PREMATURE MORTALITIES ATTRIBUTABLE TO OZONE AND FINE PARTICULATE EXPOSURE: THE EFFECT OF GRID SIZE ON HEALTH BURDEN ESTIMATES IN THE UNITED STATES

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ABSTRACT

ELIZABETH BLAYNEY: Premature Mortalities Attributable to Ozone and Fine Particulate Exposure: The Effect of Grid Size on Health Burden Estimates in the United States
(Under the direction of J. Jason West)

We quantify how estimates of mortality in the United States attributable to ozone (O_3) and fine particulate matter ($PM_{2.5}$) driven with modeled concentrations at coarse resolution differ from those at finer resolution. Modeled concentrations of O_3 and $PM_{2.5}$ were used to estimate mortalities at 12 km and coarser resolutions greater than that of global models. We estimate that 66,000 (95% CI, 44,700 – 86,500) and 21,400 (5,600 – 34,200) mortalities per year are attributable to $PM_{2.5}$ and O_3 concentrations above low-concentration thresholds, respectively. Coarse grid resolutions produce mortality estimates that are substantially biased low for $PM_{2.5}$ (38% lower than the best estimate at >300 km resolution), but only 5% higher for O_3 (at >96 km resolution). Mortality estimates for primary $PM_{2.5}$ species were more affected by grid resolution than for secondary species. These results suggest that using coarse resolution global models (>100 km) are likely biased low for $PM_{2.5}$, but with little error for ozone.

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LIST OF ABBREVIATIONS AND SYMBOLS

ACS	American Cancer Society
AQS AIRS	Air Quality System (AQS) Aerometric Information Retrieval System (AIRS)
BenMAP	The Environmental Benefits Mapping & Analysis Program
CI	Confidence interval
CMAQ	Community Multiscale Air Quality model
CPD	Cardiopulmonary disease
CRF	Concentration-response function
EC	Elemental carbon
EPA	U.S. Environmental Protection Agency
GIS	Geographic Information System
IMPROVE	Interagency Monitoring of Protected Visual Environments
NH_4	Ammonium ion
$(NH_4)_2SO_4$	Ammonium sulfate
NH_4NO_3	Ammonium nitrate
NO_3	Nitrate aerosols
O_3	Ozone
OC	Organic carbon
OTHR	Other $PM_{2.5}$ species
$PM_{2.5}$	Particulate matter finer than 2.5 microns
ppb	Parts per billion
SD	Standard deviation
SO_4	Sulfate aerosols
STN	Speciation Trends Network

CHAPTER 1: INTRODUCTION

The negative health effects of ozone (O_3) and fine particulate matter ($PM_{2.5}$) are well documented. Acute exposure to these pollutants damages airways and leads to decreased lung function (Broeckaert et al., 1999). Epidemiology studies build upon toxicology results to demonstrate the link between population exposure and adverse health outcomes. $PM_{2.5}$ and ozone exposures are correlated with short-term and long-term mortality (Bell et al., 2004; Jerrett et al., 2009; Krewski et al., 2009).

Previous health impact analyses have evaluated changes in health endpoints resulting from changes in pollutant concentrations modeled with air quality models, but both measurement and modeling provide viable data for impact assessment (Anenberg et al., 2010; Fann et al., 2011a; West et al., 2006). However, atmospheric models provide concentration data that is more spatially and temporally complete than monitoring data. Globally, air quality monitors are often located in urban areas and pollutant concentrations in rural areas must be inferred using other methods (Cohen et al., 2004). Consequently, extrapolation of measurement data to estimate exposure concentrations in unmonitored areas may introduce substantial error into impact analysis as pollutant concentrations change across space and time (Jerrret et al., 2005).

Where atmospheric models drive health impact analysis, grid cell resolution is limited by computational speed. The U.S. Environmental Protection Agency (EPA) often employs model resolutions of 12 km or 36 km to assess the impact of proposed regulation on the

health burden attributable to a pollutant in the United States (US) (Fann et al., 2011b; Hubbell et al., 2005, 2009; U.S. EPA, 2007). To assess the impact of air pollution on a larger scale, global atmospheric models (with resolutions of approximately 200 km) are used to estimate the total burden of disease due to anthropogenic air pollution and the effect of changing emissions on mortality (Anenberg et al., 2010; Corbett et al., 2007; West et al., 2006). However, health burden estimations on a global scale are limited in their ability to capture concentration and population gradients around populous areas with a coarse resolution (Bader et al., 2008).

The grid resolution of atmospheric models can influence predicted pollutant concentrations, as well as the modeled exposure of populations, especially near urban areas where concentrations and populations have strong spatial gradients. The effect of resolution on model chemistry in small domains is well studied for ozone (Liang and Jacobson, 2000; Cohen et al., 2006), but not for $PM_{2.5}$. Previous studies to evaluate the effect of O_3 model resolution on health impact assessments evaluate the effect of resolution on exposure for grids sized 36 km and smaller (Thompson and Selin, in review). We are not aware of work to quantify the effect of resolution on concentration estimates or population exposure to $PM_{2.5}$. The bias due to coarse resolution has not been quantified for $PM_{2.5}$ or O_3 at the resolutions used in global models.

The bias due to coarse resolution may be different for different pollutants, as pollutants with different lifetimes affect concentrations over different spatial scales. Ozone has a sufficiently long atmospheric lifetime, approximately 23 days in the northern hemisphere (Seinfeld and Pandis, 2006), to travel from precursor emissions sources and elevate concentrations in rural areas. $PM_{2.5}$ is composed of many individual particulate

species with distinct atmospheric lifetimes ranging from days to weeks (Seinfeld and Pandis, 2006). Primary particulates, such as elemental carbon (EC), crustal material, and a fraction of organic carbon (OC), are emitted directly from their sources and have small dispersal ranges. Secondary particles, such as ammonium sulfate ($(NH_4)_2SO_4$), ammonium nitrate (NH_4NO_3), and some organic aerosols, form from precursors and can travel far from the precursor emission sources (Greco et al., 2007). Secondary particulate matter is thought to be minimally affected by coarse model resolutions, while dilution into larger grid sizes may cause peak values of primary particulates to be underestimated (U.S. EPA, 2007). Pollutants with short atmospheric lifetimes, and therefore small dispersal ranges, are not well captured by coarse resolution models (U.S. EPA, 2007). Therefore, we expect that biases due to coarse resolution may be greater for primary $PM_{2.5}$ species than for O_3 or secondary $PM_{2.5}$ species.

This research aims to quantify the effect model grid size has on estimates of human mortality attributable to air pollution in the United States. Explicitly, we explore how estimates of human mortality driven with coarse resolution (<12 km) concentrations of ozone, $PM_{2.5,}$ and the species of $PM_{2.5}$ differ from results produced with finer resolution (12 km) conducted in the same geographical area. In doing so, we estimate the total burden of mortality due to exposure to $PM_{2.5}$ and O_3 in the US. We simulate coarse resolution by scaling modeled concentrations at fine resolution. Therefore, this analysis only accounts for the effect that resolution has on exposure, and does not fully account for the effect of resolution on model chemistry. This work addresses important unresolved uncertainties from studies conducted using global chemical transport models to assess health impacts (e.g., Anenberg et al., 2010).

CHAPTER 2: METHODOLOGY

2.1 Experimental Design

We first estimate the number of yearly mortalities attributable to $PM_{2.5}$ and O_3 concentrations in the continental United States by conducting a health impact assessment at fine resolution. Pollutant concentrations are related to premature mortality using concentration-response functions derived from epidemiology studies. We then evaluate the effect of grid cell resolution on mortality by artificially scaling fine resolution modeled concentrations to coarser resolution and repeating the health impact assessment. We compare the health burden results obtained using the coarser pollutant concentrations to the finest scale to understand the bias introduced by changing grid cell resolution.

2.2 Pollutant Concentrations

Concentrations for ozone, $PM_{2.5}$, and species of $PM_{2.5}$ were obtained from the Community Multiscale Air Quality (CMAQ) model version 4.7.1 from the EPA analysis of the Light-Duty Vehicle Greenhouse Gas Final Rule (U.S. EPA, 2010), which used 2005 estimated meteorology and emissions. A formal model evaluation concluded that total $PM_{2.5}$ concentration is underestimated when compared to annual average measurements (mean bias = -4.65% compared to IMPROVE monitors, -1.7% compared to STN monitors), although some species of $PM_{2.5}$ are underestimated in some regions but overestimated in others. Daily one-hour maximum ozone concentrations were slightly underestimated (mean bias = -1.65% compared to AQS AIRS monitoring sites) in most regions of the United States with the best performance in the southeast (U.S. EPA, 2010). Output from the model was provided for 36

km horizontal resolution over the whole US and 12 km horizontal resolution for eastern and western domains of the US. The two 12 km domains were merged onto a larger grid and, in grid cells where the two domains overlapped, the concentration was calculated as the simple average of the two modeled concentrations. Only the first vertical layer, extending approximately 38 m from ground level, was used for analysis to simulate ground-level exposure.

Modeled concentrations were reported hourly for numerous pollutants including O_3, total $PM_{2.5}$, and several species of $PM_{2.5}$. From the hourly data, we computed metrics consistent with the concentration-response functions used in our health impact analysis. For O_3, we calculated the average one-hour daily maximum values for the ozone season of April through September. We calculated the annual average concentration for $PM_{2.5}$ and each $PM_{2.5}$ species. To evaluate the health effects of specific species of $PM_{2.5}$, we focused on individual species that constitute most of total $PM_{2.5}$: ammonium, sulfate, nitrate, elemental carbon, organic carbon, and "other" $PM_{2.5}$, which is defined as unspecified anthropogenic materials and dusts (Binkowski and Roselle, 2003). Although ammonium, sulfate, and nitrate were presented separately in the $PM_{2.5}$ concentration data, these compounds occur in particulate matter predominantly as ammonium sulfate and ammonium nitrate. We assumed that all of the ammonium neutralizes sulfate and nitrate (Blanchard et al., 2000), and apportioned the ammonium between these species. The fraction of total ammonium in each grid cell apportioned to ammonium nitrate was calculated as $[NO_3^-] / ([NO_3^-] + 2[SO_4^{2-}])$, and the fraction apportioned to ammonium sulfate was $2[SO_4^{2-}] / ([NO_3^-] + 2[SO_4^{2-}])$.

2.3 Creating Coarse Grid Resolutions

Coarse grid resolutions were created at resolutions from 24 km to 408 km, in 12 km increments, using the Lambert Conformal Conic projection, consistent with the CMAQ model. The concentration of a coarser grid cell was computed as the simple average of the 12 km cell concentrations contained within the larger cell. Every 12 km cell was completely contained within a larger cell, and no cells were split. A health impact assessment was conducted at each coarse resolution.

Coarse resolution introduces uncertainty into health impact assessments as the ability to capture population and concentration gradients become limited ("exposure"). In addition, coarse resolution in chemical transport models can limit the ability of the model to resolve chemical processes important for secondary pollutants ("chemistry"). In this analysis, we examine only the effect of model resolution on exposure by scaling fine resolution data. Specifically, as grid cell size increased, chemistry was not changed because we used the pollutant concentrations estimated by the model at 12 km resolution scaled to coarser resolution. However, we also used the 36 km output from the CMAQ model to calculate mortalities. Comparing the CMAQ 36 km results with the results at 12 km demonstrates the combined effect of resolution on model chemistry and on exposure, while the scaled estimates illustrate only the effect of resolution on exposure.

2.4 Health Impact Assessment

Mortality was estimated using The Environmental Benefits Mapping & Analysis Program's (BenMAP) version 4.0.44 (Abt Associates, 2010). BenMAP estimates health endpoints based on concentration-response functions and modeled changes in concentration

between a base and perturbation case. Concentration-response functions were obtained from Krewski et al. (2009) for $PM_{2.5}$ and Jerrett et al. (2009) for O_3, independent analyses of the American Cancer Society (ACS) cohort study of adults 30 years of age and older. These studies were selected because they relate mortality to long-term pollutant exposure. While the evidence relating long-term exposure to $PM_{2.5}$ and mortality is strong (Pope et al., 2002; Laden et al, 2006; Ostro et al., 2007), the relationship between long-term exposure to O_3 and mortality is not as well established. However, the relationship between ozone exposure and mortality has been consistently demonstrated in short-term mortality studies for a range of exposure concentrations (Bell et al., 2004, 2006; Ito et al., 2005).

Krewski et al. (2009) reported risk ratios for $PM_{2.5}$ exposure based on a 1999-2000 random-effects Cox model adjusted for seven ecological and 44 individual covariates. This model predicted risk ratios of 1.06 (95% CI, 1.04 – 1.08), 1.13 (95% CI, 1.10 – 1.16), 1.24 (95% CI, 1.19 – 1.29) and 1.14 (95% CI, 1.06 – 1.23) for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration for all-cause, cardiopulmonary disease (CPD), ischemic heart disease, and lung cancer mortalities, respectively. We used the risk ratios for total $PM_{2.5}$ to evaluate the burden of disease from specific species of $PM_{2.5}$, although some studies have suggested that certain species may have a stronger relationship with mortality (Ostro et al., 2007). For ozone, the relative risk of respiratory mortality, using a two-pollutant model with $PM_{2.5}$ as a co-pollutant, was 1.04 (95% CI, 1.13 – 1.67) for a 10 ppb increase in O_3 concentration (Jerrett et al., 2009). We quantified cause-specific mortalities relative to low-concentration exposure thresholds, defined as the lowest measured values in these studies, of 5.8 $\mu g/m^3$ for $PM_{2.5}$ (Krewski et al., 2009) and 33.3 ppb for O_3 (Jerrett et al., 2009). $PM_{2.5}$ and O_3 concentrations below these thresholds were assumed to have no health impact, although there is no clear

confirmation of a threshold below which no health effect is observed (Ostro, 2004; Bell et al., 2006). For individual $PM_{2.5}$ species, the mortality burden was calculated as the total $PM_{2.5}$ concentration relative to the concentration of $PM_{2.5}$ total with the concentration of the species removed. We did not apply a low-concentration exposure threshold to the estimates of mortality from $PM_{2.5}$ species because some species concentrations were large enough that total minus species was frequently below the threshold.

Population (Figure A.1) and baseline mortality rates from 2005 are built into BenMAP and a spatial-weighting algorithm assigns them to the grid at the resolution of interest (Woods and Poole Economics, Inc., 2001; Abt Associates, Inc., 2009). To remain consistent with the ACS population used to derive the concentration response functions, only those age 30 years and older were considered as the exposed population. County-level baseline cause-specific mortality incidence rates from 2005 (Figure A.2) are preloaded into BenMAP from the Center for Disease Control (CDC), National Center for Health Statistics (CDC, 2009; Abt Associates, Inc., 2009).

We estimate the yearly number of cause-specific mortalities that occur in the US population older than 30 due to $PM_{2.5}$ or O_3 concentrations above exposure thresholds. Results at 12 km resolution are considered the best estimate of annual mortalities, as this is the finest resolution available from the model. Total predicted mortalities at a coarse resolution are evaluated relative to total predicted mortalities from the 12 km resolution.

CHAPTER 3: RESULTS AND DISCUSSION

3.1 Air Quality at Various Resolutions

Figures 3.1 and 3.2 show the geographic distribution of 6-month mean daily one hour maximum ozone and annual average $PM_{2.5}$ for the continental US at a 12 km resolution using the CMAQ output. In general, the highest $PM_{2.5}$ values occur in the eastern United States. Ozone concentration is more geographically uniform, but peak values occur in southern California. Table 3.1 provides the mean, maximum, and standard deviation of all concentration values for O_3 and $PM_{2.5}$ in the cells that overlap the continental US for various grid cell resolutions. The small drop in average concentrations of both O_3 and $PM_{2.5}$ is due to the averaging of modeled concentrations at a 12 km resolution with areas beyond the modeling domain that tend to have lower concentrations. For $PM_{2.5}$, the maximum concentration drops 68% from the fine resolution of 12 km to the coarsest resolution of 408 km. Maximum ozone concentration at 408 km is 25% lower than the concentration at the finest resolution. O_3, with its longer atmospheric lifetime, is more even across domains and less affected by increasing cell size.

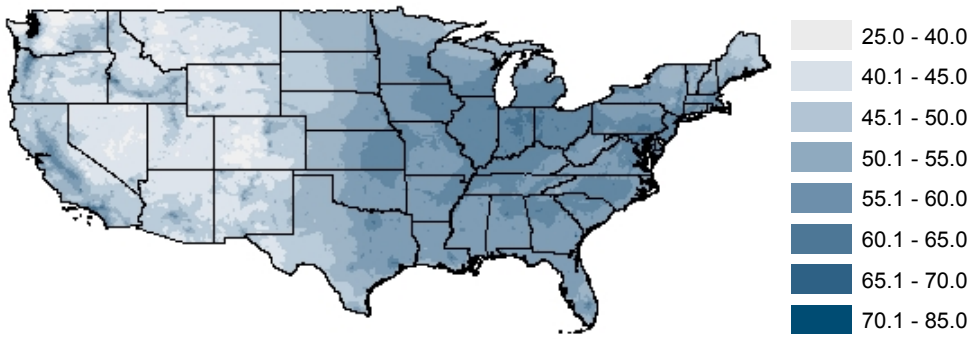

Figure 3.1: Six month average from April to September of 1-hour daily maximum ozone concentrations (ppb) derived from CMAQ output.

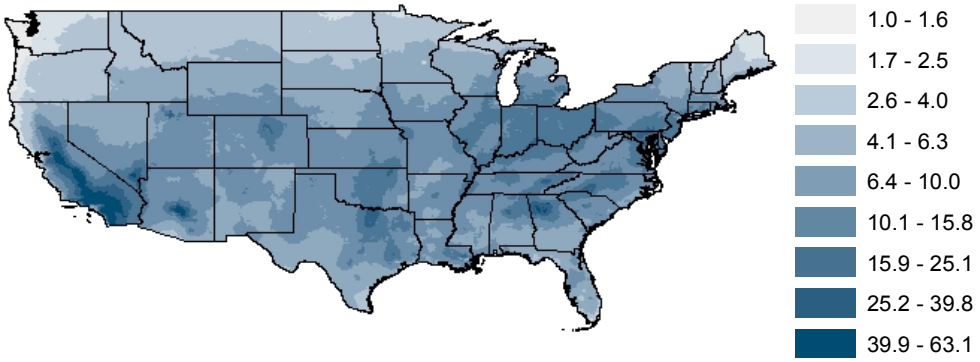

Figure 3.2: Annual average total $PM_{2.5}$ concentration ($\mu g/m^3$) derived from CMAQ output.

Table 3.1: Mean, maximum, and standard deviations of concentrations of $PM_{2.5}$ and O_3 in the cells that intersect the continental US, for various resolutions.

Resolution (km)	O_3 (ppb)			$PM_{2.5}$ ($\mu g/m^3$)		
	Mean	Max	St. Dev	Mean	Max	St. Dev
12	49.26	77.12	5.57	5.68	43.14	3.76
36	49.22	74.51	5.60	5.63	18.75	3.69
CMAQ 36 km	48.26	81.96	9.68	4.12	36.31	3.44
96	48.98	68.75	5.69	5.53	15.72	3.56
144	48.73	65.61	5.77	5.42	15.44	3.47
192	48.49	62.98	5.81	5.85	15.12	3.39
300	47.89	59.89	5.95	5.11	14.11	3.22
408	47.11	57.60	6.38	4.97	13.66	3.13

3.2 Premature Mortalities Due to Ozone

Using the finest resolution, we estimate 21,400 (95% CI, 5,600 – 34,200) annual respiratory mortalities from simulated 2005 O_3 concentrations (Figure 3.3). In this study, the 95% confidence intervals represent only the uncertainty in the concentration-response function. Other sources of uncertainty are discussed later. These results are comparable with those from other studies. Fann et al. (2011a) estimated that 19,000 (95% CI, 7,600 –29,000) respiratory mortalities occur each year in the US due to 2005 simulated maximum 8-hour ozone concentrations above modeled natural background levels. The highest density of mortalities from ozone concentrations in the Fann et al. study also occurred in southern California and in the eastern US. Anenberg et al. (2010) estimated that 25,000 (1 SD, 10,000 – 40,000) mortalities occur in North America due to present day (2000) ozone concentrations above preindustrial levels with low-concentration thresholds applied. Results from the Anenberg et al. study are higher than our estimates due to a larger population size and different present day concentrations simulated using different atmospheric models.

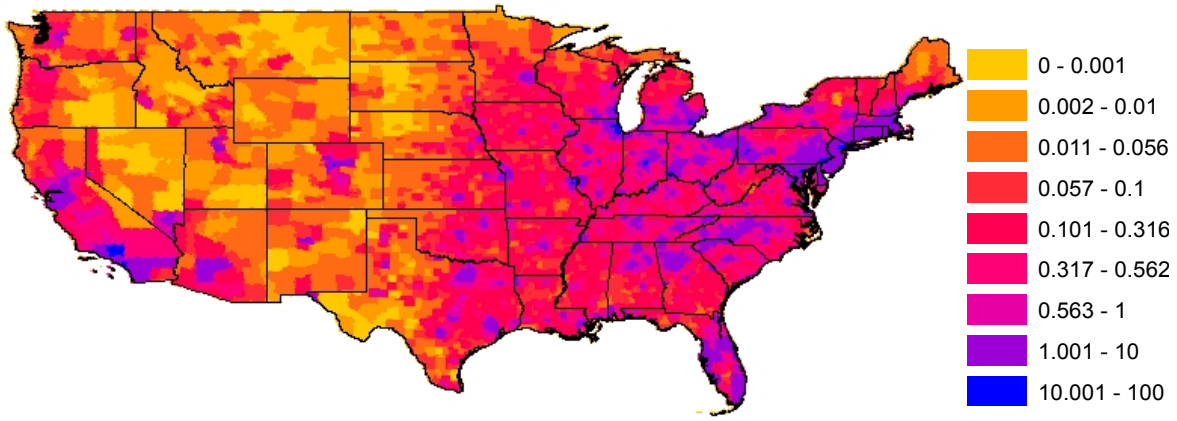

Figure 3.3: Annual respiratory mortalities attributable to ozone at 12 km resolution.

The predicted number of premature mortalities due to respiratory causes from ozone exposure above the low concentration threshold at various resolutions is shown in Figure 3.4. Small fluctuations in the estimates are an artifact of how grid cells line up with population and emissions. Estimates at coarse resolutions, larger than approximately 96 km, were not more than 5% different from the best estimate (Figure 3.5) (calculated as the coarse resolution estimate minus the 12 km estimate, divided by the coarse estimate). Diluting the concentration of one grid cell into adjacent grid cells did not change the total concentration of the new cell because ozone is relatively uniform spatially. When the grid resolution was increased from 12 km to 36 km, the estimate of national mortalities increased by two percent. However, the estimate of mortality calculated by using the 36 km output from the CMAQ model produced an estimate of mortality that was 9% higher than the 36 km scaled estimate and 11% higher than the 12 km estimate. The difference between the estimate using scaled data and the estimate from model data demonstrates the addition of uncertainty due to the effect of resolution on model chemistry.

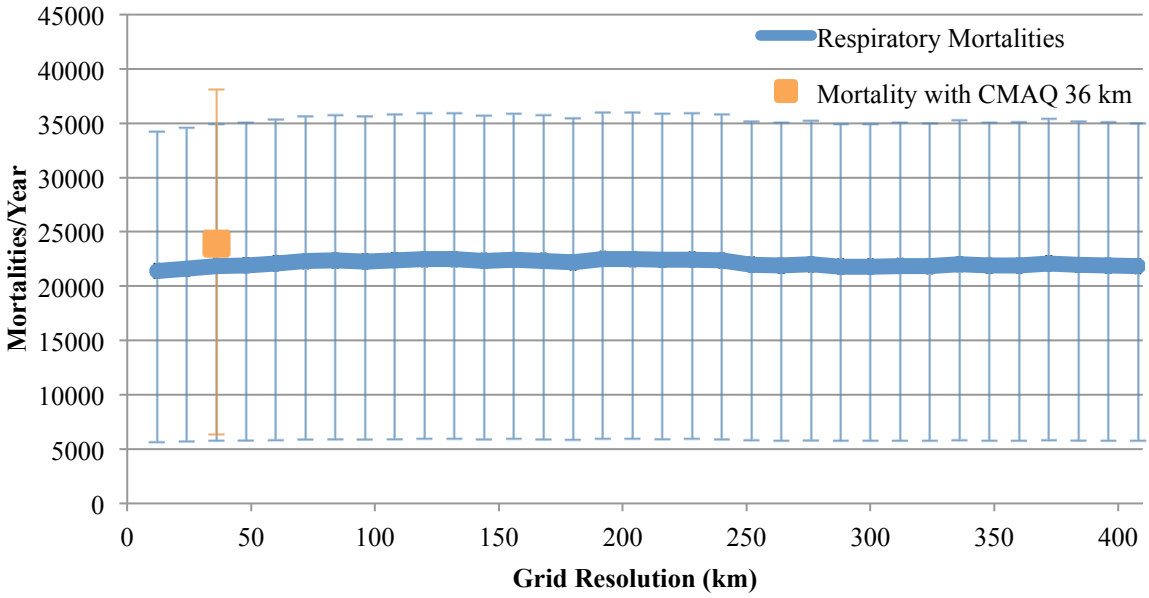

Figure 3.4: Number of annual respiratory mortalities, and 95% confidence limits, due to ozone exposure estimated at various resolutions, including the results from the CMAQ 36 km resolution.

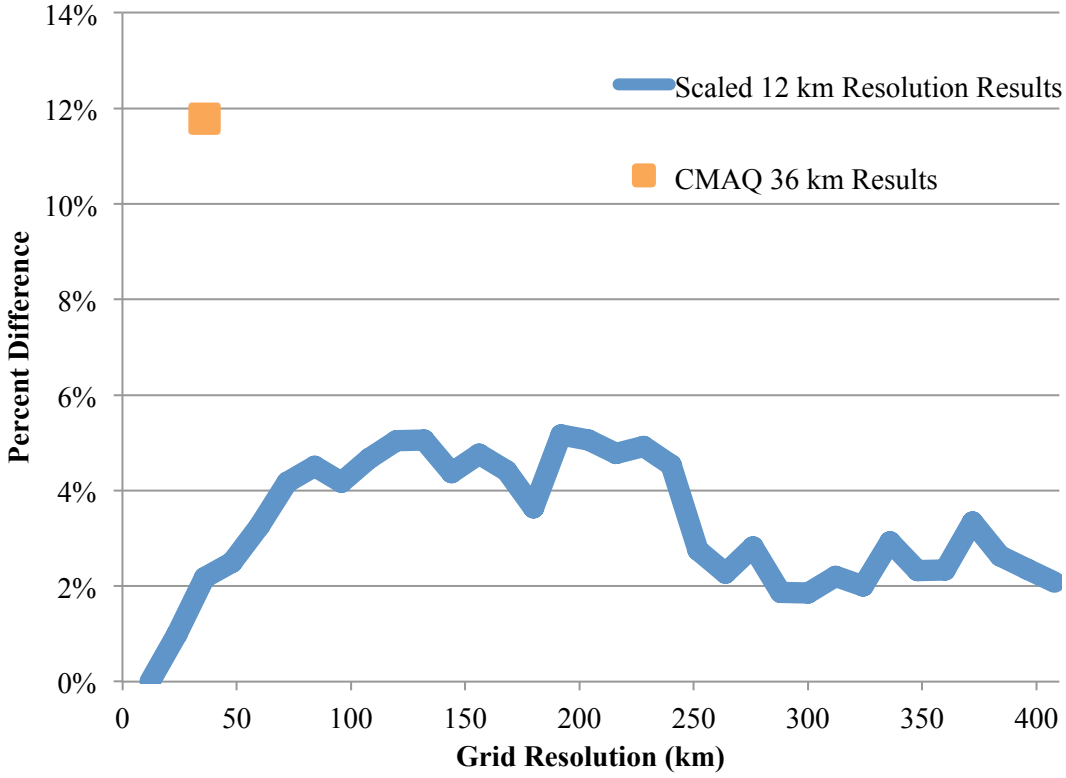

Figure 3.5. Percent difference, relative to coarse estimate, between the best estimate at 12 km of ozone respiratory mortalities and estimates at a coarse resolution for various resolutions, including the CMAQ 36 km estimate, calculated as (coarse results -12km results)/coarse results.

Figure 3.6 shows the difference in the number of mortalities estimated by the model at a 12 km resolution minus the mortalities at scaled coarser resolutions. At 36 km resolution, there is no noticeable trend in the bias. At 144 km resolution, more cells, especially in the mid-west, overestimate mortality and few cells are able to correctly estimate the number of deaths predicted at the finest resolution. A coarse resolution of 144 km is unable to capture the changes in concentration and population that occur in and around cities. At 408 km resolution, most of the coarse cells are predicting fewer deaths than the 12 km estimate projected. However, the 408 km cell for the New York City region greatly over estimated the number of deaths. The magnitude of the bias increases around New York City, southern California, and the industrialized portions around the Great Lakes region.

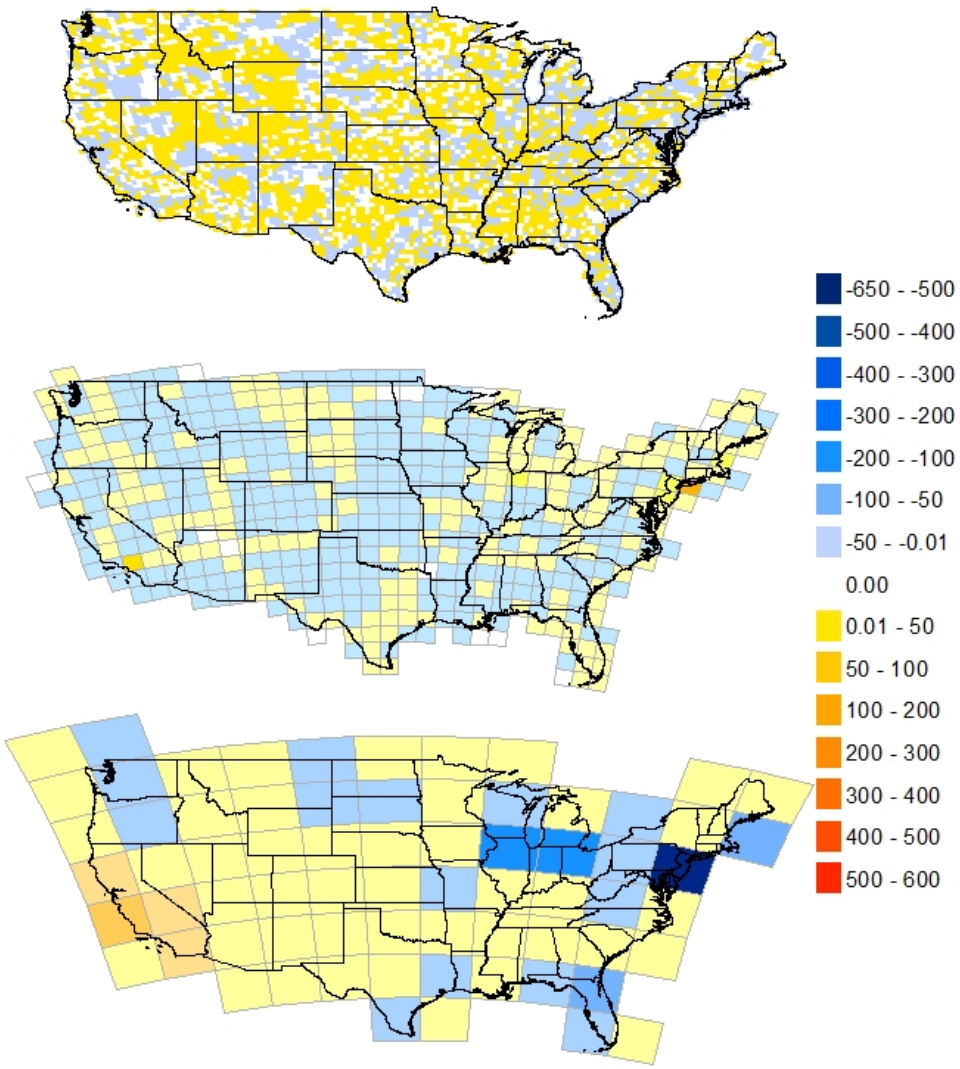

Figure 3.6: Difference in the number of annual ozone respiratory mortalities, calculated as the 12 km estimate minus the 36 km, 144 km, and 408 km estimate, respectively.

3.3 Premature Mortalities Due to Total $PM_{2.5}$

Using the finest resolution, we estimate that 66,000 (95% CI, 44,700 – 86,500) all-cause mortalities, 61,000 (95% CI, 48,400 – 73,200) CPD mortalities and 9,900 (95% CI, 4,500 – 15,100) lung cancer mortalities occur each year due to 2005 annual average $PM_{2.5}$ concentration. Cardiopulmonary mortalities and lung cancer mortalities can be summed to give an alternate estimate of the total burden that agrees well with the all-cause estimate. The greatest densities of mortalities occur in highly populated areas (Figure 3.7). Our estimates of

$PM_{2.5}$ mortality are similar to what Anenberg et al. (2010) estimated for North America, but less than the mean estimates of national mortality from the U.S. EPA (2010) and Fann et al. (2011a). Anenberg et al. estimated 65,000 (1 SD, 35,000 – 95,000) cardiopulmonary mortalities and 10,000 (1 SD, 5,000 – 15,000) lung cancer mortalities each year due to 2000 annual average $PM_{2.5}$ concentrations above a low concentration threshold of 5.8 $\mu g/m^3$. The U.S. EPA approximates 80,000 (95% CI, 54,000 – 110,000) all-cause mortalities occur each year in the US from 2005 $PM_{2.5}$ concentrations above a 5.8 $\mu g/m^3$ exposure threshold. Fann et al. evaluated all-cause mortality relative to non-anthropogenic pollutant levels and estimated 130,000 (95% CI, 51,000 – 200,000) all-cause mortalities occur in the US annually. Differences between our results and the other national estimates are due in part to the endpoints analyzed and our application of a low-concentration threshold.

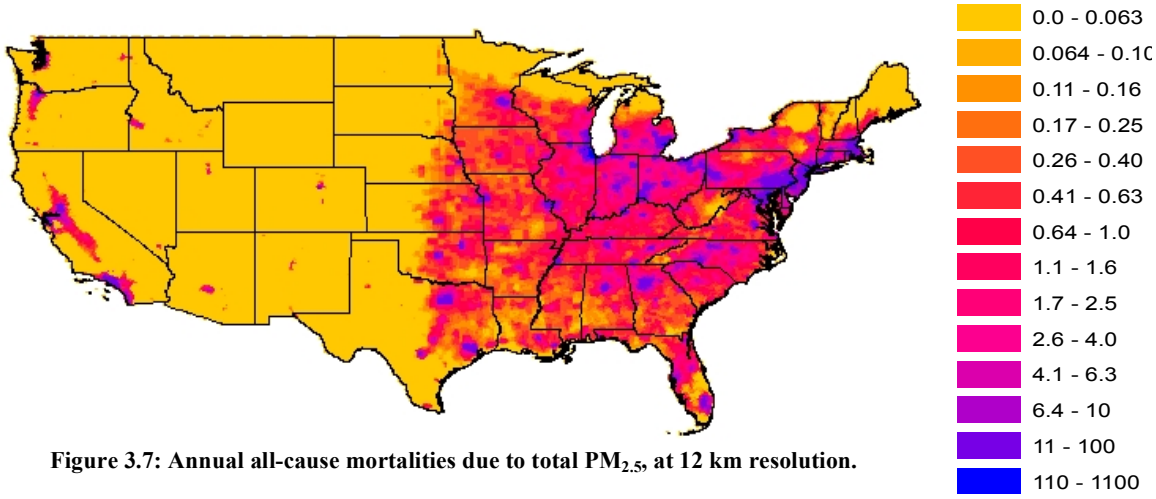

Figure 3.7: Annual all-cause mortalities due to total $PM_{2.5}$, at 12 km resolution.

Increasing grid size decreases the estimated number of mortalities from all-cause, CPD, lung cancer, and ischemic heart disease (Figure 3.8). The effect of resolution on exposure decreases the estimate of mortalities from $PM_{2.5}$ by up to 38% from the best estimate (Figure 3.9). At a resolution of 96 km, the all-cause mortality estimate is 18% lower than the best estimate. A resolution of 144 km produces an estimate 25% lower than the best

estimate. Resolutions coarser than approximately 300 km result in estimates 38% different from the best estimate. The decrease in the estimated mortalities can be explained by the decrease in peak $PM_{2.5}$ concentrations due to dilution in coarser grid cells (Table 3.1).

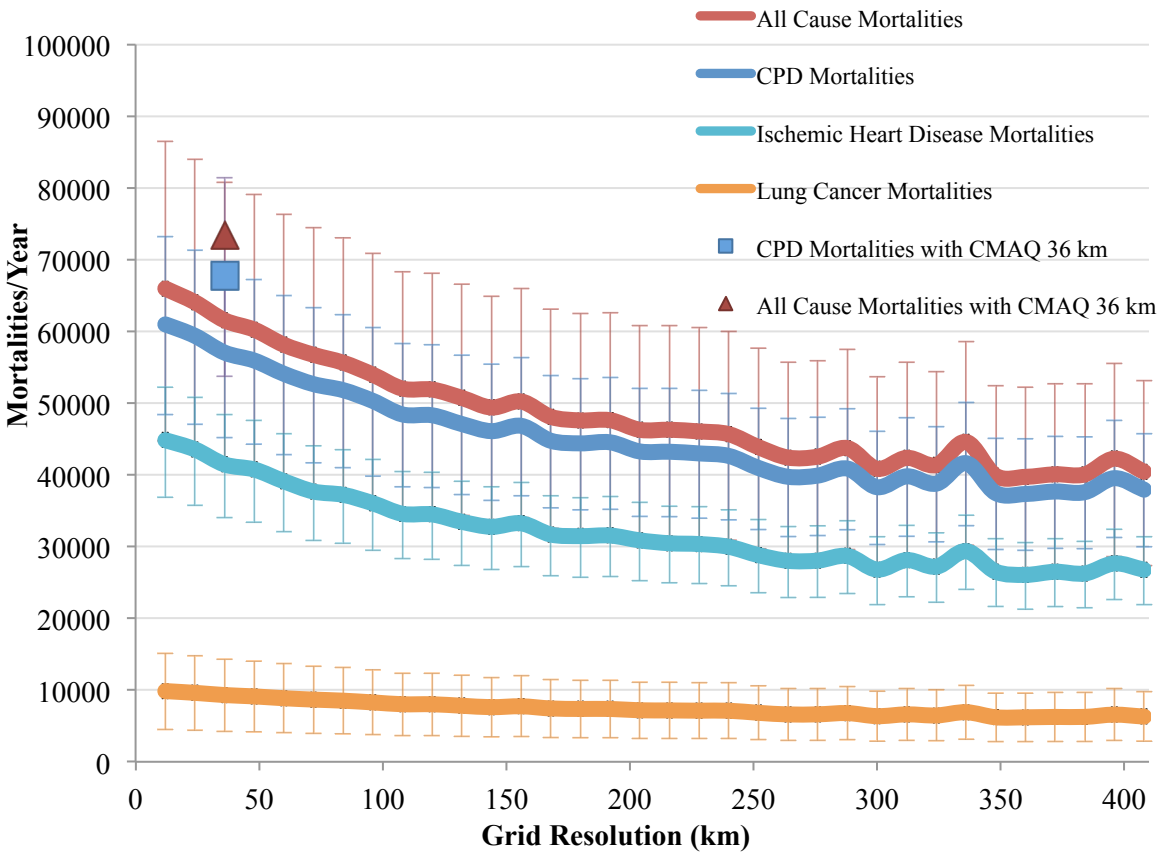

Figure 3.8: Estimated mortalities due to total $PM_{2.5}$ exposure calculated at various grid cell resolutions for all-cause, ischemic heart disease, lung cancer, and cardiopulmonary disease mortalities, including the CMAQ 36 km results for all-cause and CPD mortality.

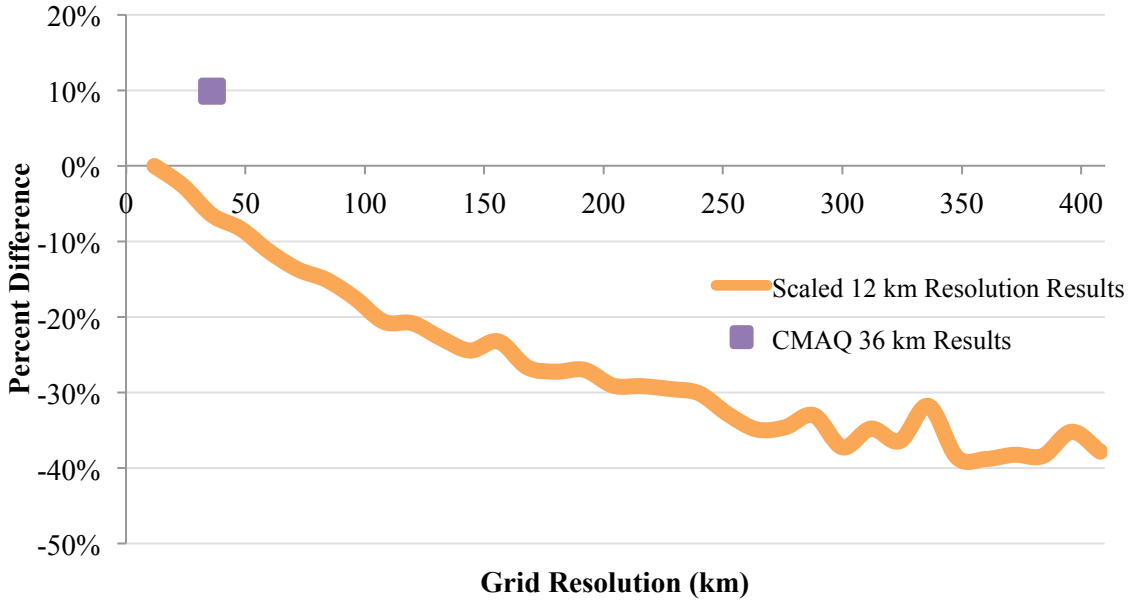

Figure 3.9: Percent difference, relative to coarse estimate, between best estimate and estimates at the scaled coarse resolution for $PM_{2.5}$ CPD mortalities for various resolutions, including CMAQ 36 km estimate, calculated as (coarse results -12km results)/coarse results.

The estimate of burden using the CMAQ 36 km data demonstrates the uncertainty in health impact estimates due to resolution's affect on chemistry. At 36 km resolution, the scaled 12 km data produces an estimate that is 6.5% lower than the best estimate, while the 36 km CMAQ data results in an estimate that is that is 11% higher than the best estimate at 12 km resolution. The effect of resolution on chemistry introduces additional uncertainty into coarse grid modeling, but the magnitude and sign of the bias is not well studied for $PM_{2.5}$.

Figure 3.10 reveals locations where coarse model resolutions overestimate or underestimate the number of mortalities when compared to the 12 km resolution. At 36 km resolution, the grid cells in the eastern US and on the west coast are slightly over predicting or under predicting mortalities. The mortalities in mid-western US and northern New England are well predicted by this resolution. At 144 km resolution, the mid-west is still well predicted by the model, but a majority of the cells on the west coast and in the eastern US are

under predicting mortality. Some cells around cities, such as New York City, Miami, Houston, and Los Angeles, are predicting far fewer deaths at a coarse resolution when compared to 12 km resolution. Dilution of $PM_{2.5}$ concentrations produces grid cells where a larger population is exposed to a concentration different than what they are exposed to at fine resolution. Because $PM_{2.5}$ concentrations are not geographically homogenous, averaging with adjacent cells can dramatically change a population's exposure concentration. At 408 km resolution, all cells are under estimating mortality except for a few in rural areas. Cells containing high population and high $PM_{2.5}$ concentration produce estimates that differ greatly from the 12 km estimate.

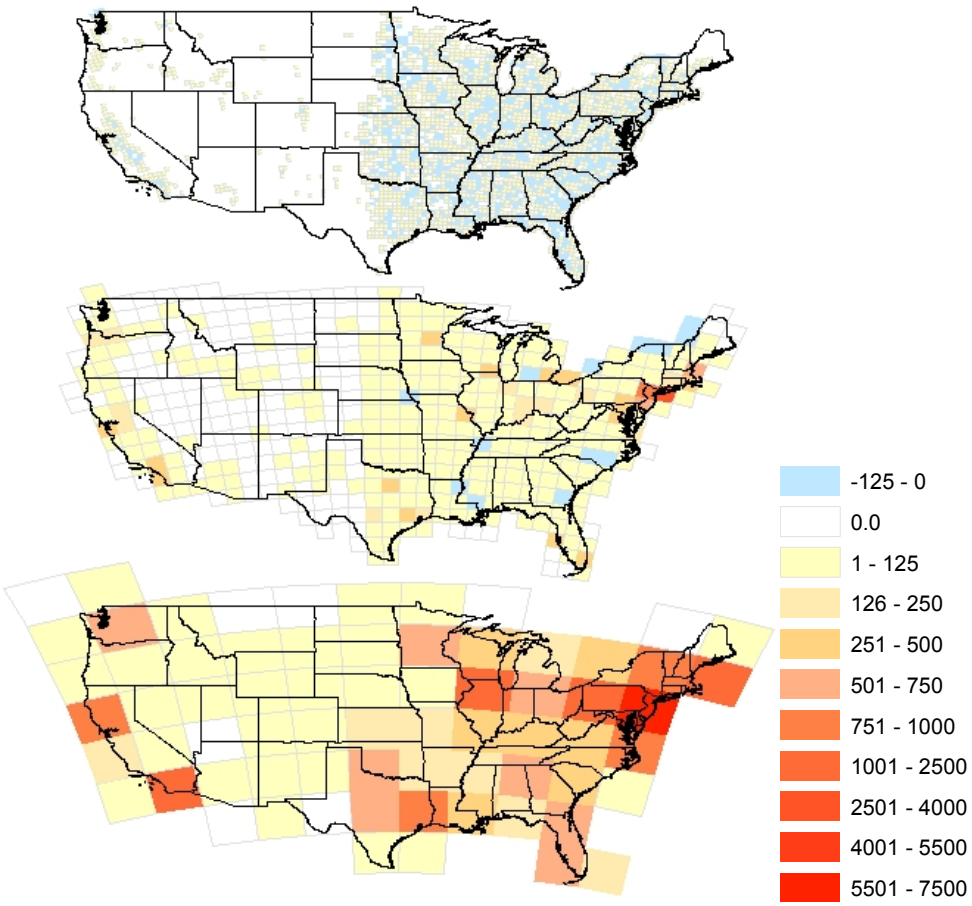

Figure 3.10: Difference in estimated total $PM_{2.5}$ attributable all-cause mortalities due to resolution, calculated as the 12 km estimate minus the 36 km, 144 km, and 408 km estimate, respectively.

3.4 Effect of Grid Resolution on Primary and Secondary Species of $PM_{2.5}$

Mortalities attributable to primary and secondary species of $PM_{2.5}$ are under predicted at coarse grid cell resolution (Figure 3.12). Coarse resolution has the greatest percentage effect on estimates of mortality driven with primary species (EC and other; OC contains portions of primary and secondary) (Figure 3.11) with estimates at 408 km resolution that are 25-43% less than the best estimate at 12 km resolution. Primary species are directly emitted, have a short atmospheric lifetime, and are minimally influenced by chemical processes in the atmospheric model.

Mortalities attributable to secondary species are less biased than $PM_{2.5}$ total or primary species and, at 408 km resolution, are 7-18% different from the best estimate. For ammonium nitrate, a secondary species, increasing grid cell size introduces less percent bias into mortality estimates than for primary species. Ammonium sulfate has the highest concentration of the $PM_{2.5}$ species and it is the least affected by grid cell size. The greater bias for primary species reflects the shorter atmospheric lifetimes and spatial ranges, compared to secondary species.

Figure 3.13 demonstrates that both EC and ammonium sulfate under predict mortalities in most locations across the United States with significant bias introduced in areas with steep population and concentration gradients. However, the coarse resolution model leads to less overall bias in mortality for ammonium sulfate than for elemental carbon. The decrease in the ability of coarse resolution to correctly capture the burden of mortality from total $PM_{2.5}$ is due mainly to the spatial heterogeneity of primary particulate species.

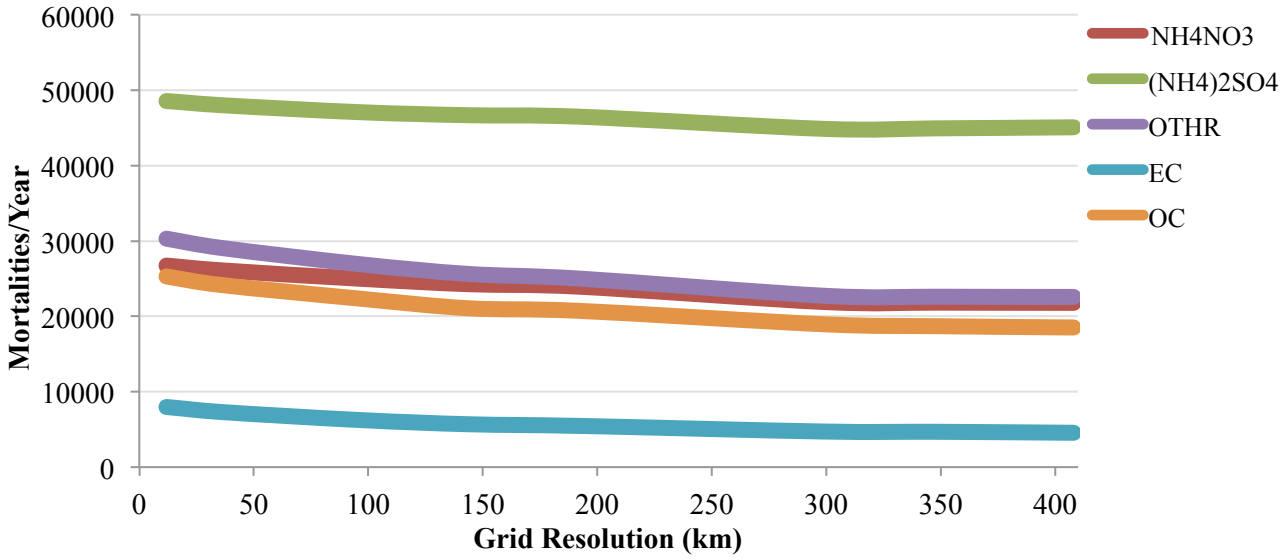

Figure 3.11: Annual all-cause mortalities due to $PM_{2.5}$ species concentrations in the US as a function of grid resolution, shown without low concentration threshold.

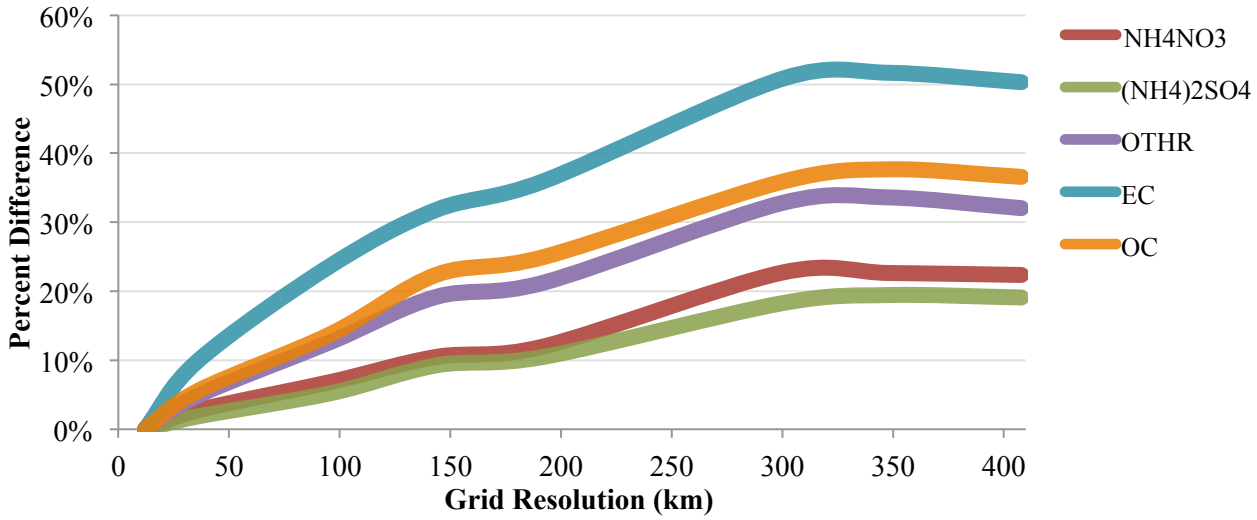

Figure 3.12: Percent difference between all-cause mortality estimate at 12 km resolution and at coarser resolutions for $PM_{2.5}$ species, without a low-concentration threshold.

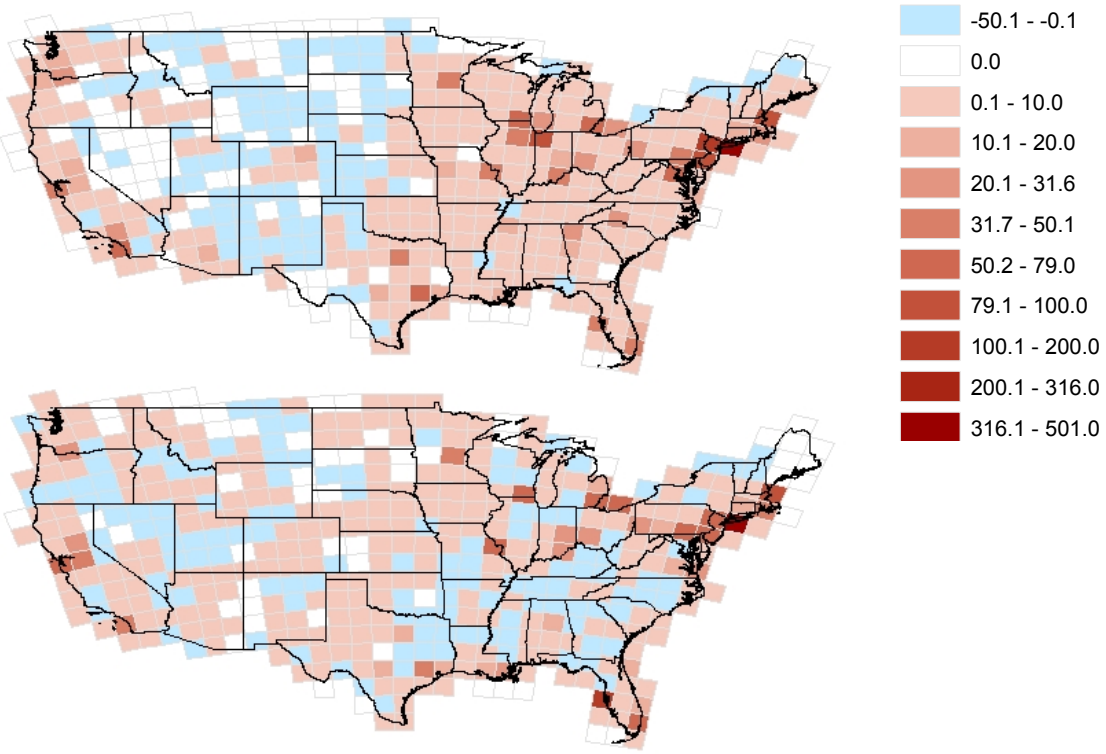

Figure 3.13: Difference in estimated number all-cause mortalities for EC (top) and ammonium sulfate (bottom) without a low-concentration threshold, calculated as the 12 km estimate minus the 144 km estimate.

CHAPTER 4: SUMMARY AND CONCLUSIONS

We estimated the annual burden of mortality in the US attributable to $PM_{2.5}$ and O_3 concentrations relative to low-concentration thresholds by conducting a health impact assessment. From this assessment, we estimate that 66,000 (95% CI, 44,700 – 86,500) and 21,400 (95% CI, 5,600 – 34,200) mortalities occur each year due to $PM_{2.5}$ and ozone, respectively. Cardiopulmonary disease induced by $PM_{2.5}$ contributes the majority of the national burden. Because our calculation only includes the US population 30 years of age and older, although negative health impacts likely occur for all age groups, we likely underestimate the total mortality associated with ozone and $PM_{2.5}$ in the US. We also do not account for the known influences of ozone and $PM_{2.5}$ on morbidity health outcomes.

The finest resolutions of our results show that the density of mortalities is greatest around urban areas where population is the greatest, but mortalities are also appreciable in rural areas. We evaluated the effect of increasing grid size on national estimates of mortality attributable to ozone, $PM_{2.5}$, and several species of $PM_{2.5}$ relative to results obtained at 12 km resolution. The estimate of total national respiratory mortalities attributable to ozone was minimally affected by increasing grid cell size, and no resolution increased results by more than 5% from the best estimate. Our results indicate that the effect of resolution on chemistry is likely to be the greatest source of error in mortality results driven with ozone concentrations.

Results driven with coarse total $PM_{2.5}$ concentrations were up to 38% lower than the best estimate conducted with fine resolution concentrations. Overall, large grid cell resolution is inadequate for capturing local variations in $PM_{2.5}$ concentrations. However, mortality estimates at a coarse resolution may produce estimates that are comparable to fine scale results when the majority of $PM_{2.5}$ is comprised of secondary species. Discrepancies between fine and coarse resolution estimates of mortality are greatest near highly populated urban regions.

Our estimates of total premature mortalities are constrained by important limitations and assumptions. In our health impact assessment, we assumed that different populations demonstrate the same relationship between mortality and change in air pollutant concentration. Our $PM_{2.5}$ species results are limited by the assumption that all $PM_{2.5}$ species have the same effect on mortality. As noted earlier, some $PM_{2.5}$ species may have greater influences on mortality than is represented by the risk ratio derived from total $PM_{2.5}$ concentrations, but these influences are not well understood. The effect of different concentration response functions on mortality attributable to the species of $PM_{2.5}$ should be studied further.

Resolution introduces uncertainty into health impact assessments due to model chemistry and population and concentration gradients smoothed by assuming uniformity in a cell. Results from this study show the important effect resolution has on exposure, but they are insufficient to fully evaluate the effect of resolution on model chemistry for $PM_{2.5}$ or O_3. Fine resolution models are useful for capturing areas of high and low ozone precursor concentrations, but they do not capture the mixing that occurs in ozone plumes as well as coarser resolutions (Sillman, 1999). Therefore, the effect of resolution on model chemistry,

in concurrence with other atmospheric conditions, may lead to an overestimation of ozone concentrations in rural areas, and an underestimation in urban areas (Liang and Jacobson, 2000; Haney and Douglas, 1996).

Few studies have been conducted to evaluate the effect of model resolution on $PM_{2.5}$ concentrations and consequent health burden estimates. The EPA recommends fine resolution (<12 km) to model particle concentrations and, in areas with primary particle sources, the recommended model resolution is 4 km. Concentrations of primary $PM_{2.5}$ species can produce steep spatial gradients that are best captured by air quality models run with fine spatial resolution (U.S. EPA, 2007).

This research has important implications for health impact assessments. Modeled estimates of air quality are used to analyze the effect that pollutant concentrations have on mortality regionally, nationally, and globally. Grid cell size may introduce additional bias into the estimate of health burden from $PM_{2.5}$ beyond uncertainties in the chemical formation and distribution of pollutants. For $PM_{2.5}$, health burden calculations performed with grid sizes above 12 km but below 150 km should increase mortality estimates by two to 25 percent. Estimates driven with grids sized 150 km and larger should increase mortality by 25 to 61 percent. However, there is added uncertainty of the effect of grid resolution on particle chemistry, and this uncertainty could amplify or minimize the effect of resolution on $PM_{2.5}$ exposure. The bias introduced by grid cell resolution into estimates of mortality from ozone is small and no adjustment is needed when calculating burden with coarse resolution, but the bias introduced by chemistry may require burden estimates to be adjusted. To further our understanding of how health burden results are influenced by coarse grid cell resolution, this analysis should be repeated using coarse pollutant output from the CMAQ or another air

quality model executed at multiple coarse grid resolutions. In addition, future work should investigate the importance of resolution for evaluating changes in emissions, rather than the total burdens evaluated here, and subsequent changes in mortality.

APPENDIX

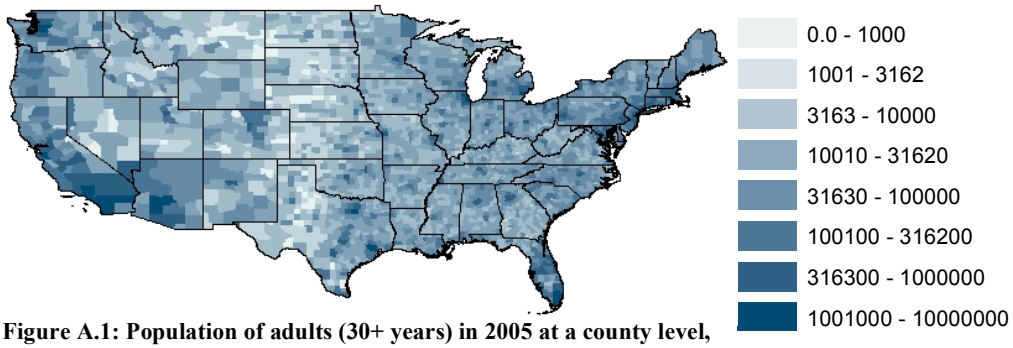

Figure A.1: Population of adults (30+ years) in 2005 at a county level, obtained from BenMAP.

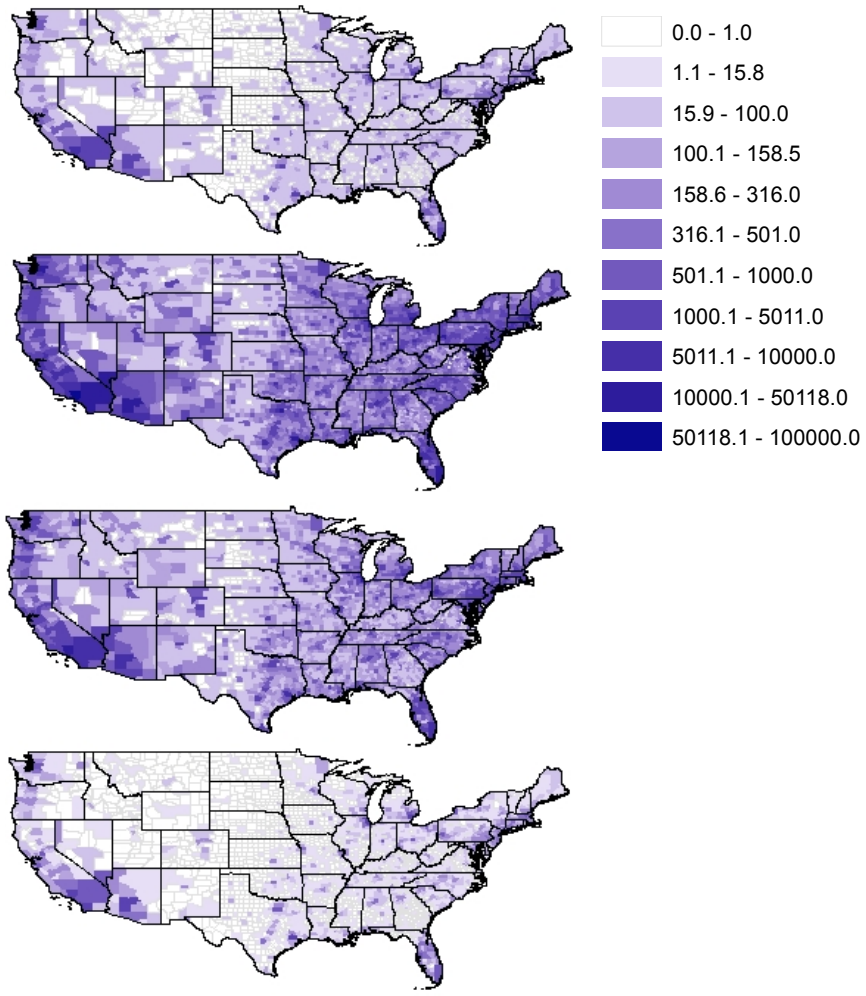

Figure A.2: Baseline respiratory (top), all-cause (middle-top), CPD (middle-bottom), and lung cancer (bottom) mortalities per year for adults (30+ years), at a county level.

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