Impacts of global, regional, and sectoral black carbon emission reductions on surface air quality and human mortality

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Abstract

As a component of fine particulate matter (PM$_{2.5}$), black carbon (BC) is associated with premature human mortality. BC also affects climate by absorbing solar radiation and reducing planetary albedo. Several studies have examined the climate impacts of BC emissions, but the associated health impacts have been studied less extensively. Here, we examine the surface PM$_{2.5}$ and premature mortality impacts of halving anthropogenic BC emissions globally, from eight world regions, and from three major economic sectors. We use a global chemical transport model, MOZART-4, to simulate PM$_{2.5}$ concentrations and a health impact function to calculate premature cardiopulmonary and lung cancer deaths. We estimate that halving global anthropogenic BC emissions reduces outdoor population-weighted average PM$_{2.5}$ by 542 ng m$^{-3}$ (1.8%) and avoids 157,000 (95% confidence interval, 120,000–194,000) annual premature deaths globally, with the vast majority occurring within the source region. While most of these avoided deaths can be achieved by halving East Asian emissions (54%), followed by South Asian emissions (31%), South Asian emissions have 50% greater mortality impacts per unit BC emitted than East Asian emissions. Globally, the contribution of residential, industrial, and transportation BC emissions to PM$_{2.5}$-related mortality is 1.3, 1.2, and 0.6 times each sector’s contribution to anthropogenic BC emissions, owing to the degree of co-location with population. Impacts of residential BC emissions are underestimated since indoor PM$_{2.5}$ exposure is excluded. We estimate ∼8 times more avoided deaths when BC and organic carbon (OC) emissions are halved together, suggesting that these results greatly underestimate the full air pollution-related mortality benefits of BC mitigation strategies which generally decrease both BC and OC. Confidence in our results would be strengthened by reducing uncertainties in emissions, model parameterization of aerosol processes, grid resolution, and PM$_{2.5}$ concentration-mortality relationships globally.
1 Introduction

Black carbon (BC) is a component of fine particulate matter (PM$_{2.5}$) produced by incomplete combustion of fuel and is mainly emitted by residential, transportation, and industrial sources (Bond et al., 2004). PM$_{2.5}$ is associated with negative health impacts, including premature mortality (e.g. Krewski et al., 2009), and some evidence suggests that PM$_{2.5}$ mixtures containing high BC fractions may have larger mortality effects than other mixtures (Smith et al., 2009). BC also warms the atmosphere by absorbing solar radiation (e.g. Horvath, 1993), indirectly impacts cloud lifetime and reflectivity (Koch and Del Genio, 2010), and deposits on snow and ice, reducing albedo and quickening melting (e.g. Hansen and Nazarenko, 2004). Although the net effects of BC on climate remain uncertain, mitigation of BC emissions offers an opportunity to address climate change and air pollution simultaneously (e.g. Jacobson, 2002; Bond and Sun, 2005; Ramanathan and Carmichael, 2008; Kopp and Mauzerall, 2010). Both climate and health benefits should therefore be considered when evaluating mitigation strategies.

While several recent studies have examined the climate impacts of BC emissions (e.g. Koch et al., 2007; Reddy and Boucher, 2007; Levy et al., 2008; Shindell et al., 2008; Fuglestvedt et al., 2010), the associated health impacts have been studied less extensively. In contrast with direct atmospheric warming by BC absorption of sunlight, which depends on total column BC concentrations, BC health impacts depend on population exposure at the surface, where humans breathe. In addition, the net climate impacts of BC emitting sources can be offset by co-emitted PM$_{2.5}$ components that reflect radiation, such as organic carbon (OC) and sulfate (SO$_4$; Unger et al., 2010), but all PM$_{2.5}$ components are thought to be damaging to health. Since the drivers for climate and health impacts of BC emissions differ, mitigation strategies that achieve the greatest near-term climate benefits may not yield the greatest co-benefits for public health, and vice versa.
Previous studies of the health impacts of outdoor air pollution have found that the greatest burden of outdoor air pollution on human health occurs in Asia, where large populations are exposed to high ozone and PM$_{2.5}$ concentrations (Cohen et al., 2004; Anenberg et al., 2010), and that biofuel combustion causes eight times more premature deaths globally than fossil fuel emissions, largely because biofuel combustion occurs mainly in very populated regions of the world (Jacobson, 2010). Surface BC concentrations, specifically, have also been found to be highest over East Asia, South Asia, and Southeast Asia (e.g. Koch et al., 2009). The co-location of high BC concentrations and large populations may translate into a substantial impact on global public health, and significant potential benefits of BC mitigation.

Quantifying the health benefits of BC emission reductions and their variation by source region and sector may inform strategies to mitigate near-term climate change and air pollution simultaneously. Here, we calculate the surface air quality and premature human mortality impacts of halving anthropogenic BC emissions globally, regionally, and from three major economic sectors. We also examine a scenario in which BC and OC emissions are reduced together, as they are co-emitted and likely to both be affected by BC mitigation strategies. We simulate PM$_{2.5}$ concentration changes with a global chemical transport model (CTM) and calculate mortality changes using a health impact function based on epidemiologically-derived concentration-response relationships.

2 Methods

2.1 Model setup

We simulate a base case and several sensitivity cases using the global CTM MOZART-4 (Model of Ozone And Related Tracers, version 4; Emmons et al., 2010b). MOZART-4 has 85 gas-phase species and 39 photolysis and 157 gas-phase reactions. The representation of tropospheric aerosols includes sulfate (SO$_4$; assumed here to exist...
as ammonium sulfate), BC, OC, secondary organic aerosol (SOA), ammonium nitrate (NO$_3$), and sea salt (Emmons et al., 2010b). An online photolysis scheme accounts for the impact of aerosols on photolysis rates, affecting production of photochemical oxidants (Tie et al., 2005; Emmons et al., 2010b). Here, MOZART-4 is configured with a horizontal resolution of 1.9° × 1.9°, 28 vertical levels, and is driven with meteorology from the NCEP/NCAR reanalysis (6 h timestep; Kalnay et al., 1996; Kistler et al., 2001) for the year 2002 with six months of spinup starting in July 2001. BC and OC are initially emitted as 80% and 50% hydrophobic and are converted to hydrophilic with a time constant of 1.6 days to simulate the gradual coating of hydrophobic particles with sulfate and other compounds (Emmons et al., 2010b). Dry deposition of BC and OC is set to 0.1 cm s$^{-1}$ and wet deposition of hydrophilic BC and OC is set to 20% of the wet deposition rate of HNO$_3$ (Tie et al., 2005; Emmons et al., 2010b).

Anthropogenic BC and OC emissions for the year 2000 are from the inventory developed for the Intergovernmental Panel on Climate Change Fifth Assessment Report (IPCC AR5), based on Bond et al. (2007) and Junker and Lioussse (2008) with updates described by Lamarque et al. (2010). Since this inventory includes only submicron particles, we multiply BC and OC emissions by 1.15 and 1.4 to include all particles up to 2.5 µm in diameter for these species (Cooke et al., 1999), as in Liu et al. (2009a). The IPCC AR5 inventory reports emissions from the residential, transportation, and industrial sectors (includes non-road transportation), and smaller sectors, such as waste treatment, agricultural waste burning, and shipping. The residential, industrial, and transportation sectors together comprise >90% of the total anthropogenic BC inventory (Fig. 1). As BC and OC are generally co-emitted with CO (e.g. Dickerson et al., 2002), we vary these emissions monthly by scaling to the monthly profile of CO from the RETRO emissions inventory (http://retro.enes.org/data_emissions.shtml), which does not include BC and OC, in each grid cell and sector (Figs. S1–S2).

Anthropogenic emissions of all other species are those used by Emmons et al. (2010b), which are from the POET (Precursors of Ozone and their Effects in the Troposphere; Olivier et al., 2003; Granier et al., 2005) inventory for the year 2000, with
Asian emissions replaced by the annually varying Regional Emissions Inventory in Asia (REAS; Ohara et al., 2007) (Table S1). Biomass burning emissions for all species are from the Global Fire Emissions Database version 2 (GFED2; van der Werf et al., 2006) for the specific months in 2001 and 2002 modeled here. To account for emission altitude and plume buoyancy, we distribute all biomass burning emissions vertically up to 6 km above the surface using the vertical profile from Dentener et al. (2006). All other emissions are injected at the surface, including power generation (<1% of global BC emissions) and industrial emissions, which include non-road transportation and small sources with no smokestacks.

Relative to this base case, we calculate changes in PM$_{2.5}$ concentration and mortality for 12 sensitivity cases wherein anthropogenic BC emissions are halved globally, individually in eight major world regions (North America, South America, Europe, the Former Soviet Union, Africa/Middle East, South Asia (India), East Asia (China), and Southeast Asia/Australia) plus in the United States (US) alone, and individually in three major economic sectors (residential, industrial, transportation). These regions are defined in Table 1 and Fig. S3. We also examine a scenario in which global anthropogenic BC + OC emissions are halved together, since they are co-emitted. Because each source emits BC and OC in different ratios and each control measure may reduce them in different percentages, this experiment is meant to be illustrative of including OC reductions and does not represent the impacts of actual mitigation measures. We use simulated concentrations in the first vertical level (height = ~80 m) as surface concentrations. We multiply simulated hydrophobic and hydrophilic OC concentrations by 1.3 and 1.7 to account for associated species other than carbon (Ming et al., 2005) and add this to SOA to give total organic mass (OM). We estimate PM$_{2.5}$ as BC + OM + SO$_4^-$+NO$_3^-$, assuming that these species exist entirely as PM$_{2.5}$ and ignoring other species (dust and sea salt) that are dominated by natural emissions and are unaffected by changing BC and OC emissions.
2.2 Evaluation of base case surface concentrations

Simulated annual average PM$_{2.5}$ concentrations are highest in EA and IN, due to large anthropogenic and biomass burning emissions (Table 1 and Figs. S4–S8). BC is 3–5% of total PM$_{2.5}$ among world regions and OM is 14–46% (Fig. 2 and Table S2). SO$_4$ generally contributes the most to PM$_{2.5}$, except for IN and EA, where OM and NO$_3$ also contribute significantly, and SE/AU, where OM is high due to wildfires. Population-weighted average BC and NO$_3$ concentrations are generally larger than simple average concentrations (Table S2), indicating closer co-location with population compared with OM and SO$_4$, which are more widespread.

MOZART-4 has been evaluated extensively against satellite data and measurements at altitude and at the surface by Emmons et al. (2010b) and Tie et al. (2005), who comprehensively evaluated the aerosol scheme specifically. Here we focus on changes in PM$_{2.5}$ components at the surface, and therefore evaluate simulated BC, OC, and SO$_4$ concentrations in the first vertical level. Since modeled concentrations are volume averages over large grid cells, they are expected to represent concentrations in remote locations that are more homogenous better than in urban areas. We therefore compare simulated concentrations to surface observations in remote locations from the Interagency Monitoring of Protected Visual Environments (IMPROVE; http://vista.cira.colostate.edu/improve/) network for the US and the European Monitoring and Evaluation Programme (EMEP; http://www.emep.int/) network for Europe. Surface observations outside of the US and Europe are limited. We compare modeled BC and OC with observations at 15 locations in China (Zhang et al., 2008) and eight in India (Beegum et al., 2009) from 2006. Each of these monitoring networks reports elemental carbon (EC), which is measured by optical rather than thermal-optical techniques and may be between 30% and 100% of BC; since the emissions inventory is more representative of EC than BC, comparing modeled concentrations with EC is most appropriate (Vignati et al., 2010). For OC, we compare observations with simulated concentrations prior to conversion to OM, and include SOA. We compare SO$_4$
prior to mass conversion to ammonium sulfate. Although we simulate 2002 for our base case, for the model evaluation only, we ran the base case through 2003 to leverage additional observations from IMPROVE (available for both 2002 and 2003) and EMEP (available for July 2002–June 2003).

Compared with observations from IMPROVE (average 2002—2003) for the US (Figs. 3 and S9), BC is generally simulated within a factor of two (inside the dashed lines), with some simulated concentrations higher than observations in the Northwest, California, and Northeast, and lower than observations in the South. Simulated OC is generally lower than observations, particularly in the Southeast, likely due to unrealistically low simulated SOA concentrations, consistent with previous studies using MOZART-4 (Dunlea et al., 2009; Emmons et al., 2010a). However, simulated OC is higher than observations in the Northwest. Simulated SO$_4$ generally matches observations in the East, but is higher than observations in the West (Fig. S10). Compared with observations from EMEP in Europe (average July 2002 to July 2003), simulated BC concentrations are generally lower than observations, particularly in the West (Figs. 3 and S9). As for IMPROVE, simulated OC is lower than observations in Europe (Figs. 3 and S9) and simulated SO$_4$ is higher than observations (Fig. S10). For the few observations available from China and India, simulated concentrations are within a factor of two of observations in remote locations, but are lower than observations in regional and urban locations (Figs. S11 and S12). Some of these discrepancies may be due to differences between volume averaged modeled concentrations in large grid cells and point measurements (Swall and Foley, 2009; Gilardoni et al., 2011), or the mismatch between 2006 measurements in Asia and 2002 simulated concentrations. Measurement methods may also cause overestimation of ambient EC concentrations (Novakov et al., 2005), with the reflectance method for IMPROVE potentially measuring higher EC concentrations than the transmittance method used by EMEP (Chow et al., 2001).

Simulated simple (population-weighted) regional average surface NO$_3$ concentrations range as high as 10.6 (30.6) µg m$^{-3}$ in EA and 6.3 (15.1) µg m$^{-3}$ in IN (Fig. 2 and Table S2). While a lack of global NO$_3$ measurements limits our ability to evaluate
simulated concentrations, these concentrations are consistent with those simulated by other global CTMs (e.g. Park et al., 2004). Any biases in simulated NO\textsubscript{3} concentrations would not significantly affect our results, since NO\textsubscript{3} varies little among the BC emission scenarios.

2.3 Health impact function

We calculate avoided premature deaths in each grid cell using the change in simulated PM\textsubscript{2.5} concentration between the base case and the emission reduction scenario and a health impact function (HIF). Global CTMs have been used previously to estimate mortality due to total anthropogenic air pollution (Anenberg et al., 2010), long-range transport of air pollution (Duncan et al., 2008; Anenberg et al., 2009; Liu et al., 2009a; West et al., 2009), future changes in emissions (West et al., 2006, 2007; Selin et al., 2009), changes in one sector's emissions (Corbett et al., 2007; Barrett et al., 2010), fossil fuel and biofuel emissions (Jacobson, 2010), and air pollution changes associated with carbon dioxide emissions (Jacobson, 2008).

Here we use a log-linear relationship between long-term PM\textsubscript{2.5} exposure and relative risk (RR), following Anenberg et al. (2010). RR > 1 indicates that PM\textsubscript{2.5} exposure increases risk of mortality. We use RR to calculate \( \beta \), the concentration-response factor (CRF), and, as shown in Eq. (1), the attributable fraction (AF), the fraction of the disease burden attributable to the change in annual average PM\textsubscript{2.5}(\( \Delta X \)). This HIF is applied in each grid cell by multiplying the AF by the baseline mortality rate (\( y_0 \)) and exposed population (Pop), as shown in Eq. (2).

\[ AF = (RR - 1)/RR = 1 - \exp^{-\beta \Delta X} \]  
\[ \Delta \text{Mort} = y_0 (1 - \exp^{-\beta \Delta X}) \text{Pop} \]  

Some evidence suggests that air pollution mixtures with high BC fractions, “black smoke,” “diesel PM\textsubscript{2.5},” and “traffic PM\textsubscript{2.5},” have stronger associations with mortality than other mixtures (Cooke et al., 2007; Brunekreef et al., 2009). Studies that use
ambient BC concentrations as a marker for air pollution mixtures also find stronger associations with mortality than those using total PM$_{2.5}$ (Ostro et al., 2007, 2008; Bell et al., 2009; Peng et al., 2009; Smith et al., 2009). These studies are subject to substantial exposure error since BC is very spatially heterogeneous, potentially resulting in risk underestimation (Smith et al., 2009; Bell et al., 2010). However, evidence for differential toxicity of BC and BC-containing mixtures remains inconclusive. We therefore assume that all mixtures of PM$_{2.5}$ are equally potent in causing premature mortality, and use the change in total PM$_{2.5}$ in Eq. (2).

We calculate CRFs using estimates of RR of chronic mortality due to total PM$_{2.5}$ from Krewski et al. (2009), the latest reanalysis of the American Cancer Society PM$_{2.5}$ studies (e.g. Pope et al., 2002) and the largest among long-term PM$_{2.5}$ mortality studies (e.g. Laden et al., 2006). For a 10 µg m$^{-3}$ increase in PM$_{2.5}$, RR was 1.06 (95% CI, 1.04–1.08), 1.13 (95% CI, 1.10–1.16), and 1.14 (95% CI, 1.06–1.23) for total, cardiopulmonary, and lung cancer mortality in adults age 30+. These RRs were determined for the observed range of concentrations, 5.8–22.2 µg m$^{-3}$, and the linearity of the concentration-response relationship was also demonstrated up to 30 µg m$^{-3}$ based on 1979–1983 PM$_{2.5}$ data. Causes of death differ globally from those in the US, and we estimate cardiopulmonary and lung cancer mortality, as they are more comparable around the world than all-cause mortality. We assume these CRFs apply globally, despite differences in health status, lifestyle, age structure, and medical care among global populations.

We use baseline cardiopulmonary and lung cancer mortality rates from the World Health Organization (WHO, 2004, 2008), population from the LandScan database (Oak Ridge National Laboratory, 2008) from 2006 (Fig. S13), and the fraction of the population age 30+ (WHO, 2004) to be consistent with Krewski et al. (2009), as described by Anenberg et al. (2010; see Table S3).
3 Results

3.1 Global emission reductions

Halving global anthropogenic BC emissions reduces the global annual average PM$_{2.5}$ concentration by 33 ng m$^{-3}$ (1.2%), ranging among regions from 43 ng m$^{-3}$ in NA to 453 ng m$^{-3}$ in EA (Table 1 and Fig. 4). Population-weighted average PM$_{2.5}$ concentrations decrease by 542 µg m$^{-3}$ (1.8%) globally, ranging from 126 ng m$^{-3}$ in AF/ME to 1201 ng m$^{-3}$ in EA. Regional BC concentrations decrease 25–49%, with smaller percentage reductions in regions with frequent wildfires (e.g. SA, AF/ME, SE/AU; Figs. 5a and S14). Wildfires are assumed to be natural and are therefore excluded from the emission reduction. We estimate that these PM$_{2.5}$ reductions would avoid $\sim$157 000 (95% CI, 120 000–194 000) annual premature deaths worldwide (Table 1 and Fig. 6). In all regions except NA which has a relatively high baseline lung cancer mortality rate, >90% of avoided deaths are from cardiopulmonary disease. Over 80% of global avoided deaths occur in EA, with 81 000 (95% CI, 61 000–100 000) avoided deaths, and IN, with 48 000 (95% CI, 37 000–59 000). These regions have large emissions and exposed populations, and IN also has high baseline cardiopulmonary mortality rates.

Estimated reductions in PM$_{2.5}$ concentrations are generally smaller than reductions in BC concentrations due to increased SO$_4$ production (Fig. 5a). Aerosols affect gas-phase chemistry by absorbing or scattering radiation that drives photochemistry (He and Carmichael, 1999; Liao et al., 1999; Castro et al., 2001). Previous studies using regional models (Jacobson, 1998; Li et al., 2005) and global models (Martin et al., 2003), including using the aerosol scheme used in MOZART-4 specifically (Tie et al., 2005), find that high BC concentrations lead to reduced photolysis rates for O$_3$ and nitrogen dioxide and, therefore, reduced O$_3$ concentrations. Here, we find that reduced BC concentration increases photolysis and leads to increased concentrations of O$_3$, hydroxyl radical (OH), and hydrogen peroxide (H$_2$O$_2$), as shown in Fig. S15. As described by Tie et al. (2001), gas-phase production of SO$_4$ in MOZART-4 occurs via
Reaction (R1), while aqueous phase (in-cloud) production occurs via Reactions (R2) and (R3), where S(IV) is total dissolved sulfur (HSO₃ and SO₃).

\[ \text{SO}_2 + \text{OH} \rightarrow \text{SO}_4 \]  \hspace{1cm} (R1)

\[ \text{S(IV)} + \text{O}_3 \rightarrow \text{SO}_4 + \text{products} \]  \hspace{1cm} (R2)

\[ \text{S(IV)} + \text{H}_2\text{O}_2 \rightarrow \text{SO}_4 + \text{products} \]  \hspace{1cm} (R3)

Through these reactions, increased concentrations of OH, O₃, and H₂O₂ in response to BC emission reductions lead to enhanced SO₄ production. The resulting SO₄ increases are very small percentages of total PM₂.₅ (up to 0.7% in EA; Fig. S14) but since SO₄ concentrations are much larger than BC concentrations, they can offset up to 28% (in EA) of regional BC reductions (Fig. 5a). We include changes to all PM₂.₅ species in our mortality calculation, but do not include O₃ increases.

Halving global anthropogenic BC and OC emissions together reduces BC concentrations by the same amount as halving BC emissions alone, but OM is also reduced (Fig. 5b), such that annual average PM₂.₅ reductions are larger by a factor of four in NA to over eight in IN (Table 1). These PM₂.₅ reductions are associated with ∼8 times more annual avoided deaths than is estimated for halving BC alone (Table 1). Here, changes in radiation absorption by BC and scattering by OM, which can increase the pathlength of solar radiation and accelerate photochemistry (e.g. Dickerson et al., 1997), have opposing effects on gas-phase chemistry. We find increases in OH and O₃ concentrations but decreases in H₂O₂ concentrations, resulting in mixed effects on SO₄ (Fig. S16).

Estimated avoided deaths from halving anthropogenic BC and BC + OC are consistent with estimates by Anenberg et al. (2010) of the global burden of anthropogenic PM₂.₅ on mortality (∼3.7 million deaths) when multiplied by the fraction of PM₂.₅ that is BC (9%, 300 000 deaths) and OM (46%, 1.7 million deaths) in that study. The global burden estimate is twice our estimated deaths from halving BC, but only 1.6 times those from halving OC (1.21 million from halving BC + OC minus 0.16 million from halving
5.2 Regional BC emission reductions

We now examine the surface PM$_{2.5}$ and mortality impacts of halving anthropogenic emissions in eight world regions individually. EA contributes 32% of global anthropogenic BC emissions, more than double the contribution from any other region (Fig. 1). AF/ME and IN follow, with 13% and 12%.

We find the greatest reductions in population-weighted PM$_{2.5}$ occur within the EA (1262 ng m$^{-3}$) and IN (733 ng m$^{-3}$) source regions, due to high emissions (Table 2). For all regions, the contribution of BC emissions from other regions to surface PM$_{2.5}$ concentrations is very small, although some transport occurs between FSU, AF/ME, and EU, which are close in proximity. For some regions, halving BC emissions leads to small increases in OH, O$_3$, and H$_2$O$_2$ in distant regions, which enhance SO$_4$ production where SO$_2$ emissions are high, such as in EA (Figs. S17 and S18). Since BC concentrations are not greatly impacted outside of the source region, small SO$_4$ increases in distant regions can lead to overall PM$_{2.5}$ increases (Table 2). For example, halving BC emissions in IN increases population-weighted average PM$_{2.5}$ by 20 ng m$^{-3}$ in EA. The limited influence of extra-regional BC emissions on regional surface PM$_{2.5}$ concentrations is consistent with other studies finding that BC comprises 0–3% of background surface aerosol concentrations (Liu et al., 2009b) and that foreign BC emissions contribute 0–5% of regional surface BC concentrations (TF HTAP, 2010).

Halving BC emissions in EA avoids 85 000 (95% CI, 64 000–105 000) annual premature deaths globally, more than any other region, followed by IN with 47 000 (95% CI, 36 000–58 000). EA and IN contribute 53% and 31% to all avoided deaths from halving global BC emissions, 1.6 and 2.5 times greater than their contributions to global
anthropogenic BC emissions, owing to large populations in both regions and high cardiopulmonary mortality rates in IN. The small SO$_4$ increases in distant regions resulting from some regional BC emission reductions have very little impact on mortality. To compare mortality impacts of BC emissions across regions, we calculate the mortality impacts per unit BC emissions reduced (Fig. 7). Per unit emission, the mortality impact of BC emissions is largest for IN (136 premature deaths avoided per Gg BC emitted), followed by EA (90). This is likely due to smaller per-unit impacts of within-region emission reductions on BC concentration for EA (Table 2) and higher baseline cardiopulmonary mortality rates in IN (Table S3).

Halving NA emissions reduces PM$_{2.5}$ in that region by 151 ng m$^{-3}$ and avoids 4000 (95% CI, 3000–5000) annual premature deaths (12 per Gg BC reduced), 91% of which occur within the US. Compared with halving BC emissions in the US only, halving all NA emissions causes avoided deaths in NA to increase by 12% and in the US by 1.6%, mostly in the Northeast and California near national borders (Fig. S19).

### 3.3 Sectoral BC emission reductions

We next examine the impacts of halving global BC emissions from each major economic sector individually. Globally, 93% of anthropogenic BC emissions are estimated to be from three sectors: residential (38%), industrial (includes non-road transportation; 29%), and transportation (on-road only; 26%) (Fig. 1). Each sector’s contribution to total anthropogenic BC emissions differs considerably by region, with transportation emissions estimated to contribute most in developed regions (55% in NA and 53% in EU), and the residential sector contributing most in developing regions (62% in IN and 56% in both AF/ME and FSU). In EA, 50% and 35% are from the industrial and residential sectors.

Globally, halving residential BC emissions impacts population-weighted average PM$_{2.5}$ and mortality most, with 250 ng m$^{-3}$ PM$_{2.5}$ reduced and 74 000 (95% CI, 57 000–91 000) annual avoided deaths (Table 3). Avoided deaths are likely underestimated for the residential sector since we exclude changes in indoor exposure. The global
mortality impacts of reducing industrial BC emissions are also large, while those from reducing transportation emissions are fewer. Halving residential, industrial, and transportation emissions contributes 46%, 35%, and 15% to the avoided deaths from halving all anthropogenic BC emissions. These contributions are 1.3, 1.2, and 0.6 times each sector’s portion of global BC emissions, owing to the degree of co-location with population globally. As for BC emissions, the relative magnitude of each sector’s impact differs substantially in developed versus developing regions. For example, in IN, residential, industrial, and transportation emissions contribute 66%, 18%, and 14% of the PM$_{2.5}$ decrease, while in NA these sectors contribute 15%, 19%, and 59%. Of the total avoided deaths from halving global anthropogenic BC emissions, 26% occur in EA from the industrial sector, and 20% occur in each of IN and EA from the residential sector. Impacts of residential BC emissions are underestimates since impacts due to indoor PM$_{2.5}$ exposure are excluded.

The mortality impact per unit BC emitted (“mitigation efficiency”) in each region is similar regardless of the sector from which emissions were reduced (Fig. 8) and follows the same pattern as for the regional reductions (Fig. 7). The only exception is for the transportation sector in EA, for which reducing BC emissions has a smaller per unit impact on mortality relative to the residential and industrial sectors. While the industrial and residential sectors in EA have the greatest BC emissions (“mitigation potential”), all three sectors in IN have the greatest overall mitigation efficiency. Outside of IN and EA, mitigation efficiency is greatest for FSU, SE/AU, and EU, while mitigation potential is greatest for the residential sector in AF/ME and the transportation sector in EU and NA.

4 Sensitivity analysis

For the results presented above, we assume that the CRFs estimated for the US apply globally, although simulated concentrations in some grid cells in Asia are higher than the range included in the ACS study (Table S3). Current evidence does not support
the existence of low or high thresholds beyond which changes in PM$_{2.5}$ concentration have no impact on mortality (Krewski et al., 2009). However, while no long-term epidemiology studies have examined the relationship between PM$_{2.5}$ concentration and mortality at higher concentrations, the marginal impact of PM$_{2.5}$ on mortality may be smaller at higher concentrations (Pope et al., 2009; Smith and Peel, 2010). Without quantitative evidence describing the concentration-response relationship at a wide range of concentrations, we examine the effect of low and high health effect thresholds on estimated deaths.

For the case where BC emissions are halved globally, applying a high-concentration threshold of 50 µg m$^{-3}$ (an assumption consistent with previous studies, e.g. Cohen et al., 2004) reduces global avoided deaths by 56%, 99.6% of which occurs in EA and IN (Fig. 9) where concentrations most frequently exceed the threshold, but the majority (60%) of estimated avoided deaths still occur in these regions. Applying a low-concentration threshold of 5.8 µg m$^{-3}$, the lowest measured level in Krewski et al. (2009), reduces global avoided deaths by 2.1%, 33% and 22% of which occur in AF/ME and SE/AU.

We also examine the effect of applying the significantly higher CRFs from the latest reanalysis of the Harvard Six Cities cohort study (Laden et al., 2006) which found that for a 10 µg m$^{-3}$ increase in PM$_{2.5}$, RRs of cardiovascular and lung cancer mortality were 1.28 (95% CI, 1.13–1.44) and 1.27 (95% CI, 0.96–1.69). Consistent with the relative magnitudes of the RR estimates, using the RRs from Laden et al. (2006) increases estimated global avoided deaths by 45.1%, which is distributed around the world.

5 Uncertainties

While we quantify uncertainty for mortality impacts from statistical error in the CRF, we are unable to quantify several other important uncertainties. Uncertainty in global BC emission inventories is estimated to be about a factor of 2, and could be comparatively larger for the residential and industrial sectors in developing regions, where emissions...
are more difficult to estimate (Bond et al., 2004, 2007). Global anthropogenic BC emissions are also generally allocated to grid cells according to population, an assumption that may be more accurate for some regions and sectors than others. Simulated BC concentrations vary widely among global CTMs due to differing assumptions for emissions and parameterization of aerosol processes, such as aging and wet deposition (Koch et al., 2009; Vignati et al., 2009). These model variations may affect estimated mortality impacts more than uncertainty in the CRF, as has been shown for ozone (Anenberg et al., 2009). The coarse grid resolution of the global CTM also contributes to uncertainty, since it does not capture fine spatial gradients of concentrations, particularly around urban areas. As BC and a component of OM are directly emitted, this uncertainty may be particularly important for this study.

Several uncertainties are also associated with health impact function parameters. We assume that all PM$_{2.5}$ mixtures are equally toxic, despite substantial compositional variation around the world and some evidence suggesting that BC-containing mixtures may be more toxic than the average. Additional research is needed to identify differential toxicity of air pollutant mixtures (e.g. Smith et al., 2009; Dominici et al., 2010; Vedal and Kaufman, 2011). We also assume that CRFs found in the US apply globally, despite differences in concentration levels, populations, lifestyle, age structure, and medical care. This assumption is supported by evidence suggesting that PM$_{2.5}$-mortality associations are similar among different sub-populations in the US (Pope et al., 2009), though different sub-populations around the world likely have larger differences than those in the US, and by similar findings among short-term PM$_{2.5}$ mortality studies around the world (HEI, 2010; Atkinson et al., 2011). We emphasize cause-specific mortality which may be more comparable around the world than all-cause mortality, but may result in underestimates of the total mortality impacts since other causes of death are likely also associated with PM$_{2.5}$.
6 Conclusions

We have estimated the impacts of global, regional, and sectoral BC emission reductions on surface air quality and human mortality using a global chemical transport model (CTM) to simulate PM$_{2.5}$ concentrations and a health impact function to calculate mortality changes. We find that halving global anthropogenic BC emissions would reduce global population-weighted average PM$_{2.5}$ by 542 ng m$^{-3}$ (1.8%) and avoid 157,000 (95% confidence interval, 120,000–194,000) annual premature deaths worldwide, corresponding to 4% of PM$_{2.5}$-related deaths (Anenberg et al., 2010) and 0.3% of all deaths. Since the chemical and physical processes governing BC concentrations in MOZART-4 and our health impact function are approximately linear, these results can be scaled to estimate the surface air quality and health impacts of larger or smaller changes in BC emissions. We exclude the likely substantial morbidity benefits of reducing BC due to lack of data on baseline morbidity rates. Morbidity impacts may not be directly proportional to mortality impacts because of differences in hospitalization rates and medical care around the world.

The vast majority of surface PM$_{2.5}$ and health benefits from BC emission reductions occur within the source region. Some inter-regional transport occurs between FSU, AF/ME, and EU, which are close in proximity. Regional definitions used here are very broad, encompassing many countries, which obscures smaller-scale transport of pollution across political boundaries. While most of the avoided deaths from halving global anthropogenic BC can be achieved by halving EA emissions (54%), followed by IN emissions (31%), IN emissions have 50% greater mortality impacts per unit BC emitted than EA emissions.

Globally, the contribution of residential, industrial, and transportation BC emissions to PM$_{2.5}$-related mortality is 1.3, 1.2, and 0.6 times each sector’s contribution to anthropogenic BC emissions, owing to the degree of co-location between that sector’s emissions and global population. Within each region, mortality per unit emission varies little by source sector; however, impacts of residential BC emissions are underestimated.
since impacts due to indoor PM$_{2.5}$ exposure are excluded. In addition, the coarse grid resolution used here does not capture the spatial scale at which actual exposure to emissions from these sectors occurs (e.g. near roadways for the transportation sector). These results should be further studied at finer resolutions to better simulate actual exposure.

We find that reducing BC emissions increases regional SO$_4$ concentrations up to 28% of regional BC concentration reductions due to reduced absorption of radiation that drives photochemistry. The SO$_4$ increase lessens the health benefits of BC reductions, which are calculated based on total PM$_{2.5}$ concentrations, but may enhance the climate benefit since SO$_4$ scatters radiation. We estimate ~8 times more avoided deaths when BC and OC emissions are halved together, suggesting that these results greatly underestimate the full air pollution-related mortality benefits of BC mitigation, which would affect emissions of multiple species simultaneously. Several studies have examined the net climate impacts of the total emission mixture from individual economic sectors, finding that for some sectors, cooling agents such as SO$_4$ and OM offset the warming impacts of BC (e.g. Koch et al., 2007; Unger et al., 2010). Since no pollutant is beneficial for health, future studies should assess the health impacts of individual economic sectors and mitigation measures, accounting for the full mixture of emissions and both outdoor and indoor exposure. Confidence in our results would also be strengthened by reducing uncertainties from emissions, model parameterization of aerosol processes, grid resolution, and PM$_{2.5}$ concentration-mortality relationships over a range of concentrations, component mixtures, and populations.

Supplementary material related to this article is available online at: http://www.atmos-chem-phys-discuss.net/11/10653/2011/acpd-11-10653-2011-supplement.pdf.

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References


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Park, R. J., Daniel, J. J., Field, B. D., Yantosca, R. M., and Chin, M.: Natural and transboundary...
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ACPD 11, 10653–10691, 2011

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### Table 1.
Simple and population-weighted annual average PM$_{2.5}$ (ng m$^{-3}$) concentrations for the base case and PM$_{2.5}$ reduction, avoided cardiopulmonary and lung cancer deaths (in thousands), and percent of these deaths from cardiopulmonary (CP) disease due to halving global anthropogenic BC emissions and BC + OC emissions. Confidence intervals (95%, in parentheses) reflect uncertainty in the CRF only.

<table>
<thead>
<tr>
<th>Receptor region</th>
<th>Base Case PM$_{2.5}$ Concentration (µg m$^{-3}$)</th>
<th>Global 50% BC Reduction</th>
<th>Global 50% BC + OC Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Simple Average</td>
<td>Population-weighted Average</td>
<td>Simple Average $\Delta$PM$_{2.5}$ (ng m$^{-3}$)</td>
</tr>
<tr>
<td>North America (NA)</td>
<td>3.54</td>
<td>8.28</td>
<td>43</td>
</tr>
<tr>
<td>South America (SA)</td>
<td>3.84</td>
<td>6.02</td>
<td>44</td>
</tr>
<tr>
<td>Europe (EU)</td>
<td>9.77</td>
<td>13.4</td>
<td>147</td>
</tr>
<tr>
<td>Former Soviet Union (FSU)</td>
<td>5.87</td>
<td>12.29</td>
<td>60</td>
</tr>
<tr>
<td>Africa/Middle East (AF/ME)</td>
<td>5.32</td>
<td>7.21</td>
<td>64</td>
</tr>
<tr>
<td>South Asia (India; IN)</td>
<td>19.28</td>
<td>36.29</td>
<td>420</td>
</tr>
<tr>
<td>East Asia (China; EA)</td>
<td>27.98</td>
<td>70.17</td>
<td>453</td>
</tr>
<tr>
<td>Southeast Asia/ Australia (SE/AU)</td>
<td>4.81</td>
<td>9.55</td>
<td>54</td>
</tr>
<tr>
<td>World</td>
<td>2.75</td>
<td>29.7</td>
<td>33</td>
</tr>
</tbody>
</table>

**Note:** The table data reflects reductions in PM$_{2.5}$ and BC + OC emissions, leading to avoided deaths from cardiopulmonary and lung cancer diseases.
Table 2. Reduction in population-weighted average PM$_{2.5}$ concentration (first row for each receptor region, ng m$^{-3}$) and annual avoided premature cardiopulmonary and lung cancer deaths (second row, thousands) in each receptor region after halving anthropogenic BC emissions in each source region. Confidence intervals (95%, in parentheses) reflect uncertainty in the CRF only.

<table>
<thead>
<tr>
<th>Source Region</th>
<th>Receptor Region</th>
<th>NA</th>
<th>SA</th>
<th>EU</th>
<th>FSU</th>
<th>AF/ME</th>
<th>IN</th>
<th>EA</th>
<th>SE/AU</th>
</tr>
</thead>
<tbody>
<tr>
<td>NA</td>
<td>4 (3–5)</td>
<td>151</td>
<td>−1</td>
<td>−1</td>
<td>−1</td>
<td>−1</td>
<td>−1</td>
<td>−2</td>
<td>−1</td>
</tr>
<tr>
<td></td>
<td>0 (1–2)</td>
<td>0</td>
<td>129</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>225</td>
<td>5</td>
<td>9</td>
<td>−1</td>
<td>−3</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>8 (6–10)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>−1</td>
<td>0</td>
<td>39</td>
<td>135</td>
<td>23</td>
<td>−1</td>
<td>1</td>
<td>0</td>
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</tr>
<tr>
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<td>0</td>
<td>0</td>
<td>1 (1–5)</td>
<td>4 (3–5)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>FSU</td>
<td>−1</td>
<td>0</td>
<td>7</td>
<td>5</td>
<td>116</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4 (3–5)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>AF/ME</td>
<td>−1</td>
<td>−1</td>
<td>−1</td>
<td>−1</td>
<td>0</td>
<td>733</td>
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<td>49 (37–59)</td>
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<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>IN</td>
<td>−4</td>
<td>0</td>
<td>−9</td>
<td>−3</td>
<td>−6</td>
<td>−20</td>
<td>1262</td>
<td>−9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>−1 (−1–−1)</td>
<td>−1 (0–−1)</td>
<td>−2 (−1–−2)</td>
<td>85 (64–105)</td>
<td>−1 (−1–−1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EA</td>
<td>−4</td>
<td>0</td>
<td>−9</td>
<td>−3</td>
<td>−6</td>
<td>−20</td>
<td>1262</td>
<td>−9</td>
<td></td>
</tr>
<tr>
<td></td>
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<td>0</td>
<td>−1 (−1–−1)</td>
<td>−1 (0–−1)</td>
<td>−2 (−1–−2)</td>
<td>85 (64–105)</td>
<td>−1 (−1–−1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SE/AU</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5 (4–7)</td>
<td></td>
</tr>
</tbody>
</table>
Table 3. Population-weighted reduction in annual average PM$_{2.5}$ (ng m$^{-3}$) and annual avoided premature cardiopulmonary and lung cancer deaths (in thousands) for halving global BC emissions from each sector, relative to the base case. Confidence intervals (95%, in parentheses) reflect uncertainty in the CRF only.

<table>
<thead>
<tr>
<th>Receptor region</th>
<th>Residential</th>
<th></th>
<th>Industrial</th>
<th></th>
<th>Transportation</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Population-weighted $\Delta PM_{2.5}$ (ng m$^{-3}$)</td>
<td>Avoided deaths $\times$1000</td>
<td>Population-weighted $\Delta PM_{2.5}$ (ng m$^{-3}$)</td>
<td>Avoided deaths $\times$1000</td>
<td>Population-weighted $\Delta PM_{2.5}$ (ng m$^{-3}$)</td>
<td>Avoided deaths $\times$1000</td>
</tr>
<tr>
<td>NA</td>
<td>22</td>
<td>1 (0–1)</td>
<td>29</td>
<td>1 (1–1)</td>
<td>88</td>
<td>2 (2–3)</td>
</tr>
<tr>
<td>SA</td>
<td>28</td>
<td>0 (0–0)</td>
<td>30</td>
<td>0 (0–0)</td>
<td>65</td>
<td>1 (0–1)</td>
</tr>
<tr>
<td>EU</td>
<td>50</td>
<td>2 (1–2)</td>
<td>42</td>
<td>1 (1–2)</td>
<td>124</td>
<td>4 (3–5)</td>
</tr>
<tr>
<td>FSU</td>
<td>99</td>
<td>3 (2–3)</td>
<td>32</td>
<td>1 (1–1)</td>
<td>33</td>
<td>1 (1–1)</td>
</tr>
<tr>
<td>AF/ME</td>
<td>65</td>
<td>2 (2–3)</td>
<td>21</td>
<td>1 (1–1)</td>
<td>31</td>
<td>1 (1–1)</td>
</tr>
<tr>
<td>IN</td>
<td>480</td>
<td>32 (25–39)</td>
<td>135</td>
<td>9 (7–11)</td>
<td>105</td>
<td>7 (5–9)</td>
</tr>
<tr>
<td>SE/AU</td>
<td>115</td>
<td>3 (2–3)</td>
<td>50</td>
<td>1 (1–1)</td>
<td>71</td>
<td>2 (1–2)</td>
</tr>
<tr>
<td>World</td>
<td>250</td>
<td>74 (57–91)</td>
<td>191</td>
<td>55 (42–68)</td>
<td>80</td>
<td>23 (17–28)</td>
</tr>
</tbody>
</table>
Fig. 1. Anthropogenic BC emissions by region and sector after the IPCC emissions are scaled by 1.15 to account for particles larger than 1 µm in diameter.
Fig. 2. Simulated annual average concentration ($\mu g m^{-3}$) of PM$_{2.5}$ components for the base case (2002) by geographic region.
Fig. 3. Comparison of simulated annual average surface BC and OC concentrations (µg m$^{-3}$) with the IMPROVE surface monitoring network for remote locations in the United States (average of 2002 and 2003) for (a) BC and (b) OC (includes SOA), and with the EMEP surface monitoring network for Europe (average for July 2002 to June 2003) for (c) BC and (d) OC (includes SOA).
Fig. 4. Reduction in annual average surface PM$_{2.5}$ concentration (ng m$^{-3}$) for the global, sectoral, and regional emission reductions relative to the base case.
Fig. 5. Reduction in global and regional annual average concentrations (ng m$^{-3}$) of PM$_{2.5}$ species for halving global anthropogenic (a) BC emissions and (b) BC + OC emissions, relative to the base case.
Fig. 6. Avoided annual cardiopulmonary and lung cancer deaths per 1000 km$^2$, for the global, sectoral, and regional emission reductions relative to the base case.
Fig. 7. Global annual avoided premature cardiopulmonary and lung cancer deaths (thousands; blue bars) and avoided premature deaths per Gg BC emissions reduced (red diamonds), for halving anthropogenic BC emissions in each source region relative to the base case. Confidence intervals (95%) reflect uncertainty in the CRF only.
Fig. 8. Annual avoided premature cardiopulmonary and lung cancer deaths per unit BC emissions reduced vs. total BC emissions (Gg) for particular source sectors within each region. Avoided deaths are estimated in the three simulations where global emissions in each sector are halved, and shown for each receptor region; these deaths are compared with emissions from each region, assuming that deaths from inter-regional transport are negligible (Table 2). Uncertainty in the mortality estimates is a factor of 0.23 from the central estimate, which includes uncertainties in the cardiopulmonary and lung cancer CRFs only. Uncertainty in BC emissions is assumed to be a factor of 2 from the central estimate (Bond et al., 2004, 2007). Since these uncertainties are factor differences from the central estimate, they are identical for each data point.
Fig. 9. Sensitivity of estimated avoided annual premature cardiopulmonary and lung cancer deaths from halving global anthropogenic BC emissions to a high concentration threshold (HCT) of 50 µg m\(^{-3}\), a low-concentration threshold (LCT) of 30 µg m\(^{-3}\), and relative risk estimates from Laden et al. (2006; cardiovascular and lung cancer mortality only). Confidence intervals (95%) reflect uncertainty in the CRF only.