Air Qual Atmos Health
DOI 10.1007/s11869-014-0248-9

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

Susan C. Anenberg · J. Jason West · Hongbin Yu · Mian Chin · Michael Schulz · Dan Bergmann · Isabelle Bey · Huisheng Bian · Thomas Diehl · Arlene Fiore · Peter Hess · Elina Marmer · Veronica Montanaro · Rokjin Park · Drew Shindell · Toshihiko Takemura · Frank Dentener

Received: 27 November 2013 / Accepted: 27 January 2014
© Springer Science+Business Media Dordrecht (outside the USA) 2014

Abstract Fine particulate matter with diameter of 2.5 μm or less ($PM_{2.5}$) 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 $PM_{2.5}$ and $PM_{2.5}$ 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

Electronic supplementary material The online version of this article (doi:10.1007/s11869-014-0248-9) contains supplementary material, which is available to authorized users.

S. C. Anenberg (✉)
US Environmental Protection Agency, 1200 Pennsylvania Ave NW MC6301A, Washington, DC 20460, USA
e-mail: anenberg.susan@epa.gov

J. J. West
University of North Carolina, Chapel Hill, NC, USA

H. Yu
University of Maryland, College Park, MD, USA

M. Chin · H. Bian · T. Diehl
NASA Goddard Space Flight Center, Greenbelt, MD, USA

M. Schulz
Norwegian Meteorological Institute, Oslo, Norway

D. Bergmann
Lawrence Livermore National Laboratory, Livermore, CA, USA

I. Bey
Swiss Federal Institute of Technology, Zurich, Switzerland

T. Diehl
Universities Space Research Association, Columbia, MD, USA

A. Fiore
Lamont-Doherty Earth Observatory, Columbia University, Palisades, NY, USA

P. Hess
Department of Biological and Environmental Engineering, Cornell University, Ithaca, NY, USA

E. Marmer
Department of Education, University of Hamburg, Hamburg, Germany

V. Montanaro
University of L'Aquila, L'Aquila, Italy

R. Park
Seoul National University, Seoul, Korea

D. Shindell
NASA Goddard Institute for Space Studies, New York, NY, USA

D. Shindell
Columbia Earth Institute, New York, NY, USA

T. Takemura
Research Institute for Applied Mechanics, Kyushu University, Fukuoka, Japan

F. Dentener
European Commission, Joint Research Center, Institute for Environment and Sustainability, Ispra, Italy

Published online: 06 March 2014

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 (SO_4), nitrate (NO_3), 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 km^2, 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 $0.5°\times0.5°$ 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).

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

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.

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

For a 10-μg/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 μg/m^3). Linearity was also demonstrated up to 30 μg/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 SO_4, 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 μg/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, NO_3, 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 μg/m^3 in NA, EA, SA, and EU, respectively). SO_4 reductions contribute most to reduced total $PM_{2.5}$ concentrations in all regions except SA where POM contributes more. SO_4 also contributes relatively more for foreign receptor regions (54–100 %) than for within the source region (40–77 %) since SO_4 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), and maximum of the gridded multimodel median $PM_{2.5}$ concentration in each region and for the whole world

Region	Population 30+ (millions)	Baseline mortality rate for population 30+ (% per year)		Maximum of the gridded multimodel median $PM_{2.5}$ concentration (μg/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, *SO_4* sulfate, *BC* black carbon, *POM* particulate organic matter

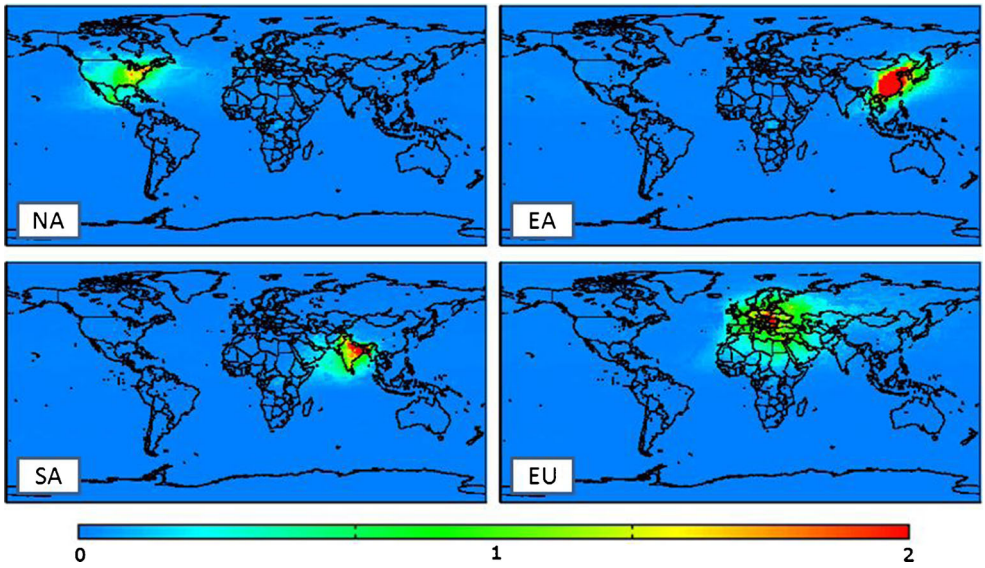

Fig. 1 Reduction in multimodel median annual average $PM_{2.5}$ concentration (microgram per cubic meter) for 20 % regional emission reduction scenarios (for region shown in *bottom left corner*) relative to the base case. Regions are North America (NA), East Asia (EA), South Asia (SA), and Europe (EU)

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

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

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

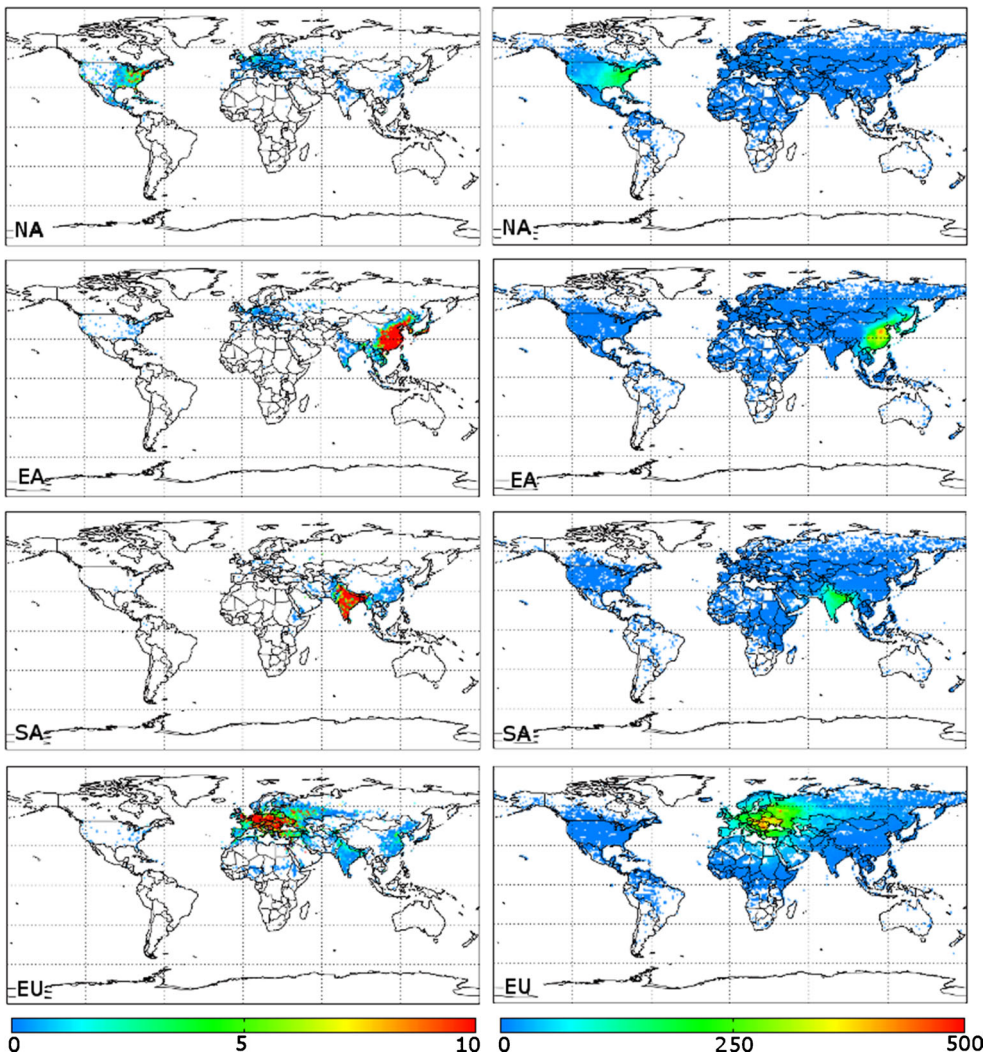

Fig. 2 Annual premature cardiopulmonary and lung cancer deaths per 1,000 km^2 (*left*) and per million people (*right*) due to 20 % primary $PM_{2.5}$ and $PM_{2.5}$ precursor emission reductions in the region shown

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,

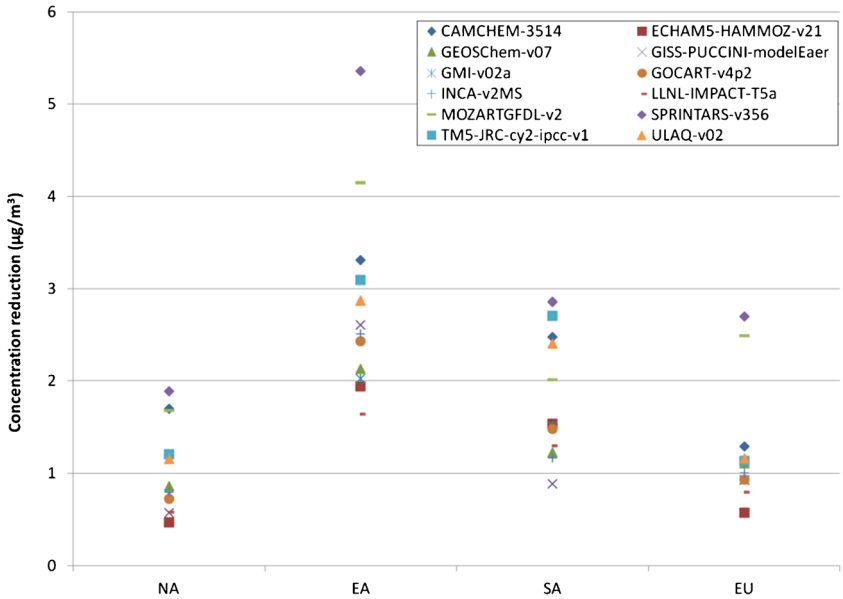

Fig. 3 Population-weighted average reduction in annual average $PM_{2.5}$ concentration due to 20 % anthropogenic emission reductions in the same region, as simulated by each of the 12 models

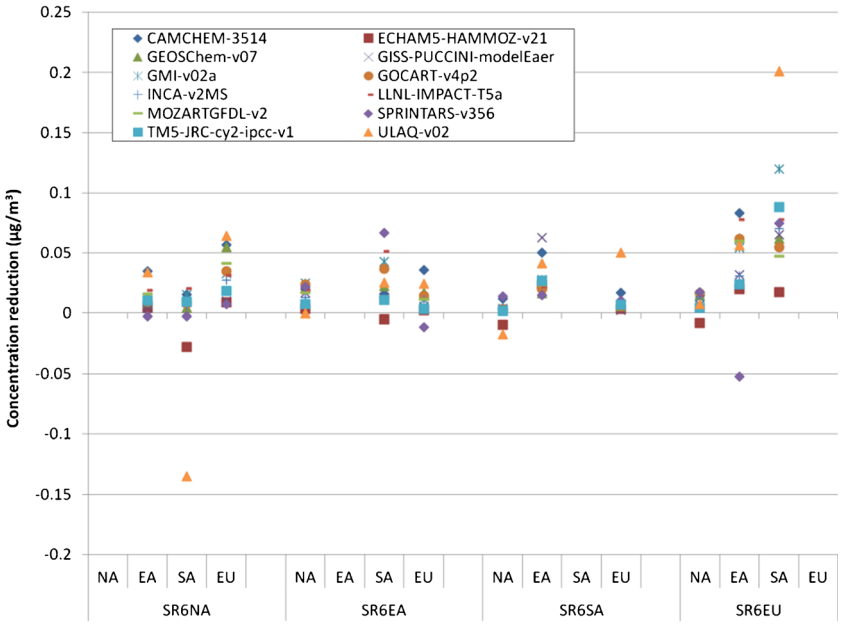

Fig. 4 Population-weighted average reduction in annual average $PM_{2.5}$ concentration in each foreign receptor region due to 20 % anthropogenic emission reductions in each source region (indicated by SR6+the region in which emissions were reduced, e.g., SR6NA for North American emission reductions), 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.

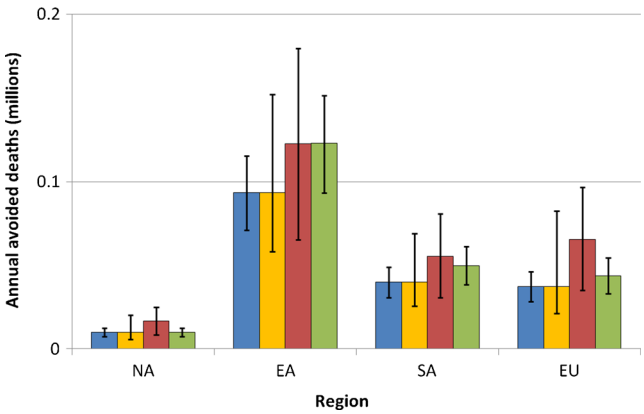

Fig. 5 Annual avoided premature deaths (millions) in each region from 20 % emission reductions in the same region using the multimodel median concentration and concentration-response factor and confidence interval from Krewski et al. (2009) as in the main results (*blue*), as for the main results but 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)

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.

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 NO_3 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).

Acknowledgments The opinions expressed in this article are the authors' and do not necessarily represent those of their employers, including the USEPA. Model simulations were performed under the UN ECE Task Force on Hemispheric Transport of Air Pollution. H.Y. was supported by the NASA Atmospheric Composition Modeling and Analysis Program administered by R. Eckman. D.B. was supported by the US Department of Energy (BER) at LLNL under contract DE-AC5207NA27344.

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.

References

Anenberg SC, West JJ, Fiore AM, Jaffe DA, Prather MJ, Bregmann D, Cuvelier K, Dentener FJ, Duncan BN, Gauss M, Hess P, Jonson JE, Lupu A, MacKenzie IA, Marmer E, Park RJ, Sanderson MG, Schultz M, Shindell DT, Szopa S, Vivanco MG, Wild O, Zeng G (2009) Intercontinental impacts of ozone air pollution on human mortality. Environ Sci Technol 43:6482–6487

Anenberg SC, Horowitz LW, Tong DQ, West JJ (2010) An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. Environ Health Perspect 118:1189–1195

Anenberg SC, Schwartz J, Shindell D, Amann M, Faluvegi G, Klimont Z, Janssens-Maenhout G, Pozzoli L, Van Dingenen R, Vignati E, Emberson L, Muller NZ, West JJ, Williams M, Demkine V, Hicks WK, Kuylenstierna J, Raes F, Ramanathan V (2012) Global air quality and health co-benefits of mitigating near-term climate change through methane and black carbon emission controls. Environ Health Perspect 120:831–839

Beelen R, Raaschou-Nielen O, Stafoggia M, Andersen ZJ, Weinmayr G, Hoffmann B, Wolf K, Samoli E, Fischer P, Nieuwenhuijsen M, Vineis P, Xun WW, Katsouyanni K, Dimakopoulou K, Oudin A, Forsberg B, Modig L, Havulinna AS, Lanki T, Turunen A, Oftedal B, Nystad W, Nafstad P, De Faire U, Pedersen NL, Ostenson C-G, Fratiglioni L, Pennell J, Korek M, Pershagen G, Eriksen KT, Overvad K, Ellermann T, Eeftens M, Peeters PH, Meliefste K, Wang M, Bueno-de-Mesquita B, Sugiri D, Kramer U, Heinrich J, de Hoogh K, Key T, Peters A, Hampel R, Concin H, Nagel G, Ineichen A, Schaffner E, Probst-Hensch N, Kunzli N, Schindler C, Schikowski T, Adam M, Krogh HV, Tsai M-Y, Ricceri F, Sacerdote C, Galassi C, Migliore E, Ranzi A, Cesaroni G, Badaloni C, Forastiere F, Tamayo I, Amiano P, Dorronsoro M, Katsoulis M, Trichopoulou A, Brunekreef B, Hoek G (2013) Effects of long-term exposure to air pollution on natural-cause mortality: an analysis of 22 European cohorts within the multicentre ESCAPE project. Lancet. doi: 10.1016/S0140-6736(13)62158-3

Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F (2004) Ozone and short-term mortality in 95 US urban communities. JAMA 292: 2372–2378

Brauer M, Amann M, Burnett RT, Cohen A, Dentener F, Ezzati M, Henderson SB, Krzyzanowski M, Martin RV, Van Dingenen R, Van Donkelaar A, Thurston GD (2012) Exposure assessment for estimation of the global burden of disease attributable to outdoor air pollution. Environ Sci Technol 46:652–660

Chin M, Diehl T, Ginoux P, Malm W (2007) Intercontinental transport of pollution and dust aerosols: implications for regional air quality. Atmos Chem Phys 7:5501–5517

Chin M, Diehl T, Tan Q, Prospero JM, Kahn RA, Remer LA, Yu H, Sayer AM, Bian H, Geogdzhayev IV, Holben BN, Howell SG, Huebert BJ, Hsu NC, Kim D, Kucsera TL, Levy RC, Mishchenko MI, Pan X, Quinn PK, Schuster GL, Streets DG, Strode SA, Torres O, Zhao X-P (2013) Multi-decadal variations of atmospheric aerosols from 1980 to 2009: sources and regional trends. Atmos Chem Phys Discuss 13:19751–19835

Duncan BN, West JJ, Yoshida Y, Fiore AM, Ziemke JR (2008) The influence of European pollution on the air quality in the Near East and northern Africa. Atmos Chem Phys 8:2267–2283

Ewing SA, Christensen JN, Brown ST, Vancuren RA, Cliff SS, Depaolo DJ (2013) Pb isotopes as an indicator of the Asian contribution to particulate air pollution in urban California. Environ Sci Technol 44: 8911–8916

Fiore AM, Dentener FJ, Wild O, Cuvelier C, Schultz MG, Hess P, Textor C, Schulz M, Doherty RM, Horowitz LW, MacKenzie IA, Sanderson MG, SHindell DT, Stevenson DS, Szopa S, Van Dingenen R, Zeng G, Atherton C, Bergmann D, Bey I, Carmichael G, Collins WJ, Duncan BN, Faluvegi G, Folberth G, Gauss M, Gong S, Hauglustaine D, Holloway T, Isaksen ISA, Jacob DJ, Jonson JE, Kaminski JW, Keating TJ, Lupu A, Marmer E, Montanaro V, Park RJ, Pitari G, Pringle KJ, Pyle JA, Schroeder S, Vivanco MG, Wind P, Wojcik G, Wu S, Zuber A (2009) Multimodel estimates of intercontinental source-receptor relationships for ozone pollution. J Geophys Res 114:D4. doi:10.1029/2008JD010816

Fry MM, Naik V, West JJ, Schwarzkopf MD, Fiore AM, Collins WJ, Dentener FJ, Shindell DT, Atherton C, Bergmann D, Duncan BN, Hess P, MacKenzie IA, Marmer E, Schultz MG, Szopa S, Wild O, Zeng G (2012) The influence of ozone precursor emissions from four world regions on tropospheric composition and radiative forcing. J Geophys Res 117, D07306. doi:10.1029/2011JD017134

Ginoux P, Horowitz LW, Ramaswamy V, Geogdzhayev IV, Holben BN, Stenchikov G, Tie X (2006) Evaluation of aerosol distribution and optical depth in the Geophysical Fluid Dynamics Laboratory coupled model CM2.1 for present climate. J Geophys Res 111:D22

Hadley OL, Ramanathan V, Carmichael GR, Tang Y, Corrigan CE, Roberts GC, Mauger GS (2007) Trans-Pacific transport of black carbon and fine aerosols (D<2.5 μm) into North America. J Geophys Res 112:D05309. doi:10.1029/2006JD007632

Heald CL, Jacob DJ, Park RJ, Alexander B, Fairlie TD, Yantosca RM, Chu DA (2006) Transpacific transport of Asian anthropogenic aerosols and its impact on surface air quality in the United States. J Geophys Res 111, D14310. doi:10.1029/2005JD006847

Health Effects Institute Public Health and Air Pollution in Asia Program (HEI) Public Health and Air Pollution in Asia: coordinated studies of short-term exposure to air pollution and daily mortality in four cities. HEI Research Report 154. Health Effects Institute, Boston, MA, 2010

Jeong JI, Park RJ (2013) Effects of the meteorological variability on regional air quality in East Asia. Atmos Environ 69:46–55

Jerrett M, Burnett RT, Pope CA III, Ito K, Thurston G, Krewski D, Shi Y, Calle E, Thun M (2009) Long-term ozone exposure and mortality. New Engl J Med 360:1085–1095

Keating TJ, West JJ, Farrell AE (2004) Prospects for international management of intercontinental air pollution transport. In: Stohl A (ed) Intercontinental transport of air pollution. Springer, Berlin

Koch D, Schmidt GA, Field C (2005) Sulfur, sea salt and radionuclide aerosols in GISS. ModelE, J Geophys Res 111, D06206. doi:10.1029/2004JD005550

Koch D, Bond TC, Streets DG, Unger N, van der Werf GR (2007) Global impacts of aerosols from particular source regions and sectors. J Geophys Res 112, D02205. doi:10.1029/2005JD007024

Koch D, Schulz M, Kinne S, Bond TC, Balkanski Y, Bauer S, Berntsen T, Boucher O, Chin M, Clarke A, De Luca N, Dentener F, Diehl T, Dubovik O, Easter R, Fahey DW, Feichter J, Fillmore D, Freitag S, Ghan S, Ginoux P, Gong S, Horowitz L, Iversen T, KirkevÃƒÂ¥g A, Klimont Z, Kondo Y, Krol M, Liu X, McNaughton C, Miller R, Montanaro V, Moteki N, Myhre G, Penner JE, Perlwitz J, Pitari G, Reddy S, Sahu L, Sakamoto H, Schuster G, Schwarz JP, Seland O, Spackman JR, Stier P, Takegawa N, Takemura T, Textor C, Van Aardenne JA, Zhao Y (2009) Evaluation of black carbon estimations in global aerosol models. Atmos Chem Phys 9:9001–9026

Koffi B, Schulz M, Breon F-M, Griesfeller J, Balkanski Y, Bauer S, Berntsen T, Chin M, Collins WD, Dentener F, Diehl T, Easter R, Ghan S, Ginoux P, Gong S, Horowitz LW, Iversen T, Kirkevag A, Koch D, Krol M, Myhre G, Stier P, Takemura T, Winker D (2012) Application of the CALIOP layer product to evaluate the vertical distribution of aerosols estimated by global models: AeroCom Phase I results. J Geophys Res 117:D10201. doi:10.1029/2011JD016858

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope CA III, Thurston G, Calle EE, Thun MJ, Beckerman B, DeLuca P, Finkelstein N, Ito K, Moore DK, Newbold KB, Ramsay T, Ross Z, Shin H, Tempalski B (2009) Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Health Effects Institute, Boston, MA

Lamarque J-F, Emmons LK, Hess PG, Kinnison DE, Tilmes S, Vitt F, Heald CL, Holland EA, Lauritzen PH, Neu J, Orlando JJ, Rasch PJ, Tyndall GK (2012) CAM-Chem: description and evaluation of interactive atmospheric chemistry in the Community Earth System Model. Geosci Model Dev 5:369–411

Langner J, Rodhe H, Crutzen PJ, Zimmerman P (1992) Anthropogenic influence on the distribution of tropospheric sulphate aerosol. Nature 359:712–716

Leibensperger EM, Mickley LJ, Jacob DJ, Barrett SRH (2011) Intercontinental influence of NO_x and CO emissions on particulate matter air quality. Atmos Environ 45:3310–3324

Lepeule J, Laden F, Dockery D, Schwartz J (2012) Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities Study from 1974–2009. Environ Health Perspect 12:965–970

Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, Amann M, Anderson HR, Andrews KG, Aryee M, Atkinson C, Bacchus LJ, Bahalim AN, Balakrishnan K, Balmes J, Barker-Collo S, Baxter A, Bell ML, Blore JD, Blyth F, Bonner C, Borges G, Bourne R, Boussinesq M, Brauer M, Brooks P, Bruce NG, Brunekreef B, Bryan-Hancock C, Bucello C, Buchbinder R, Bull F, Burnett RT, Byers TE, Calabria B, Carapetis J, Carnahan E, Chafe Z, Charlson F, Chen H, Chen JS, Cheng AT-A, Child JC, Cohen A, Colson KE, Cowie BC, Darby S, Darling S, Davis A, Degenhardt L, Dentener F, Des Jarlais DC, Devries K, Dherani M, Ding EL, Dorsey ER, Driscoll T, Edmond K, Ali SE, Engell RE, Erwin PJ, Fahimi S, Falder G, Farzadfar F, Ferrari A, Finucane MM, Flaxman S, Fowkes FGR, Freedman G, Freeman MK, Gakidou E, Ghosh S, Giovannucci E, Gmel G, Graham K, Grainger R, Grant B, Gunnell D, Gutierrez HR, Hall W, Hoek HW, Hogan A, Hosgood Iii HD, Hoy D, Hu H, Hubbell BJ, Hutchings SJ, Ibeanusi SE, Jacklyn GL, Jasrasaria R, Jonas JB, Kan H, Kanis JA, Kassebaum N, Kawakami N, Khang YH, Khatibzadeh S, Khoo J-P, Kok C, Laden F, Lalloo R, Lan Q, Lathlean T, Leasher JL, Leigh J, Li Y, Lin JK, Lipshultz SE, London S, Lozano R, Lu Y, Mak J, Malekzadeh R, Mallinger L, Marcenes W, March L, Marks R, Martin R, McGale P, McGrath J, Mehta S, Mensah GA, Merriman TR, Micha R, Michaud C, Mishra V, Hanafiah KM, Mokdad AA, Morawska L, Mozaffarian D, Murphy T, Naghavi M, Neal B, Nelson PK, Nolla JM, Norman R, Olives C, Omer SB, Orchard J, Osborne R, Ostro B, Page A, Pandey KD, Parry CDH, Passmore E, Patra J, Pearce N, Pelizzari PM, Petzold M, Phillips MR, Pope D, Pope Iii CA, Powles J, Rao M, Razavi H, Rehfuess EA, Rehm JT, Ritz B, Rivara FP, Roberts T, Robinson C, Rodriguez-Portales JA, Romieu I, Room R, Rosenfeld LC, Roy A, Rushton L, Salomon JA, Sampson U, Sanchez-Riera L, Sanman E, Sapkota A, Seedat S, Shi P, Shield K, Shivakoti R, Singh

GM, Sleet DA, Smith E, Smith KR, Stapelberg NJC, Steenland K, Stöckl H, Stovner LJ, Straif K, Straney L, Thurston GD, Tran JH, Van Dingenen R, van Donkelaar A, Veerman JL, Vijayakumar L, Weintraub R, Weissman MM, White RA, Whiteford H, Wiersma ST, Wilkinson JD, Williams HC, Williams W, Wilson N, Woolf AD, Yip P, Zielinski JM, Lopez AD, Murray CJL, Ezzati M (2013) A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 380:2224–2260

Liu J, Mauzerall DL, Horowitz LW, Ginoux P, Fiore AM (2009a) Evaluation inter-continental transport of fine aerosols: (1) methodology, global aerosol distribution and optical depth. Atmos Environ 43:4327–4338

Liu J, Mauzerall DL, Horowitz LW (2009b) Evaluating inter-continental transport of fine aerosols: (2) global health impacts. Atmos Environ 43:4339–4347

Oak Ridge National Laboratory (2008) LandScan Global Population Database 2006. http://www.ornl.gov/sci/landscan/index.html. Accessed March 2008

Park RJ, Jacob DJ, Chin M, Martin RV (2003) Sources of carbonaceous aerosols over the United States and implications for natural visibility. J Geophys Res 108:4355. doi:10.1029/2002JD003190

Park RJ, Jacob DJ, Field BD, Yantosca RM, Chin M (2004) Natural and transboundary pollution influences on sulfate-nitrate-ammonium aerosols in the United States: implications for policy. J Geophys Res 109, D15204. doi:10.1029/2003JD004473

Park RJ, Jacob DJ, Kumar N, Yantosca RM (2006) Regional visibility statistics in the United States: natural and transboundary pollution influences, and implications for the Regional Haze Rule. Atmos Environ 40:5405–5423

Pham M, Muller J-F, Brasseur GP, Granier C, Megie G (1995) A three-dimensional study of the tropospheric sulfur cycle. J Geophys Res. doi:10.1029/95JD02095

Pope CA III, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, Thun MJ (2009) Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: shape of the exposure-response relationship. Circulation 120:941–948

Pope CA III, Burnett RT, Turner MC, Cohen A, Krewski D, Jerrett M, Gapstur SM, Thun MJ (2011) Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: shape of the exposure-response relationship. Environ Health Perspect 119:1616–1621

Roman HA, Walker KD, Walsh TL, Conner L, Richmond HM, Hubbell BJ, Kinney PL (2008) Expert judgment assessment of the mortality impact of changes in ambient fine particulate matter in the US. Environ Sci Technol 42:2268–2274

Shindell DT, Chin M, Dentener F, Doherty RM, Faluvegi G, Fiore AM, Hess P, Koch DM, MacKenzie IA, Sanderson MG, Schultz MG, Schulz M, Stevenson DS, Teich H, Textor C, Wild O, Bergmann DJ, Bey I, Bian H, Cuvelier C, Duncan BN, Folberth G, Horowitz LW, Jonson J, Kaminski JW, Marmer E, Park R, Pringle KJ, Schroeder S, Szopa S, Takemura T, Zeng G, Keating TJ, Zuber A (2008) A multimodel assessment of pollution transport to the Arctic. Atmos Chem Phys 8:5353–5372

Takemura T, Okamoto H, Maruyama Y, Numaguti A, Higurashi A, Nakajima T (2000) Global three-dimensional simulation of aerosol optical thickness distribution of various origins. J Geophys Res 105:17853–17873

Takemura T, Nakajima T, Dubovik O, Holben BN, Kinne S (2002) Single-scattering albedo and radiative forcing of various aerosol species with a global three-dimensional model. J Climate 15:333–352

Task Force on Hemispheric Transport of Air Pollution (TF HTAP). Hemispheric Transport of Air Pollution 2010. United Nations Economic Commission for Europe: Geneva, 2010

West JJ, Naik V, Horowitz LW, Fiore AM (2009) Effect of regional precursor emission controls on long-range ozone transport—Part 2: steady-state changes in ozone air quality and impacts on human mortality. Atmos Chem Phys 9:6095–6107

World Health Organization (WHO) (2004) The World Health Report 2004: changing history. World Health Organization, Geneva

World Health Organization (WHO) Mortality Database. http://www.who.int/healthinfo/morttables/en/. Accessed September 2008

Yu H, Remer LA, Chin M, Bian H, Kleidman RG, Diehl T (2008) A satellite-based assessment of transpacific transport of pollution aerosol. J Geophys Res 113:D14S12. doi:10.1029/2007JD009349

Yu H, Remer LA, Chin M, Bian H, Tan Q, Yuan T, Zhang Y (2012) Aerosols from overseas rival domestic emissions over North America. Science 337:566–569

Yu H, Chin M, West JJ, Atherton CS, Bellouin N, Bergmann D, Bey I, Bian H, Diehl T, Forberth G, Hess P, Schulz M, Shindell D, Takemura T, Tan Q (2013) A multimodel assessment of the influence of regional anthropogenic emission reductions on aerosol direct radiative forcing and the role of intercontinental transport. J Geophys Res 118:700–720. doi:10.1029/2012JD0180148