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Assessing Public Health Burden Associated with Exposure to Ambient Black Carbon in the United States

Ying Li
East Tennessee State University, liy005@etsu.edu

Daven K. Henze
University of Colorado at Boulder

Darby Jack
Columbia University

Barron H. Henderson
University of Florida

Patrick L. Kinney
Columbia University

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Copyright Statement

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Ying Li[a], **Daven K. Henze**[b], **Darby Jack**[c], **Barron H. Henderson**[d], and **Patrick L. Kinney**[c,*]

[a]Department of Environmental Health, College of Public Health, East Tennessee State University, Johnson City, TN, USA

[b]Department of Mechanical Engineering, University of Colorado at Boulder, Boulder, CO, USA

[c]Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, New York, NY, USA

[d]Department of Environmental Engineering Sciences, University of Florida, Gainesville, FL, USA

Abstract

Black carbon (BC) is a significant component of fine particulate matter ($PM_{2.5}$) air pollution, which has been linked to a series of adverse health effects, in particular premature mortality. Recent scientific research indicates that BC also plays an important role in climate change. Therefore, controlling black carbon emissions provides an opportunity for a double dividend. This study quantifies the national burden of mortality and morbidity attributable to exposure to ambient BC in the United States (US). We use GEOS–Chem, a global 3-D model of atmospheric composition to estimate the 2010 annual average BC levels at $0.5 \times 0.667°$ resolution, and then re-grid to 12-km grid resolution across the continental US. Using $PM_{2.5}$ mortality risk coefficient drawn from the American Cancer Society cohort study, the numbers of deaths due to BC exposure were estimated for each 12-km grid, and then aggregated to the county, state and national level. Given evidence that BC particles may pose a greater risk on human health than other components of $PM_{2.5}$, we also conducted sensitivity analysis using BC-specific risk coefficients drawn from recent literature. We estimated approximately 14,000 deaths to result from the 2010 BC levels, and hundreds of thousands of illness cases, ranging from hospitalizations and emergency department visits to minor respiratory symptoms. Sensitivity analysis indicates that the total BC-related mortality could be even significantly larger than the above mortality estimate. Our findings indicate that controlling BC emissions would have substantial benefits for public health in the US.

Graphical Abstract

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[*]Corresponding author at: Department of Environmental Health Sciences, Mailman School of Public Health, Columbia University, 722 West 168th Street, New York, NY, USA. plk3@columbia.edu (P.L. Kinney).
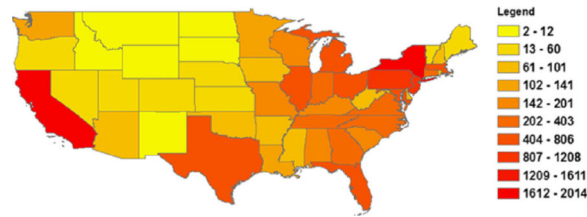
Annual premature mortality by State attributed to exposure to ambient black carbon in the United States in 2010.

Keywords

Air pollution; Black carbon; Mortality; Public health burden

1. Introduction

Black carbon (BC) is a significant component of ambient fine particulate matter (PM <= 2.5 μm in aerodynamic diameter; $PM_{2.5}$) air pollution. Recent scientific evidence has indicated that BC is the most strongly light-absorbing component of $PM_{2.5}$. BC absorbs solar radiation, influences cloud processes, and alters the melting of snow and ice cover, and thus plays an important role in the Earth's climate system (Bond et al., 2013). In addition to its climate effects, BC has been associated with adverse effects on human health (e.g. Janssen et al., 2011; Laden et al., 2006). Some suggested that BC may pose greater health risk as indicated by the higher effect estimates per mass unit for BC particles compared with PM mass as a whole (Janssen et al., 2011, 2012). Therefore, mitigating climate change through controlling BC emissions is likely to generate substantial co-benefits for human health.

Black carbon is emitted from a variety of combustion processes, mainly the incomplete combustion of fossil fuels, biofuels, and biomass (EPA, 2012). The US contributes about 8% of the global emissions of BC (EPA, 2012). Within the US, BC is estimated to account for approximately 12% of all direct $PM_{2.5}$ emissions in 2005, and transport was the largest source of BC emissions in that year, which contributed to about 52% of the total BC emissions in the US, followed by open biomass burning (35%) (EPA, 2012).

Given the significance of BC both as a health effect agent and a climate forcing pollutant, the total health burden of BC would provide valuable information in developing climate and air pollution strategies. BC emissions will be substantially reduced by 2030 due to recently promulgated regulations, including the emissions standards for new engines and the retrofit programs for in-use mobile diesel engines (EPA), 2012). The present study aims to quantify the public health burden attributable to the ambient BC levels within the continental US in 2010 prior to the expected improvement in BC levels in the future. Earlier national-level particle-related health impacts assessment have focused on total, undifferentiated $PM_{2.5}$ mass. For example, using the photochemical Community Multiscale Air Quality (CMAQ) model results in conjunction with ambient monitored data, Fann et al. (2012) estimated 130,000 $PM_{2.5}$-related deaths in 2005. Based on observational data, it has been estimated that BC comprises approximately 5–10% of average urban $PM_{2.5}$ mass in the US (EPA,

2012). However, it is difficult to estimate the BC-related health outcomes based on the PM$_{2.5}$-related estimates owing to the fact that spatial variability in concentrations for BC is often larger than for PM$_{2.5}$, particularly in urban and populous areas. Also, given recent evidence that BC particles may pose a greater risk on human heath than other components of PM$_{2.5}$, it is important to assess the national health impacts of BC separately.

We use an integrated procedure that combines exposure assessment, exposure-response relationships and baseline health statistics to quantify the public health burden attributed to BC exposure. Our methods are consistent with those used previously by the U.S. Environmental Protection Agency (U.S. EPA) to assess the public health burden attributed to criteria air pollutants in the US, such as particulate matter and ozone (Fann et al., 2012; Hubbell et al., 2005), and also those used to assess the global burden of disease (Anenberg et al., 2010; Cohen et al., 2005; Ostro, 2004). To estimate ambient BC concentrations, we use GEOS–Chem, a global 3-D model of atmospheric composition with a horizontal resolution of 0.5° latitude × 0.667° longitude (approximately 55 km × 55 km) over North America. Since observational data for BC are only available at limited monitoring locations, using simulated concentrations allows us to capture the BC impacts across the entire continental US.

We quantify the excess mortality and morbidity impacts associated with anthropogenic BC emissions nationwide and also the effects at the State and county level. In addition, previous health risk assessments of PM$_{2.5}$ generally assume that all constituents of PM$_{2.5}$, including BC, are equally toxic (Levy et al., 2012). We conducted a literature review of epidemiologic studies that investigate the health risks from both BC and total PM$_{2.5}$ mass. In this analysis, we use both the concentration response (CR) functions for PM$_{2.5}$ and several BC-specific CR functions to indicate the possible species potency of BC Finally, we discuss the significant sources of uncertainty associated with the health impact assessment and quantify their impacts on incidence estimates.

The remainder of the paper is organized as follows. Section 2 reviews the current epidemiologic literature on the health effects of BC and describes the methods used to quantify the public health burden (mortality and morbidity) associated with BC exposure in the US. Section 3 presents our estimates of BC-related premature mortality at the nation, State and county level, as well as the national estimates of various morbidity effects. Section 3 also discusses the uncertainty in our estimates. Section 4 summarizes the major conclusions of this study and issues to be further investigated in future work.

2. Methods

2.1. Health effects of ambient black carbon pollution

The negative impacts of PM$_{2.5}$ on public health have been well documented by epidemiological studies. Time-series studies of the short-term effects of air pollutants, conducted around the world, have consistently reported significant associations between daily mortality and daily exposure to PM$_{2.5}$ (Ostro, 2004). Cohort studies usually aim at investigating possible long-term chronic effects of exposure to air pollution. Two large-scale cohort studies conducted in the U.S. – the Harvard Six Cities Study and the American

Cancer Society (ACS) study – both reported increased mortality associated with an increase in annual average $PM_{2.5}$ levels (Dockery et al., 1993; Krewski et al., 2000, 2009; Laden et al., 2006; Pope III et al., 2002, 1995).

While existing epidemiological studies provide compelling evidence that $PM_{2.5}$ increases mortality rates, it is uncertain whether some components of $PM_{2.5}$ have a stronger relationship with mortality than other components. Some studies report that traffic-related fine particulate air pollution, indicated by black carbon, may pose greater risk on human health than $PM_{2.5}$ from other sources (Gan et al., 2011; Laden et al., 2006; Peng et al., 2009), while others report that some other constituents of $PM_{2.5}$, such as sulfates or organic carbon have stronger or more robust associations with adverse health effects (Ostro et al., 2010; Smith et al., 2010). However, in spite of this evidence, U.S. EPA maintains that "there is insufficient information at present to differentiate the health effects of the various constituents of $PM_{2.5}$" and "the limited scientific evidence that is currently available about the health effects of BC is generally consistent with the general $PM_{2.5}$ health literature" (EPA, 2012).

A recent systematic review on health effects of black carbon performed by the World Health Organization Regional Office for Europe found that effect estimates from both short- and long-term studies are much higher for BC compared to PM_{10} and $PM_{2.5}$ when the particulate measures are expressed per μg/m^3 (Janssen et al., 2012). This report suggests that BC might be a better indicator of harmful particulate substances from combustion sources (especially traffic) than undifferentiated $PM_{2.5}$ mass (Janssen et al., 2012). Moreover, Janssen et al. (2011) conducted a systematic review and meta-analysis of health effects of BC compared with $PM_{2.5}$ based on data from studies that measured both exposures. The study found that the pooled estimates of relative risk per 1 μg/m^3 for all-cause mortality related to long-term exposure to $PM_{2.5}$ and BC are 1.007 and 1.06, respectively, suggesting that the concentration–response coefficient β for BC could be up to ten times larger than that for $PM_{2.5}$.

Given the uncertainty with regard to the species potency of BC, we reviewed existing epidemiological studies that report the health effects of both $PM_{2.5}$ and BC. Both time-series studies of the short-term effects and cohort studies of long-term chronic effects were reviewed. As shown in Tables AI and AII in the Appendix A, our review indicates that effect estimates for BC are mostly higher than those for PM in terms of effect per μg/m^3 particulate. Among the studies we found that report the effects of both $PM_{2.5}$ and BC in the same analysis, ratios of BC/$PM_{2.5}$ effects vary from 3 to 27.7 for long-term mortality by different causes (Table A.I), and from 0.33 to 50 for short-term mortality or morbidity by different causes (Table A.II). Levy et al. (2012) is the only study we found that reports a smaller effect of BC than the effect of $PM_{2.5}$. That study conducted a meta-analysis that pooled existing short-term mortality estimates for the individual constituents as well as the total $PM_{2.5}$ mass. However, the meta-analysis includes three studies in the early 2000s that relied on coefficient of haze (COH) as a measurement of BC, and all these studies found a significant smaller effect of COH than that of $PM_{2.5}$.

Janssen et al. (2011) summarize six studies that include both single-and two-pollutant models of time-series studies including PM (PM_{10} or $PM_{2.5}$) and BC (measured as black smoke in all the studies). It was found that the effect of PM was reduced or became negative when moving from single- to two-pollutant model in all six studies, whereas the effect of BC was increased in half of the studies and reduced in the remaining studies. The fact that the effect of PM diminishes or disappears when BC is controlled in the model also supports the argument that BC may pose greater toxicity than the other components of $PM_{2.5}$.

Our review provides supporting evidence that BC may be more toxic than the other components of $PM_{2.5}$ and thus the effect estimates for BC is larger than that of undifferentiated $PM_{2.5}$ mass when expressed as effect per unit of concentration. However, the existing studies are subject to several caveats. First, as shown in Tables AI and AII, the correlations[1] between $PM_{2.5}$ and BC reported in the studies we review are generally high (greater than 0.5 in most cases). In this case, it may be difficult to estimate separate effects of $PM_{2.5}$ and BC via regression analysis. For example, Peng et al. (2009) argues that any reduction in effect size of either component when moving from the single-pollutant to the multipollutant model could be a result of the correlations between components. Also, as argued by Smith et al. (2010), it may be difficult to draw conclusions about the health effects of individual species because atmospheric black carbon, ozone, and sulfate are associated and could interact with related toxic species. Second, many studies focus on the effects of PM and BC in a single metropolitan area. In urban environments, both spatial and temporal variations of BC are greater than that of PM since BC is mostly governed by vehicle exhaust emissions, while PM concentrations are also governed by regional non-traffic emissions (Janssen et al., 2011; Reche et al., 2011). For example, Kinney et al. (2000) measured concentrations of $PM_{2.5}$ and elemental carbon (EC) on sidewalks in a community in New York City. They reported that mean concentrations of $PM_{2.5}$ exhibited only modest site-to-site variation (37–47 $\mu g/m^3$), whereas EC concentrations varied 4-fold across sites (from 1.5 to 6 $\mu g/m^3$), and were associated with bus and truck counts on adjacent streets. Also, in a study on the long-term exposure to traffic-related air pollution and the risk of coronary heart disease hospitalization and mortality in metropolitan Vancouver, Canada (Gan et al., 2011), the variation of $PM_{2.5}$ concentrations (mean value: 4.08 $\mu g/m^3$; standard deviation (SD): 1.63) is much smaller compared to that of BC concentrations (mean value: 1.49 $\mu g/m^3$; SD: 1.1). Relatively small variation of $PM_{2.5}$ may explain the reason that the effect of $PM_{2.5}$ is attenuated when BC is included in the model. Third, in multi-city studies BC concentrations measured at a single outdoor monitoring site have often been used to reflect exposure within a city. However, within-city variability in concentrations for BC is larger than for $PM_{2.5}$ owing to the considerable effect of local primary combustion sources, especially traffic, on BC concentrations (Janssen et al., 2012). Within-city variability may exceed between-city variability, which underlines the importance of taking into account small-scale variations in BC in epidemiological studies (Janssen et al., 2012).

In summary, current epidemiology studies provide supporting evidence that BC may be more harmful than other constitutes of $PM_{2.5}$, but evidence to differentiate the health effects

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[1]In most cases, cohort studies report the spatial correlations between pollutants and time-series studies report temporal correlations.

of the constituents of $PM_{2.5}$ is still insufficient. Given this, for our main analysis we use a risk estimate for $PM_{2.5}$ drawn from epidemiological studies to estimate BC-related premature mortality, the most significant health outcomes due to exposure to air pollution. In sensitivity analyses, we also apply BC-specific mortality risk estimates to reflect the species potency. For morbidity, we use risk estimates for PM from epidemiological studies.

2.2. Methods for quantifying public health burden

2.2.1. Overview of the method—Our quantitative assessment of the health impacts of ambient BC pollution is based on the following components: ambient BC concentration assessment based on air quality modeling, the affected population, the health effects of interest (e.g. mortality, morbidity), the baseline incidences of those health effects, 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 or morbidity attributed to air pollution is (Anenberg et al., 2012; Li et al., 2010):

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

where,

y: BC attributable health endpoints (i.e. mortality or morbidity);

β: concentration–response coefficient from epidemiological studies (increase in the health endpoint assessed per unit increase in BC);

x: change in air quality, taken here as the modeled BC concentration;

y_0: the baseline incidence rates for the health endpoint assessed; and

Pop: the affected population, i.e. the number of people exposed to ambient BC 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 BC concentrations across the continental U.S. We then discuss how we select the concentration–response coefficient (β) used to estimate BC-related health impacts, and how we characterized the affected population (Pop) and the baseline incidence rates (y_0) in Eq. (1).

2.2.2. Modeling annual average pollutant concentrations—To simulate the annual average BC concentrations across the continental US., we use GEOS–Chem (Bey et al., 2001) (www.geos-chem.org), a global 3-D model of atmospheric composition driven by assimilated meteorological observations from the Goddard Earth Observing System (GEOS) of the National Aeronautics and Space Administration (NASA) Global Modeling Assimilation Office. Global simulations are performed at the 4° latitude × 5° longitude degree resolution, with a six month spin-up beginning in July 2009, through the end of 2010. Simulated 3-hourly concentrations from this global calculation are used as the boundary conditions to drive a higher resolution nested simulation over North America at the 0.5°

latitude by 0.667° longitude resolution (Chen et al., 2009; Wang et al., 2004). We then convert the model results to the national 12×12 km^2 grid using a two-step process. GEOS–Chem binary punch output is first converted to Climate Forecasting compliant NetCDF with PseudoNetCDF (pseudonetcdf.googlecode.com), and then regridded using a first-order conservative method as implemented in the Climate Data Operators (code.zmaw.de/projects/cdo). The 12×12 km^2 resolution is required by the standard BenMAP configuration to evaluate gridded air quality model output.

GEOS–Chem simulations include treatment of primary elemental (BC) and organic (OC) carbonaceous aerosol (Park et al., 2003). BC is emitted as a hydrophobic species (80%) that converts to hydrophilic carbon with an e-folding time of 1.15 days. Global fossil fuel and biofuel emissions of BC are from Bond et al. (2007). Over North America, these values are replaced by the inventory of Cooke et al. (1999), with top-down monthly seasonality imposed from Park et al. (2003). Monthly emissions of BC from biomass burning are from the third version of the Global Fire Emissions Database (GFED3) (Giglio et al., 2010; van der Werf et al., 2010). Wet deposition of aerosols includes large-scale precipitation and convective scavenging (Liu et al., 2001). Dry deposition is calculated using a resistance in series scheme (Wesely, 1989) that depends on meteorology and local land surface type.

In addition to carbonaceous species, fine mode aerosol in GEOS– Chem includes sulfate ammonium nitrate (Fountoukis and Nenes, 2007; Park et al., 2004), dust, and sea salt (Jaeglé et al., 2011). We did not consider here the secondary organic aerosol, which likely constitutes a significant fraction of PM$_{2.5}$ concentrations (Jimenez et al., 2009).

Surface level BC and PM$_{2.5}$ concentrations in GEOS–Chem have been evaluated with in situ observations in the U.S. in numerous studies (Duncan Fairlie et al., 2007; Heald et al., 2012; Henze et al., 2009; Leibensperger et al., 2012; Mao et al., 2013, 2011; Park et al., 2003, 2004, 2006; Pye et al., 2009; Walker et al., 2012; Zhang et al., 2012). The initial BC estimates from Park et al. (2003) showed decent skill in simulating seasonal annual average BC concentrations (R^2 from 0.68 to 0.82, slopes of reduced major axis regressions from 0.83 to 1.17) compared to observations from the 24 h average concentrations collected one out of 3 days at IMPROVE sites (Malm et al., 1994) (http://vista.cira.colostate.edu/improve). More recently, GEOS–Chem BC simulations have been evaluated in the Western US by Mao et al. (2011, 2013) for 2006, using a previous version of the GFED biomass burning inventory (van der Werf et al., 2006). Mao et al. (2013) found the model performed well for locations at elevations less than 1 km above sea level, while performance at mountainous sites was strongly impacted by uncertainties in the biomass burning emissions as well as model resolution. Here we reevaluate the model BC compared to measurements from IMPROVE, see Fig. 2 and discussion in Section 3.

2.2.3. Selection of concentration–response relationships—We estimate both mortality and morbidity associated with BC exposure in this study, including premature deaths from long-term exposure, chronic bronchitis, respiratory and cardiovascular-related hospital admissions, asthma-related emergency room visits, acute bronchitis, respiratory symptoms (lower and upper), and lost work days and minor restricted activity days.

As discussed above, in our main analysis we use risk estimates for $PM_{2.5}$ to estimate BC-related long-term mortality. We select the estimate from the recent Krewski et al. (2009) extended analysis of the American Cancer Society (ACS) study, which is a prospective cohort study that includes a large population (500,000 adults) over 116US cities. The mortality risk estimates from the ACS study have been used to assess the health burden associated with exposure to ambient $PM_{2.5}$ in the US and globally (Anenberg et al., 2010; Fann et al., 2012; Punger and West, 2013). We apply the all-cause mortality risk estimate from Krewski et al. (2009) to estimate national BC-related premature mortality as well as the spatial distribution of mortality (mortality by State and county). In a sensitivity analysis, we apply the pooled BC-specific long-term mortality risk estimate from a recent meta-analysis of health effects of BC (Janssen et al., 2011) to estimate national BC-related mortality. In order to reflect the uncertainty associated with the species potency of BC, we also apply to the national mortality estimate two probability distribution functions (PDFs) that combine the Janssen and Krewski estimates: One as a uniform distribution and the other as a triangular distribution (Table 1).

For morbidity, we consider the same health outcomes that were estimated in Fann et al. (2012), which assessed the national public health burden associated with $PM_{2.5}$ in the US. For each health outcome, BenMAP contains a library of concentration–response functions, and we select the one from the study that concerned the population over the broadest geographic area. The health endpoints considered, their concentration–response coefficients β (mean values and probability distribution functions, PDFs), and references of β are listed in Table 1.

2.2.4. Exposed population and baseline incidence rates—We use the 2010 population and baseline incidence rates data that have been preloaded in BenMAP. BenMAP uses 2010 U.S. Census block-level population data, and aggregates to population grid-cells at different scales (Abt Associates, 2013). We use the population at the national 12-km grid resolution, and the modeled BC concentrations are matched with the population in each 12-km grid cell. We assume that the modeled BC value in each grid is the best measure of population exposure in that grid. Also, the BenMAP population data are stratified by age as well as other demographic categories such as sex, race and ethnicity. We use the population data that are consistent with the age groups in the original epidemiological studies from which we draw the risk estimates β (see Table 1).

To estimate national mortality rates, the BenMAP group used individual-level mortality data from 2004 to 2006 for the whole United States from the Centers for Disease Control (CDC) and National Center for Health Statistics (NCHS) (Abt Associates, 2013). These data were combined with U.S. Census Bureau postcensal population estimates to estimate age-, cause-, and county-specific mortality rates. The age- and county-specific mortality rates in 2010 that were used in this study were estimated using adjustment factors, which were calculated based on a series of Census Bureau projected national mortality rates (Table A.III). Hospitalizations and emergency room visits were calculated using data from the Healthcare Cost and Utilization Project, which is a family of health care databases developed through a Federal-State-Industry partnership and sponsored by the Agency for Healthcare Research and Quality (Table A.III). The other morbidity incidence and prevalence rates used in this

study were estimated using national or regional averages depending on the best available data sources (Table A.IV).

2.2.5. Public health burden calculation—Using the modeled BC concentrations, concentration–response coefficients, population and baseline incidence or prevalence data, we calculated the number of excess deaths and illnesses due to BC exposure nationwide. In estimating the 95% confidence interval (CI)of the national estimates, the only source of uncertainty that is taken into account is the uncertainty in β values. The uncertainty analysis was conducted in BenMAP using Monte Carlo simulation with a sample size of 5000. We explored the effects of uncertainties in the BC risk estimate via sensitivity analyses, as noted above. Other sources of uncertainty are discussed qualitatively. We also estimated the excess number of deaths and percent of baseline mortality by State and county and mapped the spatial variations in mortality and in percent of baseline mortality using the mean value of the risk estimate (β) for all-cause mortality from Krewski et al. (2009).

3. Results and discussion

3.1. BC air quality simulation

Fig. 1(a) shows the simulated annual average BC concentrations from anthropogenic sources in 2010. The maximum simulated BC value in the continental US is 3.0 μg/m^3, the mean BC value is 0.3 μg/m^3, and the 95th percentile value is about 1.0 μg/m^3. In general, the highest BC values are in the northeastern United States, with some 'hotspots' in California.

Fig. 1 (b) and (c) show the spatially-weighted average concentrations in 2010 at the State and county level, respectively. At the State level, New Jersey had the highest spatially-weighted annual average BC concentration in 2010 (2.5 μg/m^3), followed by Delaware (1.9 μg/m^3) and Connecticut (1.7 μg/m^3). At the county level, the top 10 counties that have the highest average levels of BC are all located in New Jersey and New York and surrounding New York City (NYC). These counties are Bergen, Passaic, Somerset, Mercer, Morris, Essex and Middlesex in New Jersey, and Bronx, Rockland New York in New York.

Our evaluation of the modeled BC concentrations compared to measurements from IMPROVE indicates that the annual average BC simulations have a normalized mean bias of 0.10%, a standard error of 0.39 μg/m^3. The slope of a reduced major axis fit through the origin is 1.10 (Fig. 2(a)). The spatial plot of the model error (GEOS–Chem – IMPROVE) in Fig. 2(b) shows the model to overestimate BC concentrations in the Northeast and California. This is likely owing to the use of BC emission factors that may not accurately reflect decreasing trends in BC from particulate standards for diesels (Murphy et al., 2011).

3.2. Comparing the spatial variations of BC to total PM$_{2.5}$ mass

In order to quantitatively compare the spatial variations of BC to total PM$_{2.5}$ mass, we simulated annual average PM$_{2.5}$ concentrations from anthropogenic sources over North America in the same year, and calculated the ratio of BC to total PM$_{2.5}$. As shown in Fig. 3, the ratio of annual average BC to total PM$_{2.5}$ mass concentration varies in the range of [0–20.9%] across the continental US, and the ratio tends to be larger in the northeast and the

West Coast regions, particularly in populous urban centers. This is explained by the fact that BC is a primary aerosol whose distribution is more closely coupled with population centers.

3.3. Estimates of national mortality and morbidity attributed to BC exposure in 2010

Using the all-cause mortality risk estimate for $PM_{2.5}$ among adults aged over 30 drawn from the Krewski et al. (2009), we estimate 14,000 (95% CI: 9400–18,000) BC-related premature deaths to result from the 2010 ambient BC levels (Table 1). Given the current evidence that the effect estimate might be higher for BC compared to $PM_{2.5}$ in terms of effect per μg/m^3, we consider the mortality estimate based on the risk coefficient for $PM_{2.5}$ as a lower-bound estimate. We estimate approximately ten times the BC-related premature deaths using the BC-specific risk estimate drawn from Janssen et al. (2011) (mean: 133,807; 95% CI: 92,259–174,523) compared to the estimate based on Krewski et al. (2009). Also we estimate approximately 75,000 deaths using the two PDFs of β generated by combining the Krewski and the Janssen risk estimates.

Using the all-cause mortality risk estimate from Krewski et al. (2009), Fann et al. (2012) estimate 130,000 $PM_{2.5}$-related deaths to result from 2005 air quality level. Our estimate of BC-related deaths in 2010 based on the same risk estimate from Krewski et al. (2009) equals to about 11% of the total $PM_{2.5}$-related death estimate in 2005 above. Since the observational data show that BC comprises approximately 5–10% of average urban $PM_{2.5}$ mass in the US, our estimate indicates that the ratio of national BC-$PM_{2.5}$ related mortality is slightly higher than the ratio of BC-$PM_{2.5}$ mass concentrations. This is not surprising given that BC is a primary aerosol, with concentrations highest near urban sources, where people are also concentrated.

We also estimate 32 (95% CI: 13–52) infant deaths due to BC exposure in 2010, and a range of morbidity impacts, including nearly 20,000 hospital admissions and emergency room visits, about 30,000 cases of bronchitis (acute and chronic), and hundreds of thousands of cases of respiratory symptoms, lost work days as well as restricted-activity days.

3.4. Spatial variability in BC-related mortality estimates

Fig. 4(a) and (b) shows the annual BC-related mortality and the percent of baseline mortality in 2010 by State, respectively, ranked from the highest values to the lowest. States are ranked differently by total number of deaths and by percent of deaths. Three States, namely, New York, Pennsylvania and New Jersey are ranked among the top 5 States in both rankings (Fig. 4).

Fig. 5(a) and (b) shows the annual BC-related mortality and the percent of baseline mortality in 2010 by county, respectively. Table 2 shows the top 10 ranked counties based on total BC-related deaths versus percent of deaths. Two counties, i.e. Bergen county, NJ and Westchester county, NY are listed among the top 10 counties in both rankings. The top 10 counties ranked based on percent of deaths are all located in the States of New Jersey or New York and surrounding NYC.

3.5. Uncertainty analysis

Uncertainties exist in every component of the health impact assessment, including the concentration–response coefficients, the simulated pollutant concentrations, the population and the baseline incidence rates. A significant source of uncertainty derives from published estimates of concentration–response coefficient, β which we have already taken into account in estimating the national BC-related mortality and morbidity. The wide confidence intervals around the mean estimates of mortality of morbidity show the considerable uncertainty in these estimates (Table 1). Also, the uncertainty associated with the species potency of BC can significantly affect the estimates of BC-related health outcomes. As indicated in Table 1, if we apply a BC-specific risk coefficient, the national total BC-related mortality estimate could be up to ten times larger than the estimate based on $PM_{2.5}$-related risk coefficient. On the other hand, BC-related health impact estimates are also highly sensitive to the assumptions about exposure to ambient BC, which is represented using air quality model output here. In order to investigate how the uncertainty in annual average BC concentrations might alter our best estimate of national BC-related mortality, we repeat our calculation by increasing the BC concentrations by 10%, 20%, 30%, 40% and 50%, and also decreasing the concentrations by the same percentages. The results indicate that the changes in mortality estimate are nearly 100% linearly related to the changes in BC concentrations over the range of BC concentrations relevant to health impact assessments in the US.

Some degree of uncertainty is also associated with air quality model resolution. In this study, we use a global model with a horizontal resolution of approximately 55 km, which is a relatively coarse resolution compared with some regional air quality models, such as the Community Multiscale Air Quality (CMAQ) model that runs with 12 km and 36 km resolutions. Punger and West (2013) examined how estimates of mortality in the US attributable to $PM_{2.5}$ at coarse resolutions differ from those at the finest resolution (12 km). They report that coarse grid resolutions produce mortality estimates that are biased low for $PM_{2.5}$, particularly for primary $PM_{2.5}$ species. More specifically, their analyses show that at the resolution of 55 km that is used in this study, the estimate of mortality attributable to BC was approximately 15% lower than the 12-km resolution estimate. If we apply the bias identified by Punger and West (2013) here, the estimates of national BC-related mortality in this study could be increased by about 15%. This indicates that our estimates of health benefits associated with BC mitigation might be conservative. In contrast, from the comparison of the simulated to observed concentrations (See Fig. 2), the model may overestimate current BC concentrations in the North East and California, hence some cancelation of error owing to emissions and downscaling may be expected.

Overall, we consider the value of concentration–response coefficient contributes the most to the uncertainty in the estimated health outcomes related to BC exposure, particularly premature mortality. Thus, reducing uncertainty in this input variable would substantially reduce the uncertainty in the estimated mortality attributable BC.

4. Conclusions

Using the all-cause mortality risk estimate for $PM_{2.5}$ from Krewski et al. (2009), we estimate that 13,910 (95% CI: 9419–18,391) deaths to result from anthropogenic BC

emissions in 2010. These deaths account for approximately 0.6% (95% CI: 0.4%–0.8%) of the national deaths occurring in the US in 2010. Using morbidity risk estimates for $PM_{2.5}$ drawn from epidemiological studies, we also estimate hundreds of thousands of cases of BC-related illnesses, such as hospital admissions, emergency room visits, bronchitis, and respiratory symptoms.

These health impact estimates are highly sensitive to the assumptions about the risk estimates drawn from published epidemiological studies. Given recent evidence that BC particles may pose a greater risk on human heath than other components of $PM_{2.5}$, we conducted a comprehensive literature review of existing epidemiological studies that report the health effects of both total $PM_{2.5}$ mass and BC particles in a single study. Our review indicates that there is supporting evidence that BC may be more harmful that other constitutes of $PM_{2.5}$ and the $PM_{2.5}$ mass as a whole, but there are also limitations of existing studies to differentiate the health effects of various constituents of $PM_{2.5}$. Based on our review, we performed sensitivity analysis of the national BC-related mortality using a BC-specific risk estimate from a recent review and meta-analysis of the health effects of BC by Janssen et al. (2011). Using the latter risk estimate, we estimate 133,807 (95% CI: 92,259–174,523) BC-related mortality, which is approximately ten time larger than the mortality estimate based on Krewski et al. (2009).

To our best knowledge, this is the first study of its kind that assesses the public health burden of ambient BC at the national, state and county level in the US. Earlier studies focus on the health impacts of total PM mass other than different constituents of PM. A recent study conducted by the U.S. EPA (Fann et al., 2012) estimate 130,000 $PM_{2.5}$-related deaths to result from 2005 ambient $PM_{2.5}$ levels in the US based on the same risk estimate drawn from Krewski et al. (2009) used in this study. Therefore, our estimate of BC-related deaths accounts for approximately 11% of the total $PM_{2.5}$-related deaths. We consider this as a lower bound estimate. When the BC-related mortality is calculated using the risk estimate for BC drawn from Janssen et al. (2011), it could account for up to 100% of the total $PM_{2.5}$ estimate. The uncertainty associated with the concentration–response coefficients of BC might be reduced in future health impact assessments as epidemiological studies focusing on the exclusive long-term health effects of exposure to BC become available.

Despite the uncertainties in our estimates, our findings indicate that controlling BC emissions would have substantial benefits for public health in the US, as indicated by avoided premature deaths and illnesses. Furthermore, our analysis of the spatial variations in BC-related mortality show that the metropolitan areas of Los Angeles, Chicago, New York City, Philadelphia and Detroit are the locations with the greatest numbers of BC-related mortality, and the metropolitan areas of New York City, Philadelphia and Newark are the locations with the greatest percentages of BC-related mortality. Therefore, further investigations of BC emissions, air quality and control measures in these metropolitan areas are warranted.

Acknowledgments

Appendix A

Table A.I

Summary of concentration–response (C–R) coefficients of mortality related to long-term exposure to $PM_{2.5}$ and BC.

Reference	Health endpoint (mortality)	Location and study group	C–R coefficient per 1-µg/m3		BC/$PM_{2.5}$ effect ratio	Coefficient of the correlation between $PM_{2.5}$ and BC
			$PM_{2.5}$	BC		
Janssen et al. (2011)	All-cause	Meta-analysis, including 4 studies: 2 in the US, 1 in France and 1 in Netherlands[2]	0.007	0.058	8.4	N/A
Smith et al. (2010)	All-cause	66 US cities, American Cancer Society cohort, age > 30	0.006	0.058	9.8	N/A
	Cardiopulmonary		0.012	0.104	8.7	
Lipfert et al. (2006)	All-cause	70,000 male, US veterans cohort	0.006	0.166	27.7	0.54
Beelen et al. (2008)	Natural-cause	120,852 adults aged 55–69; The Netherlands,	0.006	0.049	8.2	0.82
	Respiratory		0.007	0.182	26.1	
	Cardiovascular		0.004	0.039	9.8	
Filleul et al. (2005)	Natural-cause	14,284 adults, age 25–59, France	0.010	0.058	5.9	0.87
	Cardiopulmonary		0.012	0.049	4.1	
Ostro et al. (2010)	All-cause	133,479 female school teachers, California	0.077	0.486	6.3	0.84
	Cardiopulmonary		0.091	0.523	5.8	
Gan et al., (2011)	Coronary heart disease	452,735 residents, 45–85 years of age in Vancouver, Canada	0.013	0.039	3	0.13

[2] These four studies are also listed in this Table: Smith et al., 2010; Lipfert et al., 2006; Beelen et al., 2008; Filleul et al., 2005. The concentration–response coefficients of these four studies were converted from the Relative Risks listed in Table 2 in Janssen et al. (2012) rather than drawn directly from the original papers.

Table A.II

Summary of concentration–response (C–R) coefficients of mortality and morbidity related to short-term exposure to $PM_{2.5}$ and BC.

Reference	Health endpoint	Location and study group	C–R coefficient per 1-$\mu g/m^3$		BC/$PM_{2.5}$ effect ratio	Coefficient of the correlation between $PM_{2.5}$ and BC
			$PM_{2.5}$	BC		
Levy et al. (2012)	Meta-analysis: daily all-cause mortality	Including 8 studies: 5 in the US, 2 in UK and 1 in Canada	0.0012	0.0004	0.33	N/A
Klemm et al., (2004)	Daily all-cause mortality, age > 65	Atlanta	0.00398	0.01024	2.6	N/A
Ostro et al. (2007)	Daily CVD mortality	Six California counties	0.0011	0.026	23.9	0.53
Ito et al. (2010)	Daily CVD mortality	New York City	0.002 (warm season), 0.001 (cold season)	0.037 (warm season), 0.026 (cold season)	18.5 (warm season), 26 (cold season)	0.6 (warm season), 0.72 (cold season)
	Daily CVD hospitalization	New York City	N/A (warm season), 0.0011 (cold season)	0.023 (warm season), 0.024 (cold season)	22 (cold season)	
Metzger et al. (2004)	Daily CVD emergency department visits	Atlanta	0.0032	0.02	6.1	0.61
Zanobetti and Schwartz (2006)	Daily CVD hospital admissions	Boston	0.0053	0.049	9.3	0.66
Wang et al. (2013)	Emergency-room visits	Shanghai, China	0.00045	0.0049	11.1	0.67
Hua et al. (2014)	Children asthma admissions	Shanghai, China	0.00032 (age 0–4), 0.0028 (age 5–14)	0.016 (age 0–4), 0.038 (age 5–14)	50 (age 0–4), 14 (age 5–14)	0.80

CVD: cardiovascular.

Table A.III

National rates of all-cause mortality, hospital admissions and emergency room visits (per 100 people per year) by age group used in this study (Source: Abt Associates Inc., 2013(Abt Associates, 2013)).

Health endpoints and year	Infant (<1)	1–17	18–24	25–34	35–44	45–54	55–64	65–74	75–84	85+
Mortality										
Calculated CDC 2004–2006 average	0.230	0.028	0.089	0.104	0.192	0.432	0.908	2.124	5.270	13.979
2010 ratio to the calculated 2004–2006 average	0.95	0.93	0.95	0.96	0.94	0.95	0.96	0.93	0.95	0.96
Hospital admissions and emergency room visits										
Hospital admissions – all respiratory, 2007	2.872	0.426	0.205	0.231	0.408	0.791	1.313	2.972	4.970	8.045
Hospital admissions – all cardiovascular, 2007	0.056	0.017	0.107	0.164	0.481	1.221	2.272	4.681	7.749	11.583
Emergency room visits for asthma, 2007	0.865		0.557			0.441		0.381		0.368

Table A.IV

Selected acute and chronic incidence (cases/person-year) (Source: Abt Associates Inc., 2013(Abt Associates, 2013)).

Endpoint		Age	Rate	Source
Acute bronchitis		8–12	0.043	American Lung Association
Chronic bronchitis		27+	0.00378	Abbey et al. (1993)
Lower respiratory symptoms		7–14	0.438	Schwartz et al. (1994)
Upper respiratory symptoms		9–11	124.79	Pope III et al. (1991)
Asthma exacerbation	Cough	8–13	24.46	Ostro et al. (2001)
	Shortness of breath		27.74	
	Wheeze		13.51	
Work loss days		18–64	2.172	Adam et al. (1999)
Minor restricted-activity days		18–64	7.8	Ostro and Rothschild (1989)

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HIGHLIGHTS

- Black carbon (BC) is a strong climate warming forcing agent.

- This study quantifies the public health burden attributed to BC exposure in the US.

- We use an integrated assessment approach to estimate BC-related health impacts.

- Our findings suggest substantial health benefits to be resulted from BC control.
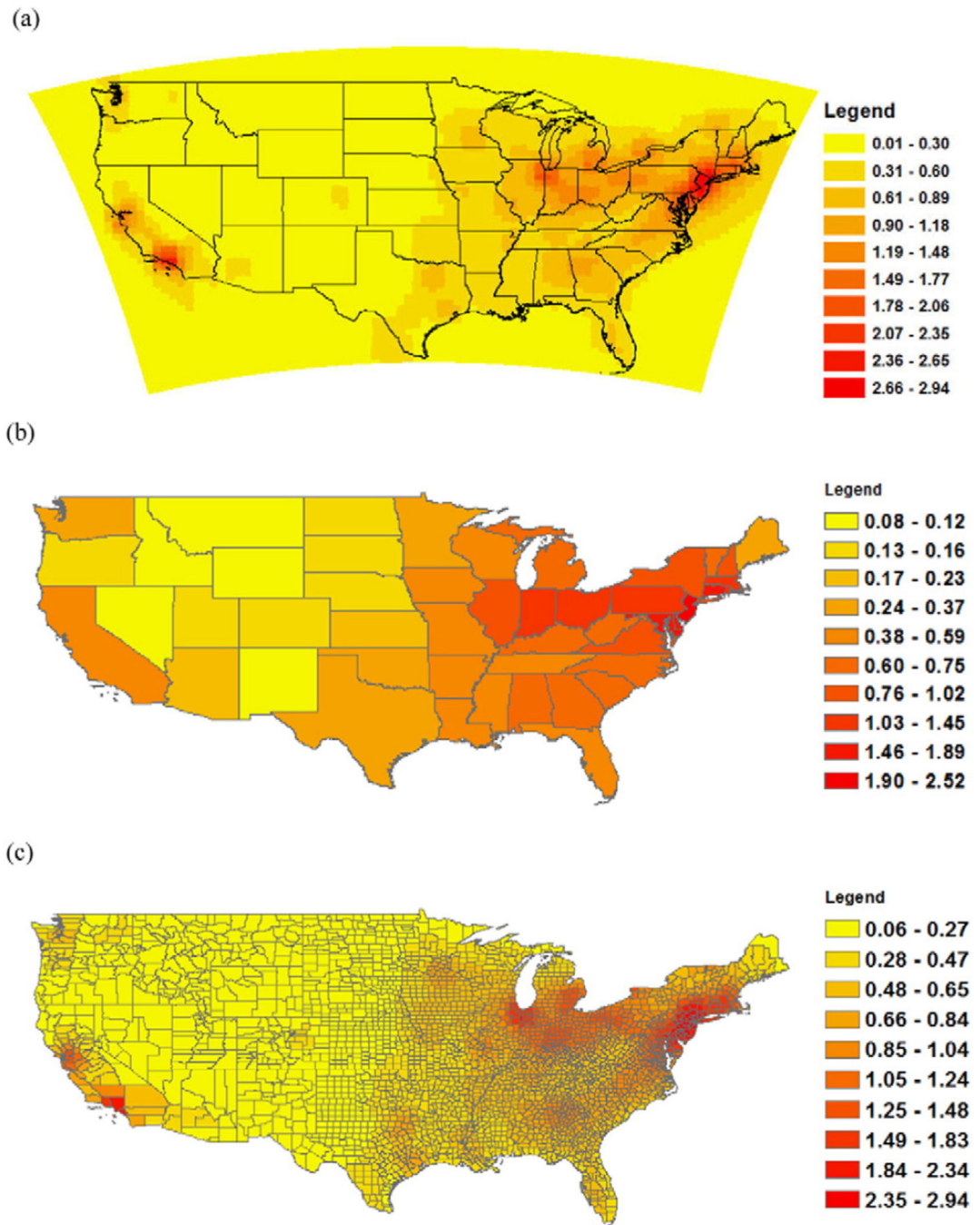
Fig. 1.
Simulated annual average black carbon concentrations in 2010: (a) At $0.5 \times 0.667°$ resolution; (b) Aggregated to State; and (c) Aggregated to county (Unit: μg/m^3).
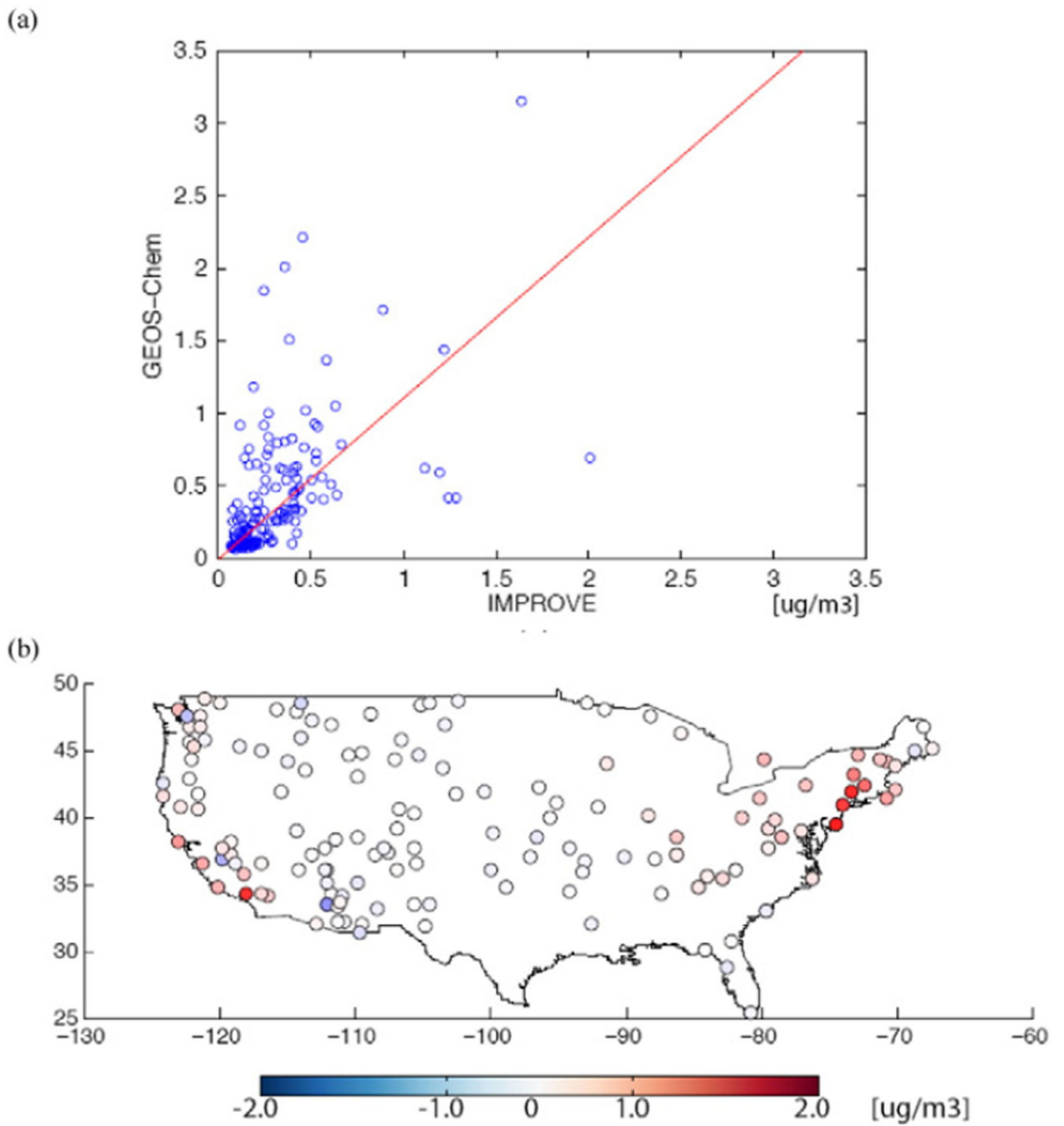
(a)

(b)

Fig. 2.
Evaluation of the modeled BC concentrations compared to measurements: (a) Results of a regression fit; and (b) The spatial plot of the model error.
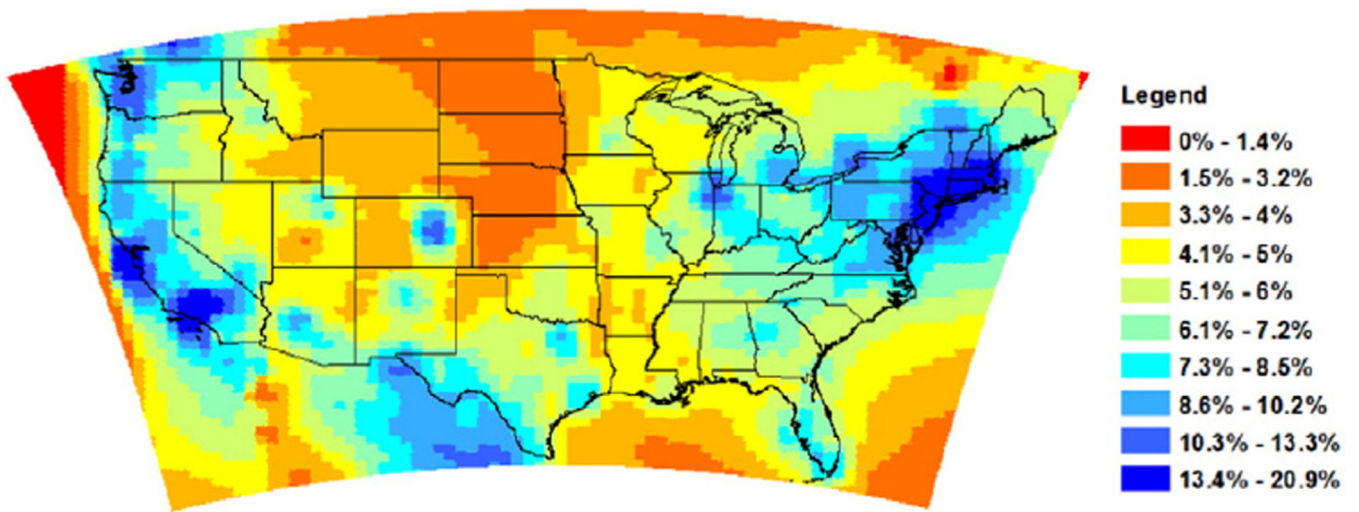
Fig. 3.
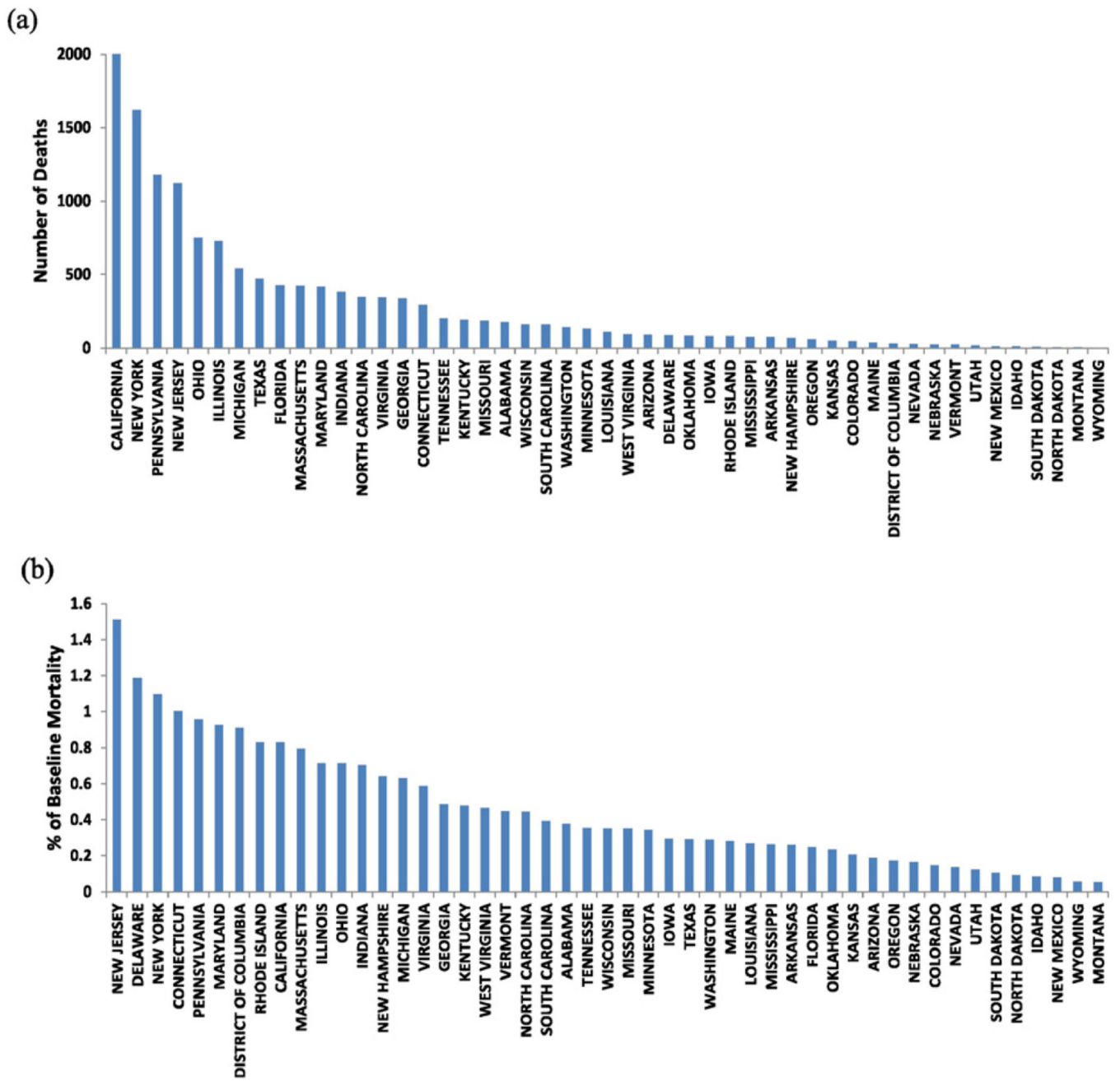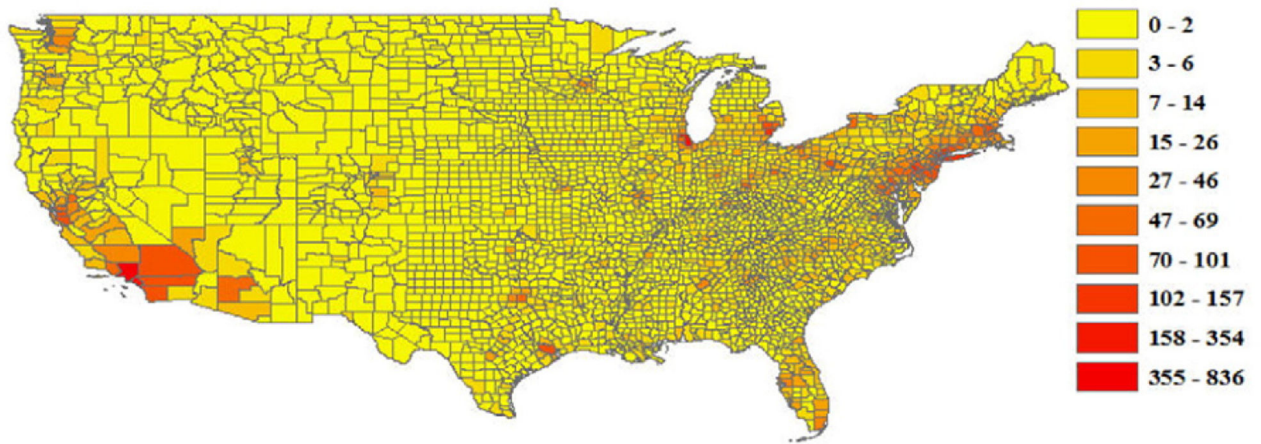Ratio of BC to PM$_{2.5}$ annual average concentration in 2010.

Fig. 4.
Ranking annual BC-related mortality by State: (a) Ranked by annual number of death; (b)
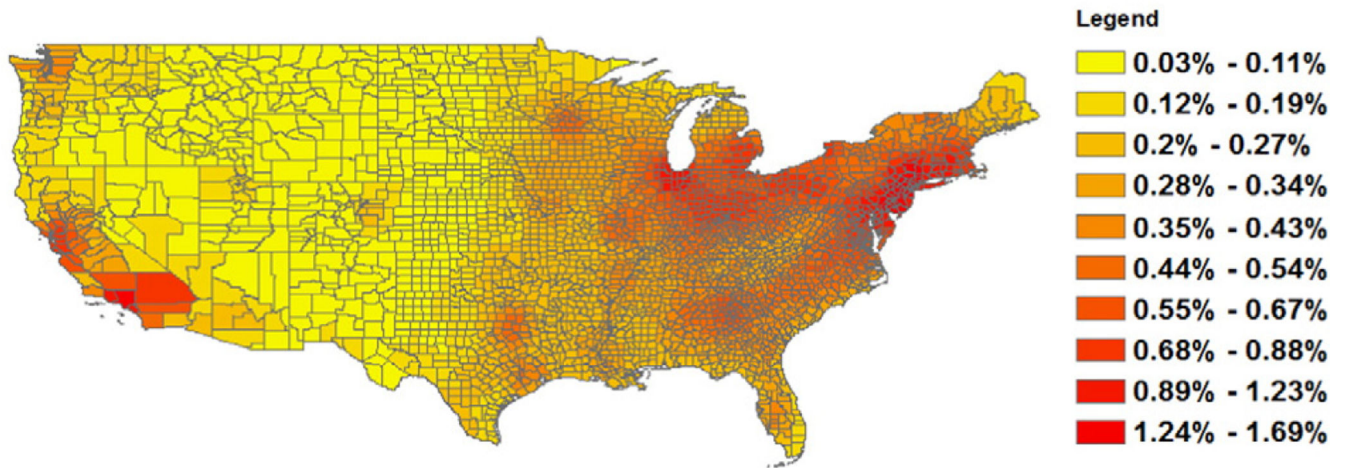Ranked by percent of baseline mortality.

(a)

(b)

Fig. 5.
Spatial distribution of annual BC-related mortality at the county level: (a) Number of deaths; and (b) Percent of baseline mortality.

Table 1

Estimated black carbon-related health impacts in the U.S. due to 2010 modeled air quality.

Health endpoint	PDF of concentration–response coefficient (β)	Reference of β (age group)	Annual estimates (95% Confidence interval)
Mortality			
Adult all-cause	Normal (0.005827, 0.000963)	Krewski et al. (2009) (age 30)	13,910 (9419–18,391)
	Normal (0.05827, 0.009524)	Janssen et al. (2011) (age 30)	133,807 (92,259–174,523)
	Uniform (0.005827, 0.05827)	Krewski et al. (2009) &	74,689 (17,026–131,050)
	Triangular (0.005827, 0.05827)	Janssen et al. (2011) (age 30)	74,882 (27,791–120,969)
Infant	Normal (0.006766, 0.007339)	Woodruff et al. (1997) (age b1)	32 (13–52)
Morbidity			
Hospital admissions – all respiratory	Normal (0.00207, 0.0004464)	Zanobetti et al. (2009) (age N64)	3380 (1952–4805)
Hospital admissions – all cardiovascular	Normal (0.00189, 0.0002832)	Zanobetti et al. (2009) (age N64)	4344 (3070–5618)
Emergency room visits for asthma	Normal (0.0056, 0.0021)	Mar et al. (2010) (all ages)	10,567 (2804–18,290)
Acute bronchitis	Normal (0.02721, 0.017)	Dockery et al. (1996) (ages 8–12)	20,913 (−4983–45,928)
Chronic bronchitis	Normal (0.0137, 0.006796)	Abbey et al. (1994) (age N27)	9604 (269–18,799)
Lower respiratory symptoms	Normal (0.019, 0.006)	Schwartz and Neas (2000) (ages 7–14)	269,180 (103,588–432,608)
Upper respiratory symptoms	Normal (0.0036, 0.0015)	Pope III et al. (1991) (ages 9–11)	389,951 (71,557–708,006)
Asthma exacerbation Cough	Normal (0.00099, 0.00075)	Ostro et al. (2001) (ages 6–18)	253,806 (−123,529–630,696)
Shortness of breath	Normal (0.0026, 0.0013)		364,800 (−7312–735,972)
Wheeze	Normal (0.0019, 0.0008)		577,220 (109,712–1,044,186)
Work loss days	Normal (0.0046, 0.00036)	Ostro (1987) (ages 18–64)	1,910,323 (1,618,200–2,202,217)
Minor restricted-activity days	Normal (0.007410, 0.000700)	Ostro and Rothschild (1989) (ages 18–64)	11,332,839 (9,244,613–13,417,870)

Table 2

Top 10 ranked counties based on the number of BC-related deaths versus percent of deaths.

Rank	State	County	Largest city	Number of death	State	County	Largest city	% of deaths
1	CA	Los Angeles	Los Angeles	836	NJ	Bergen	NYC Metro	1.695%
2	IL	Cook	Chicago	354	NJ	Passaic	NYC Metro	1.689%
3	NY	Queens	Queens	247	NY	Rockland	NYC Metro	1.684%
4	NY	Kings	Brooklyn	232	NY	Bronx	Bronx	1.674%
5	CA	Orange	Los Angeles	220	NY	Westchester	Yonkers	1.634%
6	PA	Philadelphia	Philadelphia	157	NJ	Somerset	NYC Metro	1.615%
7	NJ	Bergen	NYC Metro	155	NJ	Mercer	Philadelphia Metro	1.612%
8	NY	Nassau	Long Island	145	NY	New York	Manhattan	1.612%
9	MI	Wayne	Detroit	142	NJ	Morris	NYC Metro	1.600%
10	NY	Westchester	Yonkers	132	NJ	Essex	Newark	1.587%