since we apply the same concentration–response function from Krewski et al. (2009), differences in national mortality estimates between our study and these other studies are due in part to the selection of threshold (we apply no threshold in our calculation). If a 5.8-µg/m<sup>3</sup> threshold is applied, our estimates of PM<sub>2.5</sub>-related mortality would be 70,000 (95 % CI, 48,000–92,500) at the 0.5 × 0.66° model resolution and 58,000 (95 % CI, 39,500–76,200) at the 2 × 2.5° model resolution. These estimates are close to the Pungler and West (2013) estimates.

Figure 3 compares the mortality estimates attributed to each PM<sub>2.5</sub> species based on model estimates at the fine (0.5 × 0.66°) resolution versus the coarse (2 × 2.5°) resolution. When moving from fine to coarse resolution, mortality estimates due to all species decrease except for dust. Among the five species, mortality estimates due to SO<sub>4</sub> decrease the most (18 %), followed by BC (16 %). For dust, the mortality estimate at the coarse resolution is 38 % higher than that at the fine resolution.

To examine the influence of model resolution on aggregated mortality estimates, we calculated the population-normalized difference in estimated mortality by county between the fine-model resolution and the coarse resolution (Fig. 4; also refer to Fig. 10 in the Appendix for the map that shows the difference in mortality without population normalization). These results show that the coarse resolution tends to significantly underestimate the county-level mortality estimates in large urban centers (counties shown in red in Fig. 4).

In order to further investigate the influence of model resolution on aggregated mortality attributed to different PM<sub>2.5</sub> species, we also compared the maximum death values at the county level based on the two model resolutions. Table 3 compares the difference in the maximum mortality estimates in a county between estimates calculated using model output at the fine and coarse resolutions. For all five species except dust, the maximum mortality estimates decrease from the fine resolution to the coarse resolution. The maximum values for BC and OC decrease much more significantly compared to the values for NH<sub>4</sub>, NIT, and SO<sub>4</sub>. For total PM<sub>2.5</sub> mass, the maximum value, located in Los Angeles County, CA, is 46 % less at the coarse resolution compared to the fine resolution. These results indicate that coarse-model resolutions are likely to cause the greatest density of PM<sub>2.5</sub>-related deaths to be underestimated, particularly deaths due to primary particulates (mainly BC and OC). For dust, the maximum county-level estimate at the fine resolution, located in Maricopa County, AZ, decreases slightly by 3 % at the coarse resolution. However, the mortality estimate in Los Angeles, CA, increases 71 % from the fine to the coarse resolution. The decrease in the estimated mortality for most species can be explained by the decrease in peak PM<sub>2.5</sub> concentrations due to the use of coarser resolution, as these peaks typically occur in



populated urban centers. For dust, however, the change in the estimated mortality shows an opposite sign from the other species. This can be explained by the fact that the sources of dust are in remote areas and hence dust concentrations are largely uncorrelated with population (as shown in Fig. in the Appendix).

### **PM<sub>2.5</sub>-related premature mortality estimates based on re-gridded concentrations**

**Comparing the coarse re-gridded results with the finest model results**—For total PM<sub>2.5</sub>, increasing grid size decreases the estimated mortality (Fig. 5; also refer to Fig. 11 in the Appendix for the original estimates of national all-cause mortality due to PM<sub>2.5</sub> species at various grid resolutions). At the  $1 \times 1.25^\circ$  resolution, total estimated PM<sub>2.5</sub>-related mortality is 4 % lower than the fine resolution ( $0.5 \times 0.66^\circ$ ) estimate; the  $2 \times 2.5^\circ$  and  $4 \times 5^\circ$  resolutions produce mortality estimates that are 7 and 15 % lower than the estimate driven with the fine resolution, respectively. The decrease in total mortality estimates can be explained by the decrease in peak PM<sub>2.5</sub> concentrations, which typically occur in populated regions, due to dilution into a coarser grid cell (Punger and West 2013). As a result of the dilution of peak concentrations in populated areas, coarse resolutions tend to underestimate the high density of deaths occurring in these areas.

For all the five species except dust, the estimated mortality decreases as the resolution becomes coarser. At the coarsest  $4 \times 5^\circ$  resolution, mortality estimate decreases the most for BC (a 32 % decrease relative to the finest model result), followed by OC (a 19 % decrease relative to the finest model result). In general, coarse resolution tends to have the greatest influence on the estimates of national mortality associated with exposure to primary species (mainly BC, and OC contains both primary and secondary particles). Our findings of the effect of grid resolution on primary and secondary species of PM<sub>2.5</sub> are consistent with those of Punger and West (2013), who reported that coarse resolution has the greatest percentage effect on estimates of mortality driven with primary species. For dust, this is only a small change (a 1 % increase) in mortality estimate at the  $4 \times 5^\circ$  resolution relative to the finest resolution.

As shown in Fig. 6, at  $1 \times 1.25$  re-gridded resolution, mortalities in counties where large cities are located (mostly in the eastern USA and on the west coast) are underpredicted, whereas the biases are small in most other counties. The error is the largest in Los Angeles County in California. At  $2 \times 2.5$  resolution, the error (underprediction) becomes even larger in Los Angeles County, whereas the errors in a few counties surrounding Los Angeles County become smaller compared to the  $1 \times 1.25$  resolution. On the other hand, comparing to the  $1 \times 1.25$  resolution, most counties slightly over-predict mortality at  $2 \times 2.5$  resolution. Finally, at  $4 \times 5$  resolution, the error in Los Angeles County is the largest whereas mortality is over-predicted in most counties in the USA to a larger extent.

### **Comparing the modeled and re-gridded results both at the $2 \times 2.5^\circ$ resolution**

—We also compared the model and re-gridded results at the same  $2 \times 2.5^\circ$  resolution. As shown in Table 2, for national mortality estimates, modeled and re-gridded concentrations at the  $2 \times 2.5^\circ$  resolution produce similar results for BC, NH<sub>4</sub>, NIT, and OC. For SO<sub>4</sub>, however, the mortality estimate based on the re-gridded concentrations is 20 % greater than

the estimate based on the modeled concentrations, and for dust, the re-gridded results produce a national mortality estimate that is 27 % less. Overall, the total PM<sub>2.5</sub>-related mortality predicted from the re-gridded output is 2 % higher than that predicted from the modeled 2 × 2.5° resolution output. Although it seems that the re-gridded concentrations at the 2 × 2.5° resolution produce a national PM<sub>2.5</sub>-related mortality estimate that is very close to the estimate based on model output at the same resolution, for different PM<sub>2.5</sub> species, the differences between the two estimates vary.

Figure 7 shows the difference in mortality due to PM<sub>2.5</sub> at the county level between the modeled and re-gridded 2 × 2.5° resolution. It indicates that in the eastern USA and the west coast, the re-gridded output tends to overestimate PM<sub>2.5</sub>-related mortality whereas in the western USA (except for the west coast), the re-gridded results are likely to underestimate PM<sub>2.5</sub> mortality.

## Discussion and conclusions

Using the all-cause mortality risk estimate for PM<sub>2.5</sub> from Krewski et al. (2009), we estimate that approximately 142,000 (95 % CI, 97,500–186,400) all-cause deaths occurred in the USA in 2008 due to ambient PM<sub>2.5</sub> exposure based on the finest GEOS-Chem model resolution at 0.5 × 0.66°. These deaths account for approximately 6 % (95 % CI 4–8 %) of the national deaths occurring in the USA in 2008. The six species of PM<sub>2.5</sub>, namely BC, NH<sub>4</sub>, NIT, SO<sub>4</sub>, OC, and DST, each cause 9, 14, 22, 22, 28, and 5 % of the total PM<sub>2.5</sub> deaths, respectively. Using coarse-model output at 2 × 2.5° produces a national PM<sub>2.5</sub> mortality estimate that is 8 % lower than the estimate based on the fine-model results. Since we only include the USA population aged over 30 in our calculation, we are likely to underestimate the mortality attributable to PM<sub>2.5</sub>, as air pollution likely has adverse health impacts on the population of all ages. The change in mortality estimates when moving from the fine to the coarse-model resolution varies for different species, with the estimates decreasing by 7–18 % for the first five species and increasing by 38 % for DST. Our calculation of the county-level mortality shows that the coarse-model resolution tends to underestimate mortality in counties where large urban centers are located, particularly mortality attributed to primary particulates including BC and OC. On the other hand, deaths due to DST are overestimated by the coarse-model resolution. As we discussed earlier, deaths due to dust are overestimated by the coarse-model resolution mainly owing to the fact that dust concentrations are largely uncorrected with population, whereas concentrations of the other five species tend to be positively corrected with population.

To further explore the effects of coarse resolution on mortality estimates, we scaled modeled concentrations at 0.5 × 0.66° fine resolution to coarser resolution of 1 × 1.25°, 2 × 2.5° and 4 × 5° by simple averaging, a similar approach that was used in Pungert and West (2013). The results show that at the 1 × 1.25° resolution, the total PM<sub>2.5</sub>-related mortality estimate is 4 % lower than the fine resolution (0.5 × 0.66°) estimate, and the 2 × 2.5° and 4 × 5° resolutions produce mortality estimates that are 7 and 15 % lower than the estimate driven with the fine resolution. Furthermore, we found that re-gridded coarse resolutions tend to have the greatest influence (underprediction) on the estimates of national mortality associated with

exposure to primary PM<sub>2.5</sub> species (BC and OC), whereas the least impact on dust-related mortality estimates.

In comparing the 2 × 2.5 coarse-modeled results with the same resolution re-gridded results, we found that although the re-gridded concentrations produce a national PM<sub>2.5</sub>-related mortality estimate that is very close to the estimate based on model output, for different species, the differences between the two estimates vary by up to 30 %. Geographically, in the eastern USA and the west coast, the re-gridded output tends to overestimate PM<sub>2.5</sub>-related mortality whereas in the western USA (except for the west coast), the re-gridded results are likely to underestimate PM<sub>2.5</sub> mortality. Ideally, the 2 × 2.5 resolution model provides estimates of average concentrations within each grid cell, which would equal the aggregated, re-gridded results. The differences we see in practice are owing to model resolution error causing these two things to be different. This could be owing to chemistry and/or meteorology. For instance, meteorology would impact the dust emissions, which are a function of the cube of the surface wind speed, which is highly resolution dependent. Also, chemistry might affect the secondary aerosol species. For example, in regions with lots of nitrate, such as the Los Angeles area, large differences in estimated mortality were found (Fig. 7).

Some recent studies investigated how model resolution might affect the calculation of human health impacts associated with PM<sub>2.5</sub> exposure. Using modeled concentrations of PM<sub>2.5</sub> at the 12-km resolution from CMAQ model and scaling these concentrations to multiple coarser resolutions from 24 to 408 km by simple averaging, Pungler and West (2013) found that coarse grid resolutions produce national mortality estimates in the USA that are substantially biased low. In contrast, a regional study on nine regions in the eastern USA, which calculated the human health impacts associated with changes in PM<sub>2.5</sub> at 36-, 12-, and 4-km resolutions, found that health impacts associated with changes in concentrations were not sensitive to model resolution (Thompson et al. 2014). Given the conflicting results reported by earlier studies, this study further examines the influence of model resolution on health impact assessment by running a global atmospheric model at a fine and a coarse resolution and also scaling the finest modeled concentrations to coarser resolutions. The results suggest that coarse grid resolutions may produce national mortality estimates that are biased low for PM<sub>2.5</sub>. Our findings of the influence of resolution on national PM<sub>2.5</sub>-related mortality estimates are consistent with those of Pungler and West (2013). Furthermore, the results of species-specific mortality calculations indicate that coarse resolutions produce larger bias (underestimation) on the mortality associated with primary particles, and may overestimate the mortality due to dust. Also, our analysis of the spatial variations in mortality shows that coarse-model resolutions are most likely to underpredict mortality estimates in large urban centers, such as the Los Angeles metropolitan area, where the greatest numbers of PM<sub>2.5</sub>-related mortality are predicted.

Despite the uncertainties in our estimates, our findings provide evidence of the bias for quantitative PM<sub>2.5</sub> health impact assessment in applications of global atmospheric models where model resolutions are likely to be restricted by computational power and other factors. Future studies should extend this analysis to larger geographical regions to examine the influence of resolution at a larger scale. Moreover, future studies may consider applying

different concentration-response coefficients to various PM<sub>2.5</sub> species to reflect species potency. Doing so would further differentiate the impacts of model resolution on the estimates of burden of different species on public health.

## Acknowledgments

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## Appendix

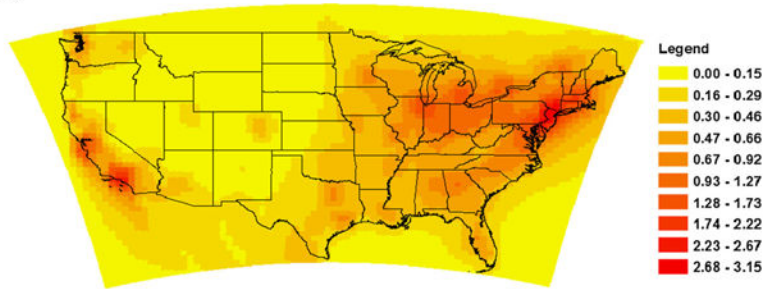
**Table 4**

Comparing national mortality estimates attributable to each PM<sub>2.5</sub> species based on modeled  $0.5 \times 0.66^\circ$  concentrations using two approaches: (1) directly applying the simulated annual average concentrations of each species (approach used in this study) and (2) calculating mortality for the total PM<sub>2.5</sub> concentration relative to the total PM<sub>2.5</sub> minus one species (Punger and West 2013)

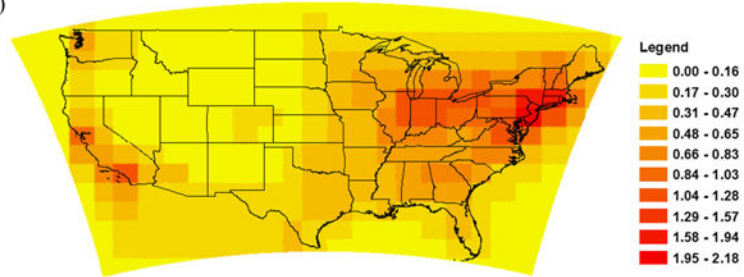
Species	National mortality		Percent difference: (2) relative to (1)
	Approach (1)	Approach (2)	
BC	13,280	12,379	-6.8 %
NH <sub>4</sub>	20,464	19,191	-6.2 %
NIT	32,683	30,864	-5.6 %
SO <sub>4</sub>	32,539	30,772	-5.4 %
OC	40,144	38,064	-5.2 %
DST	7372	6997	-5.1 %
Sum of six species	146,482	138,267	-5.6 %

Table 4 shows that our approach produces mortality estimates for the six species that are slightly higher (approximately 5–7 %) than those based on the Punger and West approach. The total PM<sub>2.5</sub>-related mortality calculated using PM<sub>2.5</sub> concentrations is 142,342 (Table 2). Therefore, when comparing the summary of mortality due to the six species with the total PM<sub>2.5</sub> deaths calculated using PM<sub>2.5</sub> concentrations directly, the estimate based on our approach is 2.9 % higher whereas the estimate based on the Punger and West approach is 2.9 % lower

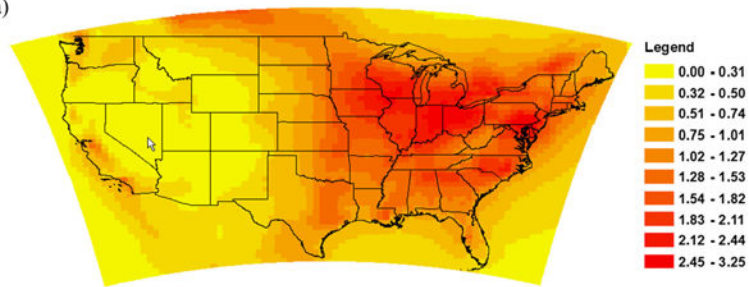
Black carbon  
(a)



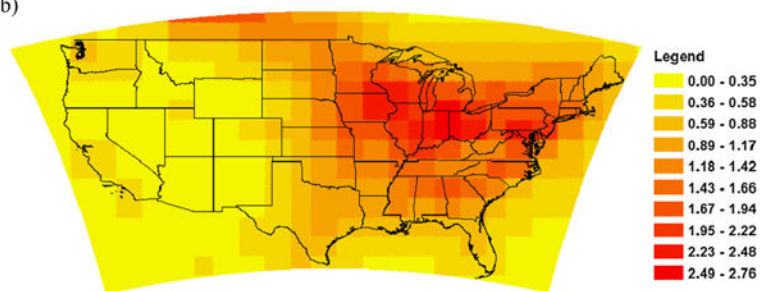
(b)



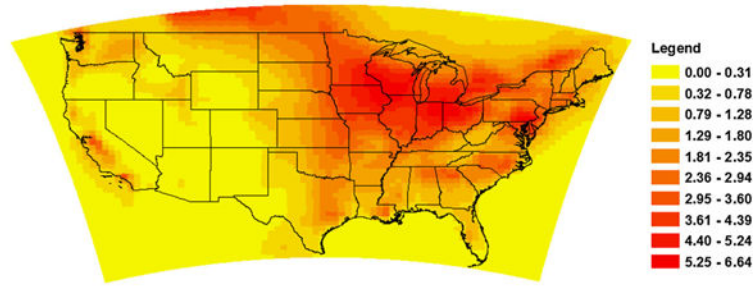
Ammonium  
(a)



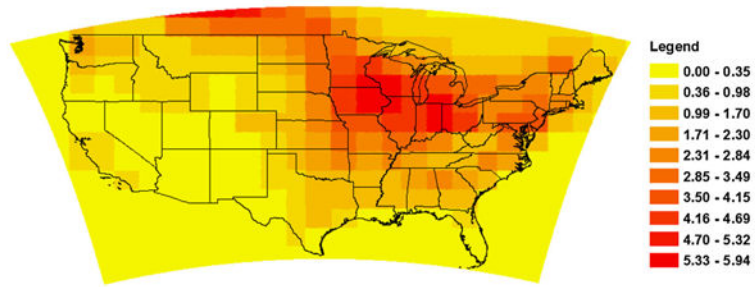
(b)



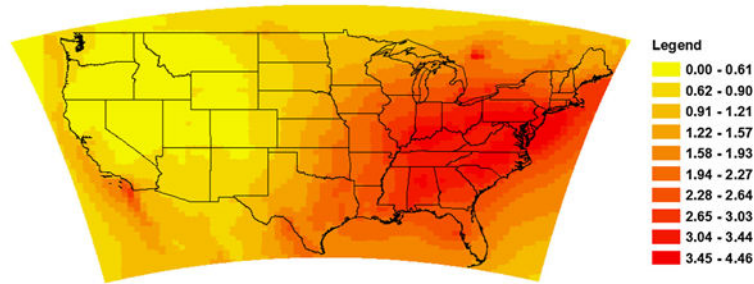
Nitrate  
(a)



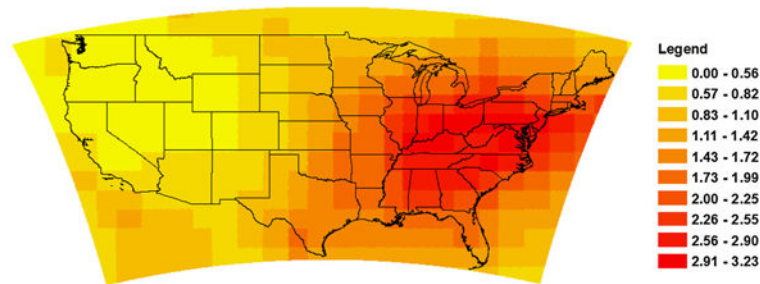
(b)

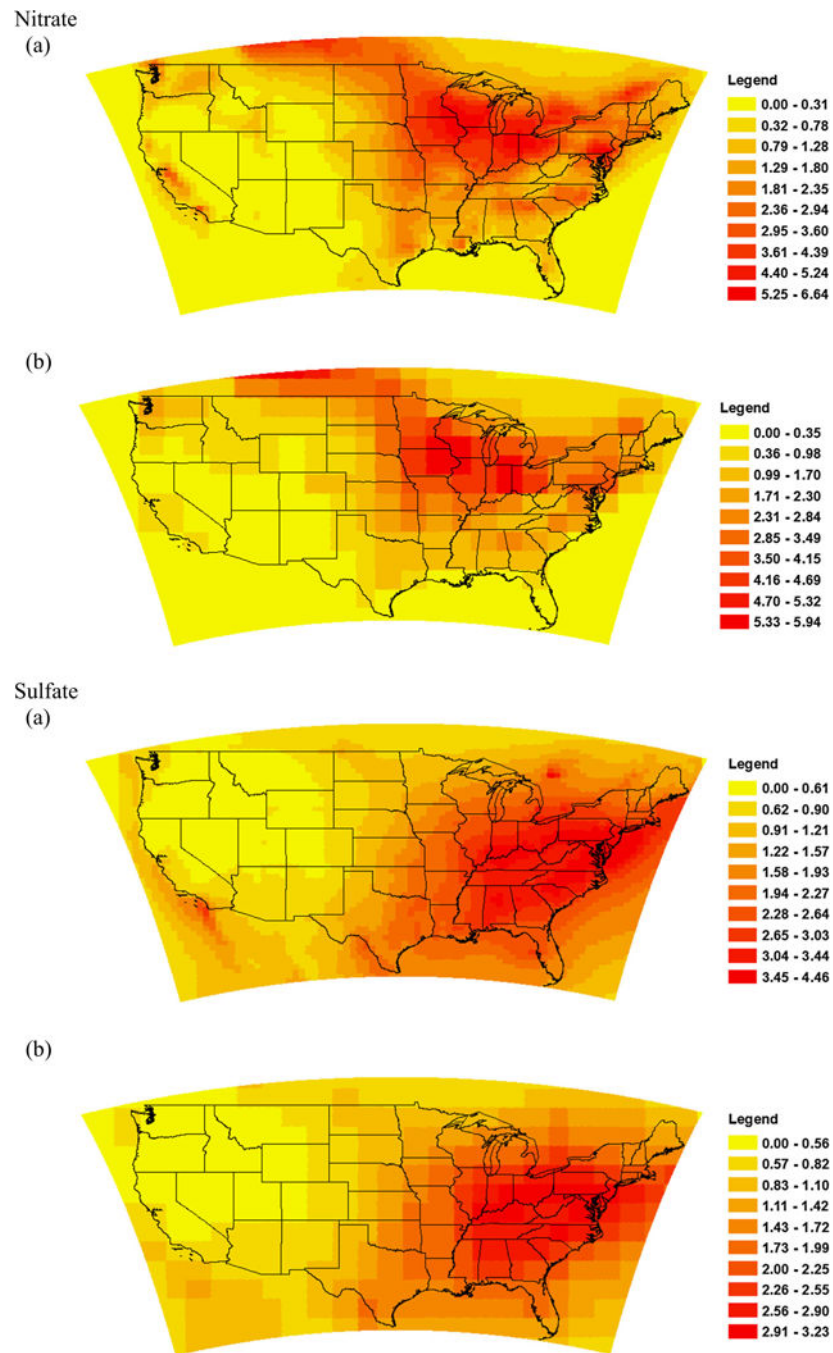


Sulfate  
(a)

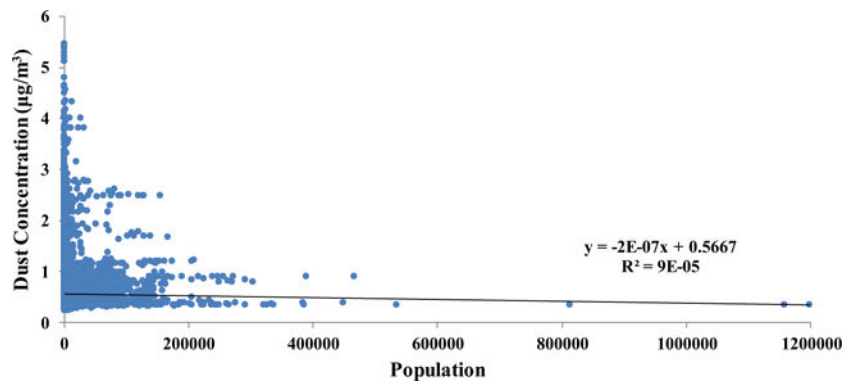


(b)

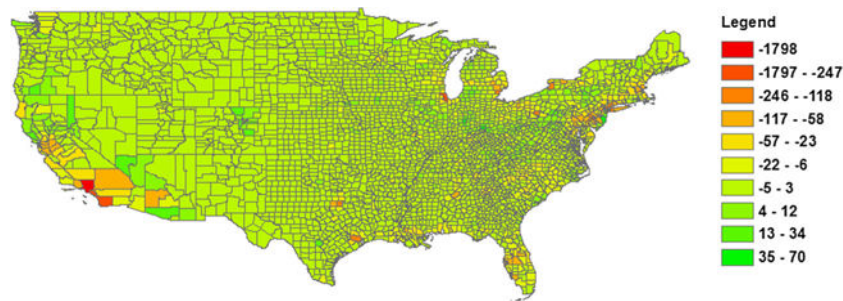




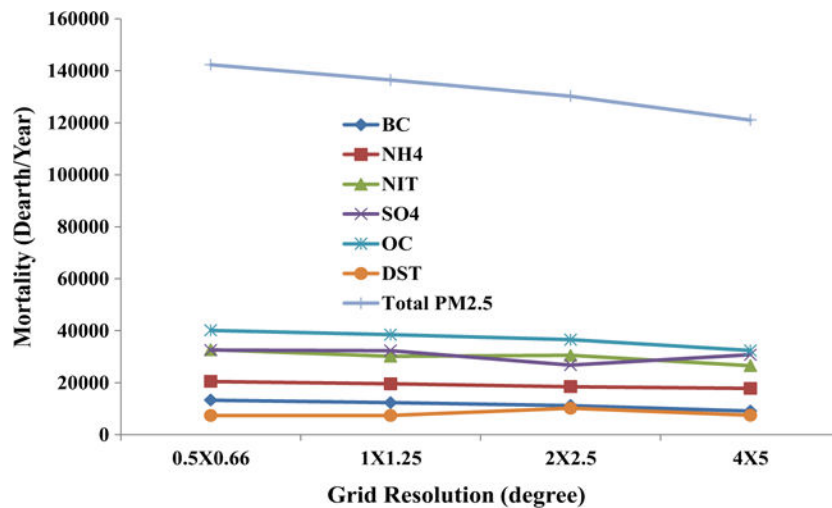
**Fig. 8.** Modeled annual average PM<sub>2.5</sub> species concentrations in 2008 using GEOS-Chem output at **a** fine ( $0.5 \times 0.66^\circ$ ) resolution and **b** coarse ( $2 \times 2.5^\circ$ ) resolution (unit,  $\mu\text{g}/\text{m}^3$ )



**Fig. 9.** Correlation analysis of the dust concentration and population using fine ( $0.5 \times 0.66^\circ$ ) model results



**Fig. 10.** The difference in estimated mortality by county attributed to total  $PM_{2.5}$  between the fine-model resolution ( $0.5 \times 0.66^\circ$ ) and the coarse resolution ( $2 \times 2.5^\circ$ )



**Fig. 11.** a Estimates of national all-cause mortality due to  $PM_{2.5}$  species at various grid resolutions, including the fine-model resolution ( $0.5 \times 0.66^\circ$ ) and three re-gridded resolution ( $1 \times 1.25$ ,  $2 \times 2.5$ , and  $4 \times 5$ )



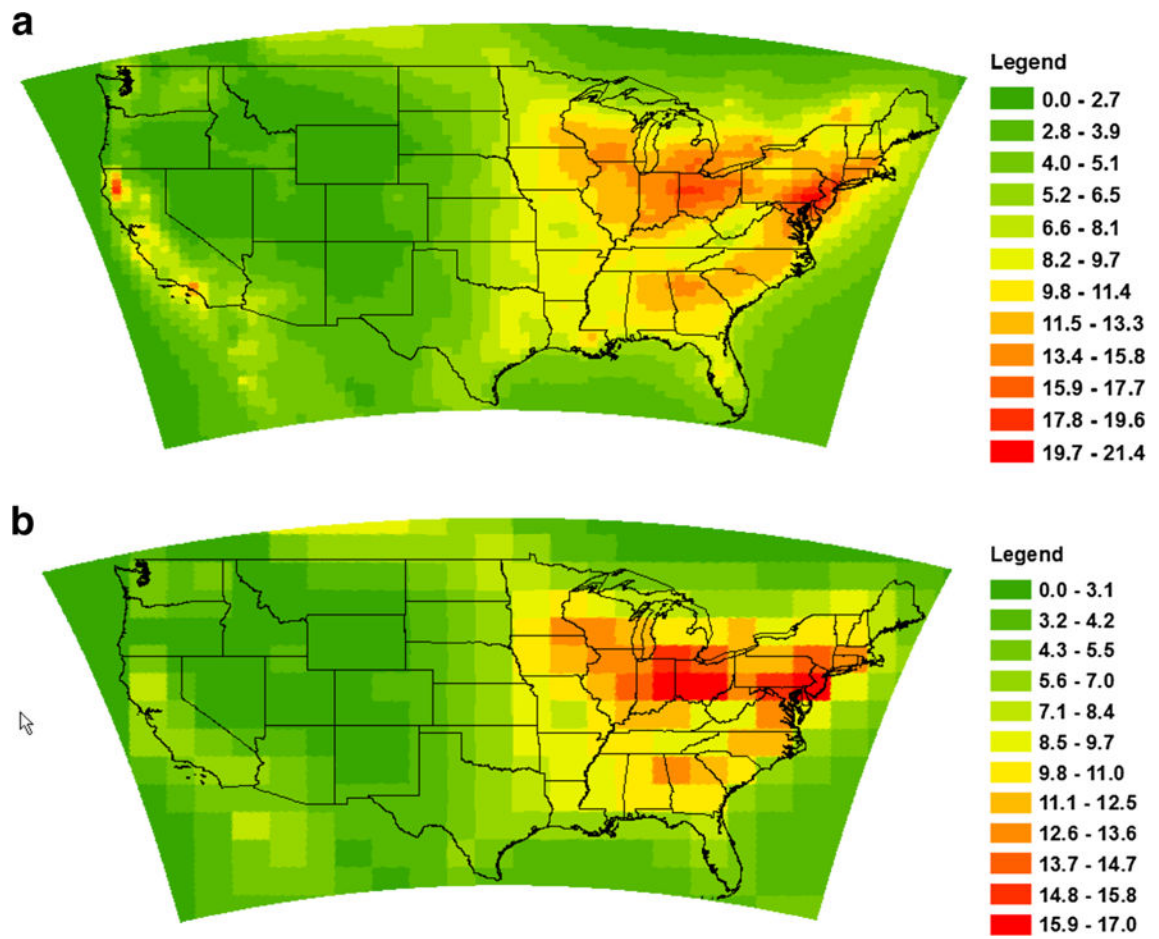
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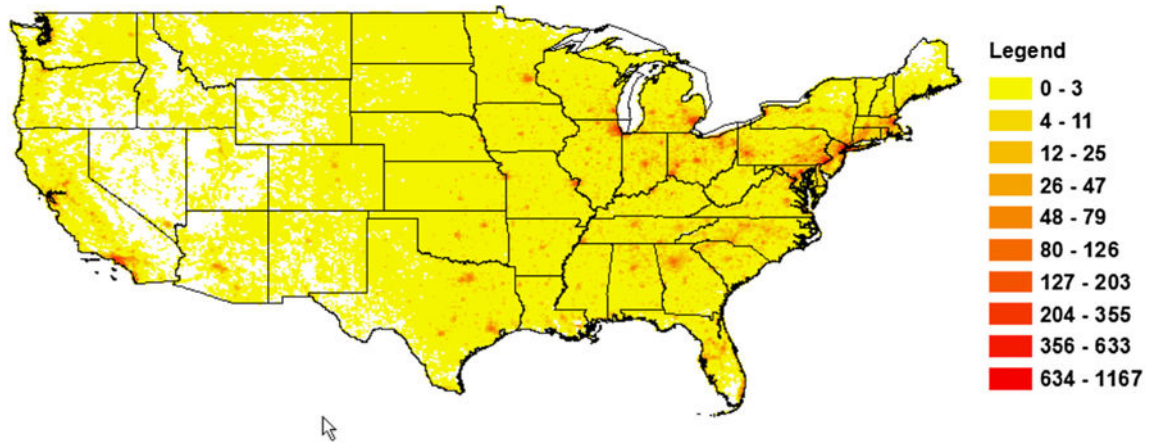
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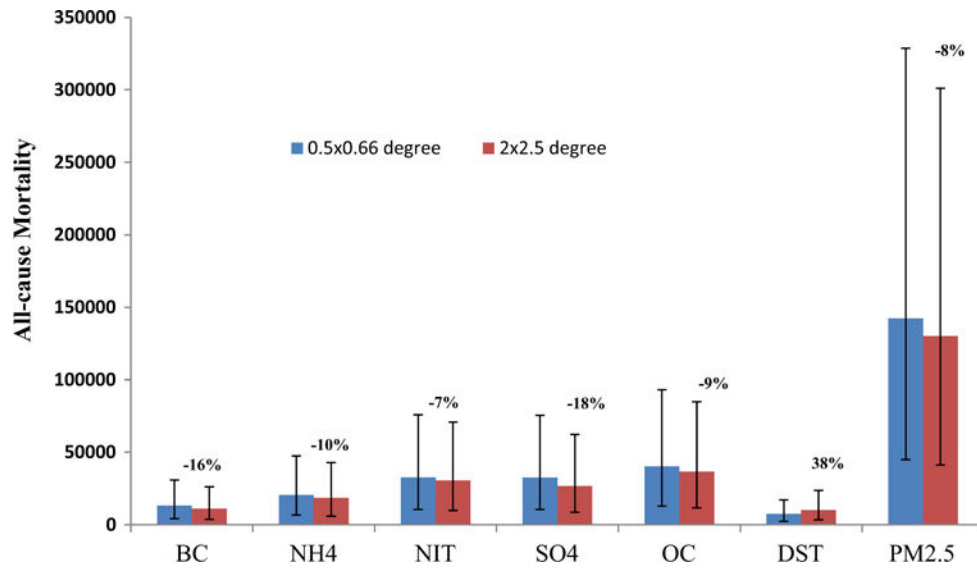
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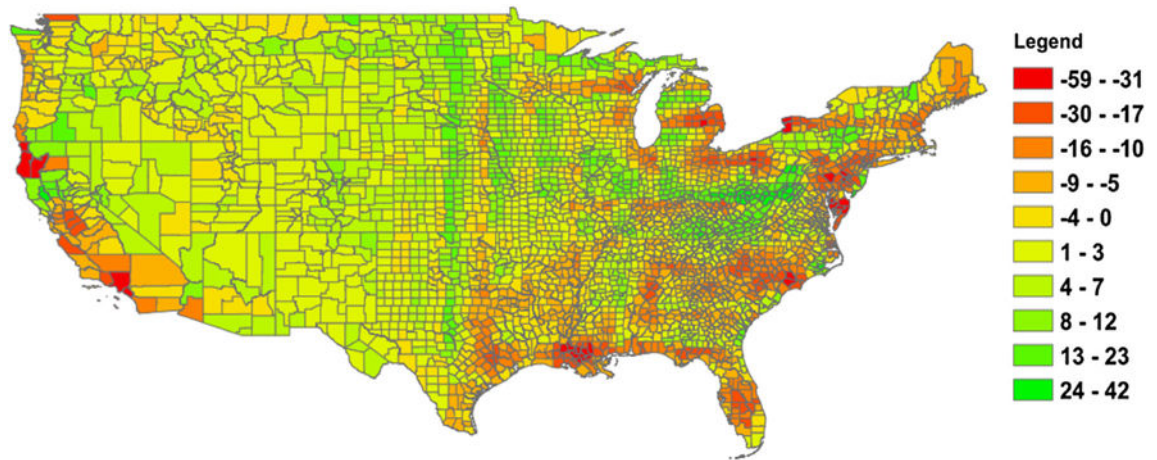
**Fig. 1.** Modeled annual average PM<sub>2.5</sub> concentrations in 2008 using GEOS-Chem output at **a** fine (0.5 × 0.66°) resolution and **b** coarse (2 × 2.5°) resolution (unit, µg/m<sup>3</sup>)



**Fig. 2.** Annual all-cause mortality at 12-km resolution due to total  $PM_{2.5}$  in 2008 estimated using GEOS-Chem output at the fine ( $0.5 \times 0.66^\circ$ ) resolution (deaths per year)

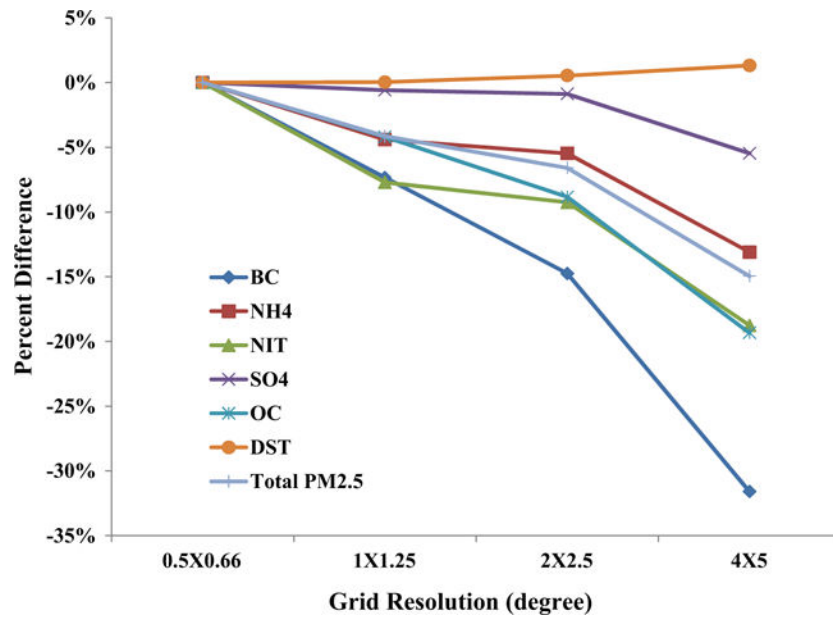


**Fig. 3.** Total mortality in the USA in 2008 attributed to each species, fine versus coarse resolution (*Error bars* show the 95 % CI; percent values shown are percentage changes relative to the estimates at the fine resolution)

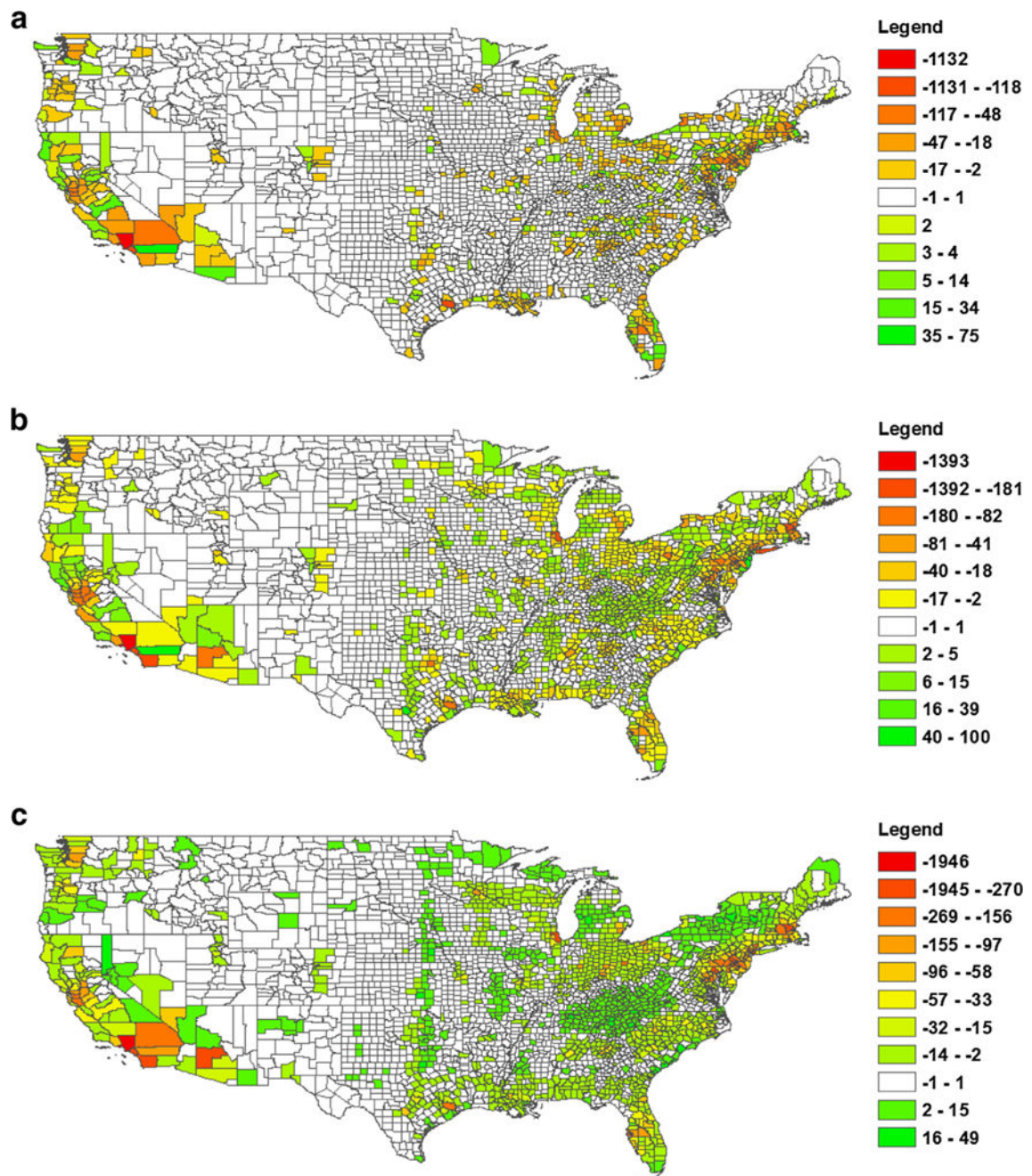


**Fig. 4.** The population-normalized difference in estimated mortality by county attributed to total  $PM_{2.5}$  between the fine-model resolution ( $0.5 \times 0.66^\circ$ ) and the coarse resolution ( $2 \times 2.5^\circ$ ) (calculated as the coarse resolution estimate minus the fine resolution estimate, divided by population; shown as difference in mortality per 100,000 people)

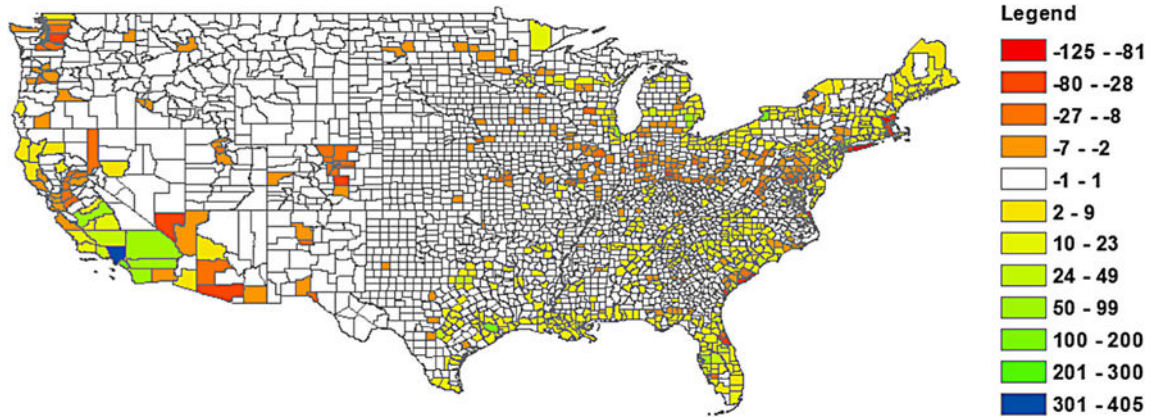




**Fig. 5.** Percent difference in estimates of national all-cause mortality due to PM<sub>2.5</sub> species between the re-gridded resolution and the fine-model resolution, calculated as (re-gridded, coarse results-fine-model results)/fine-model results



**Fig. 6.** Difference in the estimated all-cause deaths by county attributable to  $PM_{2.5}$  exposure in 2008 due to re-gridded coarse resolution, calculated as **a** the  $1 \times 1.25$  estimates minus the  $0.5 \times 0.66$  estimates; **b** the  $2 \times 2.5$  estimates minus the  $0.5 \times 0.66$  estimates; and **c** the  $4 \times 5$  estimates minus the  $0.5 \times 0.66$  estimates (counties with the difference falling into the range of  $[-1, 1]$  are shown in *white color*)



**Fig. 7.**  
The difference in estimated mortality by county attributed to total PM<sub>2.5</sub> between the 2 × 2.5 model and 2 × 2.5 re-gridded resolution

Mean and maximum simulated concentrations of PM<sub>2.5</sub> and its six components at various modeled ( $0.5 \times 0.66$  and  $2 \times 2.5^\circ$ ) and re-gridded ( $1 \times 1.25$ ,  $2 \times 2.5$ , and  $4 \times 5^\circ$ ) resolutions over the continental USA (year, 2008; unit,  $\mu\text{g}/\text{m}^3$ )

**Table 1**

Species	Modeled output						Re-gridded output					
	$0.5 \times 0.66^\circ$		$2 \times 2.5^\circ$		% change of the max value		$1 \times 1.25^\circ$		$2 \times 2.5^\circ$		$4 \times 5^\circ$	
	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max
BC	0.32	3.2	0.32	2.2	-31 %	0.32	2.8	0.32	2.3	0.32	1.5	
NH <sub>4</sub>	0.8	3.2	0.8	2.8	-13 %	0.8	2.8	0.8	2.6	0.8	2.3	
NIT	1.1	6.6	1.1	5.9	-11 %	1.1	5.8	1.1	5.4	1.1	5.0	
SO <sub>4</sub>	1.5	4.5	1.3	3.2	-29 %	1.5	3.9	1.5	3.7	1.5	3.5	
OC	1.3	14.6	1.3	5.7	-61 %	1.3	7.9	1.3	5.7	1.3	4.0	
DST	0.69	6.1	0.96	5.8	-5 %	0.69	4.6	0.69	3.0	0.69	2.4	
PM <sub>2.5</sub>	5.7	21.4	5.8	17	-21 %	5.7	18.7	5.7	17.3	5.7	14.5	

**Table 2**

National mortality in the USA in 2008 attributed to PM<sub>2.5</sub> exposure estimated using various modeled and re-gridded resolutions

Species	Modeled output			Re-gridded output								
	0.5×0.66°			2×2.5°		1×1.25°		2×2.5°		4×5°		
	Mean	95 % CI		Mean	95 % CI	Mean	95 % CI	Mean	95 % CI	Mean	95 % CI	
BC	13,280	8993–17,557		11,211	7590–14,827		12,305	8331–16,271	11,320	7664–14,970	9082	6147–12,014
NH <sub>4</sub>	20,464	13,862–27,048		18,471	12,511–24,417		19,560	13,250–25,856	19,343	13,102–25,568	17,782	12,043–23,507
NIT	32,683	22,175–43,140		30,535	20,717–40,305		30,163	20,461–39,820	29,659	20,119–39,156	26,548	18,005–35,055
SO <sub>4</sub>	32,539	22,065–42,972		26,795	18,161–35,400		32,348	21,935–42,720	32,252	21,870–42,593	30,761	20,856–40,629
OC	40,144	27,246–52,974		36,540	24,790–48,235		38,459	26,098–50,760	36,593	24,827–48,304	32,382	21,960–42,761
DST	7372	4988–9753		10,177	6888–13,461		7374	4990–9756	7411	5015–9804	7469	5054–9880
PM <sub>2.5</sub> total	142,342	97,463–186,431		130,249	89,089–170,746		136,433	93,362–178,782	132,955	90,963–174,256	121,050	82,725–158,804

**Table 3**

Maximum mortality estimate at the county level attributed to PM<sub>2.5</sub> and its six species, calculated using modeled concentrations at  $0.5 \times 0.66^\circ$  and  $2 \times 2.5^\circ$  over the continental USA

Species	<u>Maximum mortality estimate at the county level</u>		County	% change of the maximum value
	$0.5 \times 0.66^\circ$	$2 \times 2.5^\circ$		
BC	812	382	Los Angeles, CA	-53 %
NH <sub>4</sub>	492	460	Cook, IL	-7%
NIT	1039	932	Cook, IL	-10 %
SO <sub>4</sub>	533	516	Cook, IL	-3%
OC	1425	787	Los Angeles, CA	-45 %
DST	349 (max value)	339	Maricopa, AZ	-3%
	289	494	Los Angeles, CA	71 %
PM <sub>2.5</sub>	3935	2137	Los Angeles, CA	-46 %