

Impacts of intercontinental transport of anthropogenic fine particulate matter on human mortality

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Abstract Fine particulate matter with diameter of 2.5 μm or less ($\text{PM}_{2.5}$) is associated with premature mortality and can travel long distances, impacting air quality and health on intercontinental scales. We estimate the mortality impacts of 20 % anthropogenic primary $\text{PM}_{2.5}$ and $\text{PM}_{2.5}$ precursor emission reductions in each of four major industrial regions (North America, Europe, East Asia, and South Asia) using an

ensemble of global chemical transport model simulations coordinated by the Task Force on Hemispheric Transport of Air Pollution and epidemiologically-derived concentration-response functions. We estimate that while 93–97 % of avoided deaths from reducing emissions in all four regions occur within the source region, 3–7 % (11,500; 95 % confidence interval, 8,800–14,200) occur outside the source region

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from concentrations transported between continents. Approximately 17 and 13 % of global deaths avoided by reducing North America and Europe emissions occur extraregionally, owing to large downwind populations, compared with 4 and 2 % for South and East Asia. The coarse resolution global models used here may underestimate intraregional health benefits occurring on local scales, affecting these relative contributions of extraregional versus intraregional health benefits. Compared with a previous study of 20 % ozone precursor emission reductions, we find that despite greater transport efficiency for ozone, absolute mortality impacts of intercontinental PM_{2.5} transport are comparable or greater for neighboring source-receptor pairs, due to the stronger effect of PM_{2.5} on mortality. However, uncertainties in modeling and concentration-response relationships are large for both estimates.

Keywords Health impact assessment · Particulate matter · Long-range transport · Chemical transport modeling

Introduction

Fine particulate matter, particles with diameter of 2.5 µm or less (PM_{2.5}), is associated with deleterious health effects, including premature mortality due to cardiopulmonary disease and lung cancer (Krewski et al. 2009). Despite its relatively short atmosphere lifetime (days to weeks), both PM_{2.5} and its precursors can travel long distances, affecting air quality and health far from the emission source (e.g., Langner et al. 1992; Park et al. 2003; Park et al. 2004; Heald et al. 2006; Chin et al. 2007; Hadley et al. 2007; Liu et al. 2009a; Liu et al. 2009b; TF HTAP 2010; Yu et al. 2008; Ewing et al. 2013). Although PM_{2.5} is transported most efficiently at altitude in the free troposphere, PM_{2.5} originating from distant sources can influence surface PM_{2.5} concentrations where people are exposed (Park et al. 2004; Chin et al. 2007; Liu et al. 2009a). In addition to dust, which is the dominant contributor to aerosol transport globally (Chin et al. 2007; Liu et al. 2009a; Yu et al. 2012), anthropogenic emission sources can affect PM_{2.5} air quality on intercontinental scales through emissions of primary PM_{2.5} (black carbon (BC) and primary organic aerosol); precursors of secondary PM_{2.5} components including sulfate (SO₄), nitrate (NO₃), and secondary organic aerosol; and changes to oxidants that influence the formation of secondary PM_{2.5} (Pham et al. 1995; Leibensperger et al. 2011; Fry et al. 2012). Because secondary components may be formed downwind, they typically affect air quality on larger spatial scales than primary emissions (Heald et al. 2006; Liu et al. 2009a; Leibensperger et al. 2011).

Compared with aerosols, intercontinental transport of ozone has generally received more attention in both science and policy arenas, since ozone has a longer atmospheric

lifetime (about a month) and is transported in the atmosphere more efficiently (TF HTAP 2010). However, PM_{2.5} has a stronger effect on mortality (e.g., Bell et al. 2004; Jerrett et al. 2009; Krewski et al. 2009) and is the dominant contributor to premature mortality from outdoor air pollution (Anenberg et al. 2010; Lim et al. 2013). Previous studies find that ozone precursor emissions affect health globally, with 20 to >50 % of regional ozone-related deaths caused by extraregional emissions (Anenberg et al. 2009; West et al. 2009). North American and European emissions are estimated to have greater health impacts outside the source region than within, mainly due to large exposed populations in East and South Asia (Duncan et al. 2008; Anenberg et al. 2009; West et al. 2009). One previous study addressed the health impacts of intercontinental PM_{2.5} transport, using a tagging approach to estimate that intercontinental transport of nondust aerosols is associated with nearly 90,000 annual premature deaths globally, approximately 60 % of which occur in the densely populated East Asia, India, and Southeast Asia (Liu et al. 2009b).

Here, we calculate the impacts of intercontinental transport of anthropogenic PM_{2.5} on surface air quality and human mortality using an ensemble of global chemical transport models coordinated by the Task Force on Hemispheric Transport of Air Pollution (TF HTAP 2010). We use multimodel simulations of 20 % anthropogenic primary PM_{2.5} and PM_{2.5} precursor emission reductions in each of four major industrial regions to calculate their impact on premature mortality within the region and elsewhere in the world. Compared with estimates made using a single atmospheric model, using a multimodel ensemble allows a more robust estimate and characterization of uncertainty due to intermodel differences (e.g., Fiore et al. 2009). As ambient air quality standards continue to tighten and controlling local emissions becomes increasingly expensive in some countries, improved understanding of foreign emission contributions to PM_{2.5} concentrations and mortality may help inform future mitigation strategies (Keating et al. 2004).

Methods

We use TF HTAP multimodel ensemble estimates of the impact of 20 % regional emission reductions on PM_{2.5} concentrations around the world. The TF HTAP was established in 2004 by the Convention on Long-Range Transboundary Air Pollution (CLRTAP) to improve understanding of the intercontinental transport of air pollutants across the Northern Hemisphere for consideration by the CLRTAP. Over the last decade, the TF HTAP has organized a series of multimodel experiments to advance the state of the science related to the transport of pollutants, including ozone and PM_{2.5}, among others. The first set of multimodel experiments

concluded in 2010 and was reported on extensively by HTAP (2010) and in the peer-reviewed literature (e.g., Fiore et al. 2009). Here, we describe the subset of experiments and models used for the present study. Additional information on the methods can be found in the Supplemental Material.

The TF HTAP model ensemble simulated a base 2001 simulation and 20 % emission reductions of anthropogenic primary $PM_{2.5}$ (BC and primary organic aerosol) and gases that influence secondary $PM_{2.5}$ (SO_2 , ammonia, NO_x , nonmethane volatile organic compounds, and carbon monoxide), individually in each of four major world regions (North America (NA), East Asia (EA), South Asia (SA), and Europe (EU)). NA, EA, SA, and EU, shown in Supplementary Fig. S1, have areas of 2.6×10^7 , 2.3×10^7 , 1.5×10^7 , and 2.1×10^7 km², respectively. The TF HTAP base and 20 % regional anthropogenic emission reduction scenarios were termed SR1 and SR6 scenarios, respectively. The models included in the ensemble used different resolutions, meteorology, physical and chemical parameters, and emission inputs, but all models reduced anthropogenic emissions by 20 % over the same geographical regions. Emission differences across the models that participated in the TF HTAP experiments were described by Fiore et al. (2009) and Yu et al. (2013). Model simulations were conducted for a full year following 6 months or longer of initialization, allowing time for simulated trace gas and particle concentrations to respond fully to the emission reductions. This study complements previous analyses using the TF HTAP model ensemble to estimate intercontinental ozone transport (Fiore et al. 2009) and resulting health (Anenberg et al. 2009) and climate impacts (Fry et al. 2012), climate impacts of $PM_{2.5}$ transport (Yu et al. 2013), and the transport of pollutants to the Arctic (Shindell et al. 2008).

We use results from the 12 models that simulated changes in BC, particulate organic matter (POM=primary organic aerosol+secondary organic aerosol), and SO_4^{2-} (SO_4 , see Supplementary Table S1), excluding three models that performed the model experiments but did not simulate all three species. We excluded NO_3^- (NO_3) which was only simulated by five out of 12 models. Anthropogenic secondary organic aerosol is included in POM by some of the models but is not diagnosed separately. For each species, we regrid concentrations in the first vertical level of each model to a common $0.5^\circ \times 0.5^\circ$ resolution and calculate the median and standard deviations of gridded concentrations across the models, summing the ensemble median SO_4 (assumed to be ammonium sulfate, $(NH_4)_2SO_4$), BC, and POM for total $PM_{2.5}$. We use multimodel median concentrations rather than the mean to avoid potential bias from outliers. Our limited $PM_{2.5}$ definition understates total anthropogenic $PM_{2.5}$ concentrations in the atmosphere and also likely understates the impacts of intercontinental transport of anthropogenic $PM_{2.5}$. Multimodel median base case concentrations for total $PM_{2.5}$, SO_4 , BC, and POM can be found in Table 1.

Table 1 For each receptor region, area and population-weighted average $PM_{2.5}$ annual average concentration ($\mu g/m^3$) for the base case (median across all models in the ensemble), and percent contributions of each $PM_{2.5}$ component

	Receptor region				
	NA	EA	SA	EU	World
Area average					
$PM_{2.5}$ ($\mu g/m^3$)	3.22	5.91	5.11	5.77	1.48
SO_4 (%)	70.4	68.0	57.9	81.0	64.7
BC (%)	4.7	8.1	7.11	4.8	5.1
POM (%)	24.9	23.9	35.0	14.2	30.2
Population-weighted average					
$PM_{2.5}$ ($\mu g/m^3$)	6.04	16.9	10.3	7.45	9.41
SO_4 (%)	64.9	64.4	43.5	78.1	56.2
BC (%)	6.7	10.3	9.8	6.5	9.3
POM (%)	28.3	25.3	46.8	15.4	34.5

NA North America, EA East Asia, SA South Asia, EU Europe, $PM_{2.5}$ particulate matter (with diameter of 2.5 μm or less), SO_4 sulfate, BC black carbon, POM particulate organic matter

Comparisons of simulated surface aerosols with monitor and satellite measurements have been performed previously for many of these models individually (e.g., Takemura et al. 2000, 2002; Koch et al. 2005, 2007; Ginoux et al. 2006; Park et al. 2006; Chin et al. 2007, 2013; Brauer et al. 2012; Lamarque et al. 2012; Jeong and Park 2013) or as part of model ensemble experiments (e.g., Koch et al. 2009; Koffi et al. 2012), including one from the TF HTAP ensemble (Shindell et al. 2008). These evaluations suggest that the models perform reasonably well for surface $PM_{2.5}$ concentrations, although the coarse model resolutions used here are not expected to represent urban concentrations where most monitors are located. Despite their coarse resolutions, the use of global chemical transport models is appropriate for this study because we focus on $PM_{2.5}$ concentrations transported across long distances, which are more homogenous than local concentrations near emission sources.

Following Anenberg et al. (2010), annual avoided premature deaths from the 20 % regional emission reductions are calculated using a health impact function based on epidemiological relationships between ambient $PM_{2.5}$ concentration and mortality (Eq. 1), where Pop is the population, Y_0 is the baseline mortality rate, β is the concentration-response factor from the epidemiology literature, and ΔX is the change in annual average $PM_{2.5}$ (base minus 20 % regional emission reduction, or SR1–SR6).

$$\Delta \text{Mort} = \text{Pop} (1 - \exp^{-\beta \Delta X}) Y_0 \quad (1)$$

To calculate β (Eq. 2), we use relative risk (RR) estimates from the latest reanalysis of the American Cancer Society

study (Krewski et al. 2009), as it includes the most broadly representative population of the set of PM_{2.5} cohort studies (e.g., the Harvard Six Cities Study, Lepeule et al. 2012; Beelen et al. 2013), and apply these globally since there are few PM_{2.5} cohort studies elsewhere in the world.

$$\beta = \ln(RR)/\Delta X \quad (2)$$

For a 10- $\mu\text{g}/\text{m}^3$ increase in annual average PM_{2.5}, RR was 1.06 (95 % confidence interval, 1.04–1.08), 1.13 (1.10–1.16), and 1.14 (1.06–1.23) for all-cause, cardiopulmonary, and lung cancer mortality for the period 1999–2000. This relationship was approximately linear within the range of observed annual mean concentrations (5.8–22 $\mu\text{g}/\text{m}^3$). Linearity was also demonstrated up to 30 $\mu\text{g}/\text{m}^3$ for the period 1979–1983.

While the concentration-response factors were found to be approximately linear at the total PM_{2.5} concentration levels observed in the USA (Krewski et al. 2009), studies suggest that PM_{2.5} concentration-response factors may flatten out at very high concentrations (e.g., Pope et al. 2009, 2011). Because we include only SO₄, BC, and POM in our PM_{2.5} definition, the global population-weighted average PM_{2.5} concentration and the maximum multimodel median annual average PM_{2.5} concentrations across all grid cells are 9.4 and 30 $\mu\text{g}/\text{m}^3$, respectively (Tables 1 and 2 and Supplementary Table S2), significantly lower than the total PM_{2.5} concentrations observed in many places around the world (Brauer et al. 2012). Including dust, NO₃, and other PM_{2.5} components in the PM_{2.5} definition would likely raise concentrations in some grid cells, particularly in polluted areas of Asia, to substantially higher levels, as shown by output from seven of the models that simulated total PM_{2.5} (including mineral dust and biogenic aerosols; Table 2 and Supplementary Fig. S2) explicitly. While we account appropriately for the impact of intercontinental PM_{2.5} transport on changes in the species included in our PM_{2.5} definition, we may overestimate health impacts in areas where total PM_{2.5} concentrations are high

(due to anthropogenic and/or natural emissions) and are thus on the flatter portion of the concentration-response factor (Anenberg et al. 2012).

Extrapolation of these RR estimates from the USA to populations elsewhere in the world is supported by generally consistent short-term PM_{2.5} mortality relationships globally (HEI 2010) but introduces uncertainty since exposure and population characteristics vary around the world. We calculate cause-specific mortality, which may be more comparable globally than all-cause mortality. We use country-specific baseline mortality rates (WHO 2008), 2006 population (ORNL 2008), and, consistent with the American Cancer Society cohort study, only the fraction of the population aged 30 and older (WHO 2004). These quantities are also regridded to the 0.5°×0.5° resolution, and mortality changes are estimated by applying Eq. 1 in each grid cell. The population aged 30 and older and baseline mortality rates for each region can be found in Table 2.

Results

Impacts on surface PM_{2.5} concentrations

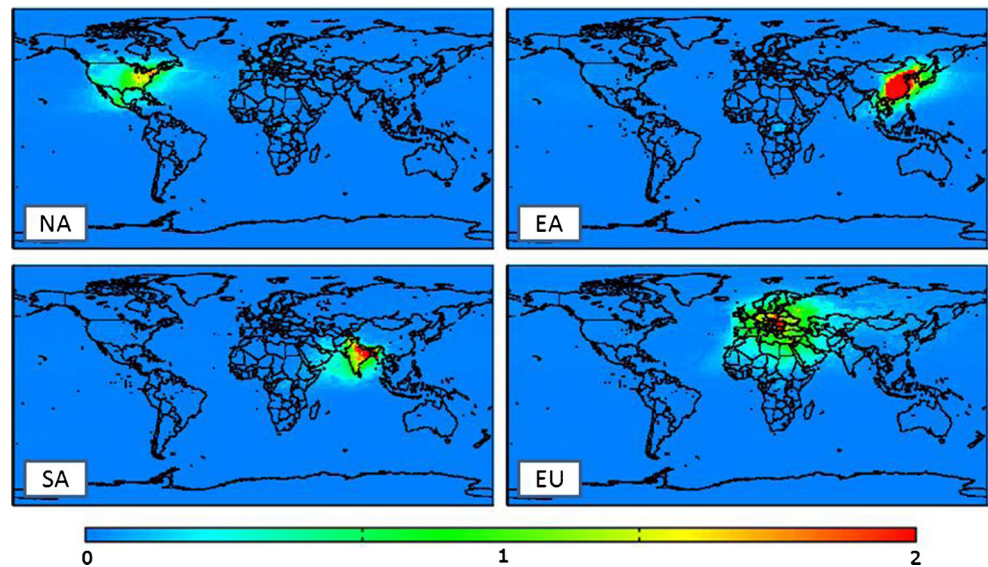
In general, regional PM_{2.5} concentrations are dominated by emissions from within the same region (Fig. 1 and Table 3). For each receptor region, the 20 % emission reductions in the three foreign regions combined reduce population-weighted PM_{2.5} by 3–7 % (0.03, 0.08, 0.12, and 0.05 $\mu\text{g}/\text{m}^3$ in NA, EA, SA, and EU, respectively). SO₄ reductions contribute most to reduced total PM_{2.5} concentrations in all regions except SA where POM contributes more. SO₄ also contributes relatively more for foreign receptor regions (54–100 %) than for within the source region (40–77 %) since SO₄ can be formed in the atmosphere far from the precursor emission source (Table 3 and Supplementary Figs. S3–S5). BC and most of the anthropogenic POM are directly emitted and therefore contribute

Table 2 For each receptor region, present-day population aged 30 and older (2006), baseline cardiopulmonary and lung cancer mortality rates for the population aged 30 and older (latest year available 2000–2008),

Region	Population 30+ (millions)	Baseline mortality rate for population 30+ (% per year)		Maximum of the gridded multimodel median PM _{2.5} concentration ($\mu\text{g}/\text{m}^3$)	
		Cardiopulmonary	Lung cancer	SO ₄ + POM+BC	Modeled explicitly
NA	0.26	0.55	0.07	11.1	16.1
EA	0.84	0.63	0.05	30.2	62.3
SA	0.59	0.88	0.02	19.5	73.0
EU	0.53	0.84	0.05	15.4	183.4
World	2.91	0.74	0.04	30.2	239.5

NA North America, EA East Asia, SA South Asia, EU Europe, SO₄ sulfate, BC black carbon, POM particulate organic matter

Fig. 1 Reduction in multimodel median annual average $PM_{2.5}$ concentration (microgram per cubic meter) for 20 % regional emission reduction scenarios (for region shown in *bottom left corner*) relative to the base case. Regions are North America (NA), East Asia (EA), South Asia (SA), and Europe (EU)



more to total $PM_{2.5}$ concentrations within the source region compared with the foreign regions. Although global models typically inject emissions at the same altitude for all anthropogenic sectors, SO_4 may also have more widespread impacts in reality, because SO_2 -rich power plant emissions are

Table 3 For 20 % primary $PM_{2.5}$ and $PM_{2.5}$ precursor emission reductions in each source region, reduction in population-weighted annual average surface $PM_{2.5}$ concentration (micrograms per cubic meter) and percentage contribution of each $PM_{2.5}$ component to the total population-weighted surface $PM_{2.5}$ reduction in each receptor region

Source region	$PM_{2.5}$ component	Receptor region				
		NA	EA	SA	EU	World
NA	$PM_{2.5}$ ($\mu g/m^3$)	0.87	0.01	0.01	0.04	0.08
	SO_4 (%)	69.9	80.5	100	95.8	73.2
	BC (%)	7.9	6.8	0	1.2	7.0
	POM (%)	22.2	12.8	0	3.1	19.8
EA	$PM_{2.5}$ ($\mu g/m^3$)	0.02	2.66	0.03	0.01	0.70
	SO_4 (%)	90.6	61.6	82.7	92.5	62.1
	BC (%)	2.9	12.0	3.2	3.2	11.8
	POM (%)	6.5	26.5	14.1	4.4	26.1
SA	$PM_{2.5}$ ($\mu g/m^3$)	0.00	0.03	1.59	0.01	0.39
	SO_4 (%)	88.4	53.5	40.1	86.7	40.9
	BC (%)	0.4	8.6	11.0	6.3	10.8
	POM (%)	11.3	37.8	48.9	7.0	48.2
EU	$PM_{2.5}$ ($\mu g/m^3$)	0.01	0.04	0.08	1.09	0.22
	SO_4 (%)	98.1	91.1	94.6	77.1	80.4
	BC (%)	0.4	2.8	1.5	8.2	6.9
	POM (%)	1.6	6.1	3.9	14.8	12.7

Area averages are shown in Table S2

NA North America, EA East Asia, SA South Asia, EU Europe, $PM_{2.5}$ particulate matter (with diameter of 2.5 μm or less), SO_4 sulfate, BC black carbon, POM particulate organic matter

released at higher altitudes than BC- and POM-rich emissions from other sectors (e.g., transportation, residential cooking, and heating).

For the impact of each source region on foreign receptor regions, emissions from EU impact concentrations in the Northern Hemisphere most broadly, owing to its relatively close proximity to two downwind regions. Reducing EU emissions by 20 % decreases population-weighted $PM_{2.5}$ most in SA ($0.08 \mu g/m^3$) followed by EA (0.04 ; Table 3). Reducing NA emissions impacts population-weighted $PM_{2.5}$ most in EU ($0.04 \mu g/m^3$), following the prevailing winds flowing west to east. Reducing EA emissions influences $PM_{2.5}$ most in nearby SA ($0.03 \mu g/m^3$) and downwind NA (0.02). SA impacts other regions least due to physical and meteorological conditions limiting transport from that region, with the greatest impact in nearby EA ($0.03 \mu g/m^3$).

Impacts on $PM_{2.5}$ -related mortality

The 20 % emission reductions in all four regions impact within-region mortality most (93–97 % of avoided deaths occur within the source region), but combined avoid 11,500 (95 % confidence interval, 8,800–14,200) annual premature deaths outside the source region (Table 4 and Fig. 2). SA and EA are impacted most in terms of absolute numbers of premature deaths avoided annually by 20 % emission reductions in other regions (2,900 each), followed by EU (1,700) and NA (400). SA is impacted most in terms of percentage of the total mortality impact attributable to extraregional emissions (6.8 %), followed by EU (4.4 %), NA (4.0 %), and EA (3.0 %).

While reducing EA emissions avoids the most premature deaths overall due to the large population density and emissions in that region, reducing EU emissions by 20 % avoids

Table 4 Annual avoided premature cardiopulmonary and lung cancer deaths due to 20 % primary PM_{2.5} and PM_{2.5} precursor emissions reductions in each region

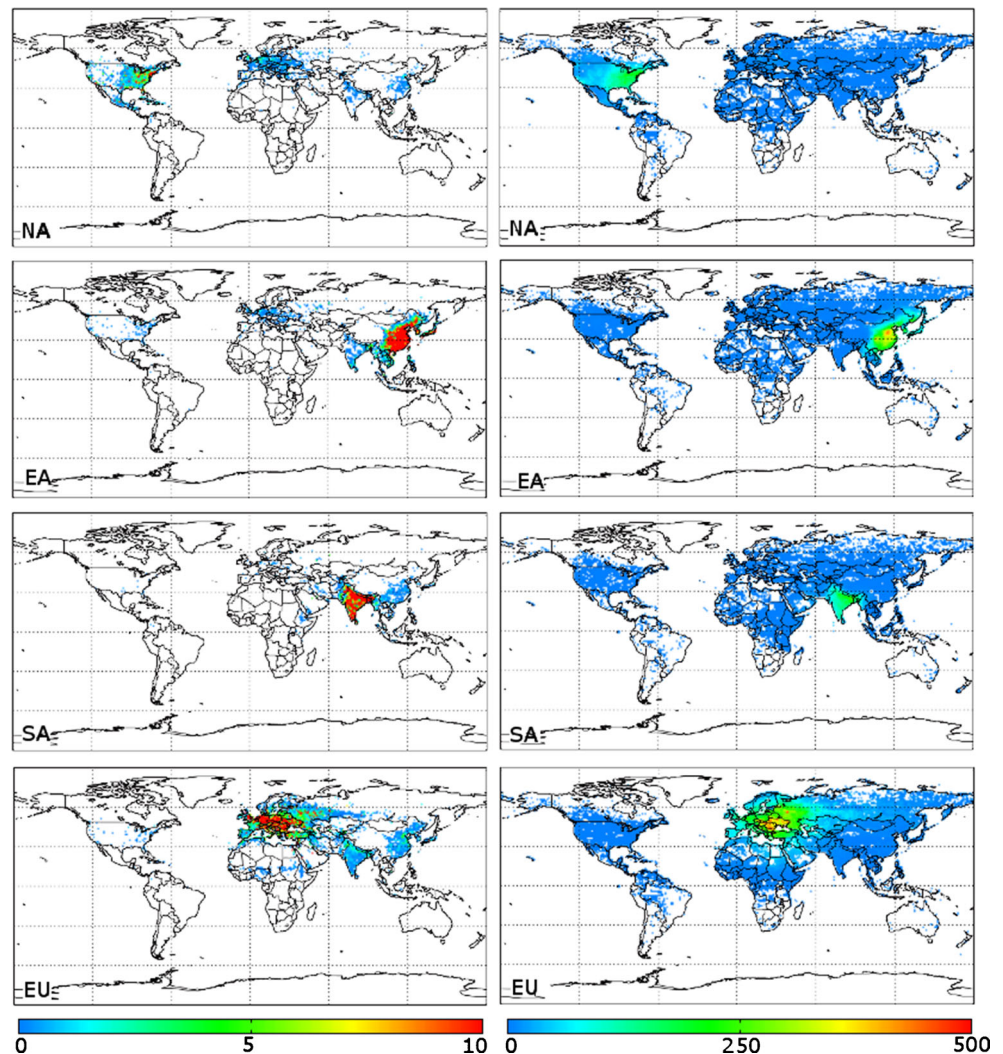
Source region	Receptor region				
	NA	EA	SA	EU	World
NA	9,900 (7,300–12,500)	400 (300–500)	200 (200–200)	1,200 (900–1,500)	11,900 (8,800–15,000)
EA	200 (200–300)	93,400 (70,900–115,300)	900 (700–1100)	400 (300–500)	95,600 (72,600–118,000)
SA	0 (0–0)	900 (700–1,200)	40,000 (30,800–48,900)	100 (100–100)	41,500 (32,000–50,700)
EU	200 (100–200)	1,600 (1,400–1,900)	1,900 (1,400–2,300)	37,400 (28,300–46,300)	43,200 (32,700–46,300)

Confidence intervals (95 %) reflect uncertainty in the concentration-response factor only

NA North America, EA East Asia, SA South Asia, EU Europe

more premature deaths outside of the source region than for any other region (5,700; 95 % confidence interval, 4,400–

7,100). Of the global avoided deaths from the emission reductions in EA and SA, only 2 and 4 % occur outside of the

Fig. 2 Annual premature cardiopulmonary and lung cancer deaths per 1,000 km² (*left*) and per million people (*right*) due to 20 % primary PM_{2.5} and PM_{2.5} precursor emission reductions in the region shown

source regions. However, 17 and 13 % of global avoided deaths from NA and EU emission reductions occur outside the source region, due mainly to smaller intraregional and larger extraregional populations.

Sensitivity analysis

We examine the sensitivity of results to the range of $PM_{2.5}$ responses simulated by the model ensemble for each grid cell (see Figs. 3–4 and Supplementary Figs. S6–S7 for individual model results) and to varying assumptions for the concentration-response factor and $PM_{2.5}$ definition (Fig. 5, confidence intervals shown by the vertical lines). We find that the range of simulated $PM_{2.5}$ concentration changes across the models contributes more to uncertainty in estimated avoided deaths than it does to uncertainty associated with the concentration-response factor from Krewski et al. (2009). While relative risk estimates from the American Cancer Society study are widely accepted and used for health impact assessment, they may be conservative (Roman et al. 2008). Using the substantially higher concentration-response factor for cardiovascular disease and lung cancer from the Harvard Six Cities cohort study (Lepeule et al. 2012) yields mortality impacts that are 1.3–1.7 times the main results. However, uncertainties in the concentration-response factor are greater than those implied by examining only the confidence interval from one $PM_{2.5}$ mortality cohort study. Using changes in total $PM_{2.5}$ modeled explicitly by seven of the 12 models (includes dust, sea salt, nitrate in three of the models, secondary organic aerosol in one model, and other $PM_{2.5}$ components) yields mortality impacts that are 1–1.3 times the main results (includes only BC, POM, and SO_4). Since dust and sea salt are assumed to be natural and thus unchanged between the base case and emission reduction scenario, the effect of using total

$PM_{2.5}$ modeled explicitly is likely due to the inclusion of nitrate by three of the models and secondary organic aerosol by one of the models. We do not examine the use of a log concentration-response curve that flattens at high concentrations because the steeper segment of the curve at the relatively low concentrations simulated by the multimodel ensemble may inappropriately inflate mortality impacts (see the sensitivity analysis by Anenberg et al. 2012).

Comparison with previous studies

Since concentrations far from the emission source tend to respond approximately linearly to emission reductions (TF HTAP 2010), these $PM_{2.5}$ concentration reductions can be extrapolated linearly to compare our results with a previous estimate of the transport of anthropogenic $PM_{2.5}$ (Liu et al. 2009b). While Liu et al. (2009b) estimated about 90,000 premature deaths due to intercontinental transport of anthropogenic aerosols, here, we estimate 58,000 (95 % confidence interval, 44,000–71,000; results for 20 % emission reductions multiplied by 5). The discrepancy may be due to methodological differences, including our use of reducing emissions in a multimodel ensemble versus the tagging method in a single model; our consideration of only the four HTAP regions (however, these contribute the majority of global anthropogenic emissions); and different baseline mortality rates, population, and concentration-response functions.

We also compare our results with a study examining ozone-related mortality impacts based on the same HTAP 20 % emission reduction scenarios (Anenberg et al. 2009). While $PM_{2.5}$ has a shorter atmospheric lifetime and is transported less efficiently than ozone, it has a stronger effect on mortality (Bell et al. 2004; Jerrett et al. 2009; Krewski et al. 2009). We find that in response to 20 % regional emission reductions,

Fig. 3 Population-weighted average reduction in annual average $PM_{2.5}$ concentration due to 20 % anthropogenic emission reductions in the same region, as simulated by each of the 12 models

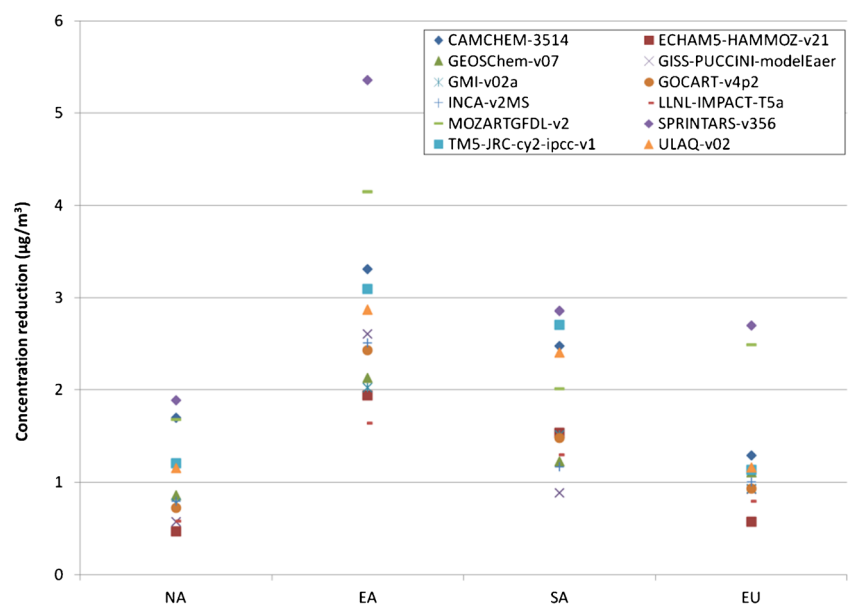
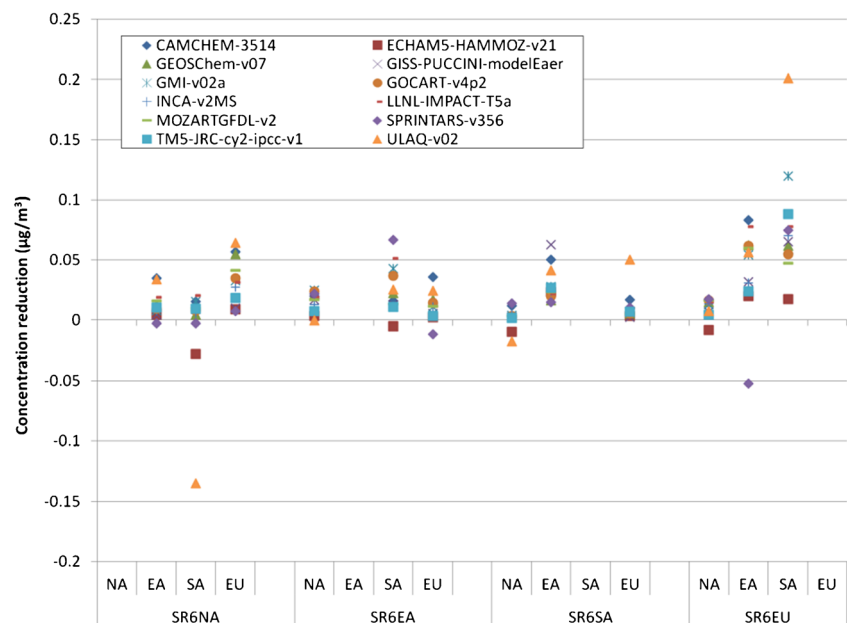


Fig. 4 Population-weighted average reduction in annual average $PM_{2.5}$ concentration in each foreign receptor region due to 20 % anthropogenic emission reductions in each source region (indicated by SR6+the region in which emissions were reduced, e.g., SR6NA for North American emission reductions), as simulated by each of the 12 models. Impacts of emission reductions in the same region are removed here and shown in Fig. 3



global avoided annual $PM_{2.5}$ deaths occurring outside the source region (11,500) are 1.6 times the Northern Hemisphere ozone deaths occurring outside the source region (7,300). For $PM_{2.5}$, >95 % of the avoided deaths occur in the same region where emissions were reduced, while for ozone, that percentage ranges from <50 % in EU to 70 % in NA and EA. Absolute impacts of intercontinental $PM_{2.5}$ and ozone on mortality are comparable for most source-receptor pairs given the large confidence intervals and uncertainties but are substantially greater for $PM_{2.5}$ for pairs not separated by an

ocean—EU on EA ($PM_{2.5}$ deaths are two times that for ozone), EU on SA (3.2 times), and SA on EA (2.25 times). Large uncertainties in modeling and concentration-response relationships for both estimates preclude our ability to draw strong conclusions from this comparison.

A study based on the same multimodel experiments found that intercontinental transport of aerosols accounts for 31 ± 9 % of direct radiative forcing in a receptor region, compared with the influence of both regional emissions and intercontinental transport together (Yu et al. 2013). Here, we find a more limited influence of intercontinental transport on regional $PM_{2.5}$ -related mortality. These results highlight the different drivers of climate versus health impacts, with climate impacts driven by concentrations aloft (where transport is more efficient), surface albedo, and meteorological conditions, while health impacts are driven by near-surface concentrations and the size and vulnerability of the exposed populations.

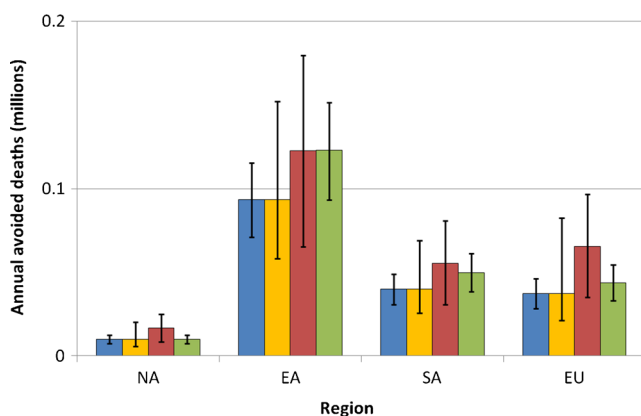


Fig. 5 Annual avoided premature deaths (millions) in each region from 20 % emission reductions in the same region using the multimodel median concentration and concentration-response factor and confidence interval from Krewski et al. (2009) as in the main results (blue), as for the main results but with confidence interval from the variability (shown by the 17th to 83rd percentile) of the model ensemble $PM_{2.5}$ reduction in each grid cell (yellow), as for the main results but using the cardiovascular and lung cancer concentration-response factors and 95 % confidence intervals from Lepeule et al. (2012; red), and as for the main results but using the median of total $PM_{2.5}$ modeled explicitly by seven of the models rather than the sum of SO_4 +POM+BC from the full set of 12 models (green) (Color figure online)

Discussion

We estimate the impacts of intercontinental transport of $PM_{2.5}$ on global premature mortality using multimodel simulations of 20 % emission reductions of anthropogenic primary $PM_{2.5}$ and $PM_{2.5}$ precursors in NA, EU, EA, and SA individually. The emission reductions in all four regions impact within-region mortality most (93–97 % of avoided deaths occur within the source region), but combined avoid 11,500 (95 % confidence interval (CI), 8,800–14,200) annual premature deaths outside the source region. Owing to large downwind populations, 17 and 13 % of the global avoided deaths resulting from NA and EU emission reductions occur outside of the source region, compared with only 4 and 2 % for the SA

and EA source regions. For EU emissions, downwind populations are also in close proximity, leading EU to have the greatest overall impacts on extraregional mortality.

These results are subject to several uncertainties in modeled concentrations and health impact function parameters. We extrapolate the PM_{2.5} concentration-response functions found in the USA to the rest of the world, despite large differences in exposure levels, PM_{2.5} composition, and demographic characteristics around the world, and use the RR estimates from the American Cancer Society study (Krewski et al. 2009) that are likely to understate true impacts. We focus on cause-specific mortality which may be more comparable around the world than all-cause mortality, but note that prevalence of disease subcategories within the broad cardiopulmonary category used here can also vary between countries. Including NO₃ and more accurate secondary organic aerosol representations would likely lead to a larger influence of intercontinental transport than has been calculated here since these secondary PM_{2.5} components can be formed far from the precursor emission location. However, estimated total PM_{2.5} concentrations in some areas are substantially higher than those estimated by the coarsely resolved global models for the three PM_{2.5} components we included in our PM_{2.5} definition (e.g., Brauer et al. 2012). The 20 % emission reductions may have a smaller mortality benefit if we were able to include all PM_{2.5} components in the PM_{2.5} definition since evidence suggests that the concentration-response curve flattens out at high concentrations (e.g., Pope et al. 2009, 2011). The coarse resolution global models used here may also underestimate intraregional health benefits occurring on local scales, affecting these relative contributions of extraregional versus intraregional health benefits. Future studies should strive to estimate the mortality burden of all PM_{2.5} components in the atmosphere in reality and the reduction in the mortality burden that would result from controlling emissions using a concentration-response curve that is appropriate for the range of concentrations examined.

Despite uncertainties, we find that long-range transport of PM_{2.5} can impact health on a global scale, with magnitudes comparable with and, for some neighboring source-receptor pairs, larger than the health impacts of long-range transport of ozone. Many of the models have improved their aerosol representations since these simulations were performed, and source-receptor relationships should be updated based on current understanding of aerosol chemistry and transport. While local emission reductions are likely to be most effective at reducing PM_{2.5}-related mortality in each country, these results suggest that reducing pollution transported internationally would also be health beneficial. In addition, as local emission reductions undertaken in some countries further reduce PM_{2.5} concentrations, pollution originating from other countries becomes increasingly important. International cooperation to reduce pollution transported over long distances

may therefore be an effective complement to national policies controlling local emissions (Liu et al. 2009a; TF HTAP 2010; Yu et al. 2012).

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Supporting information A description of the models participating in the ensemble, a map of the four regions used in this analysis, and additional results can be found in the Supporting Information.

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