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Evaluating inter-continental transport of fine aerosols:(2) Global health impact

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ABSTRACT

In this second of two companion papers, we quantify for the first time the global impact on premature mortality of the inter-continental transport of fine aerosols (including sulfate, black carbon, organic carbon, and mineral dust) using the global modeling results of (Liu et al., 2009). Our objective is to estimate the number of premature mortalities in each of ten selected continental regions resulting from fine aerosols transported from foreign regions in approximately year 2000. Our simulated annual mean population-weighted (P-W) concentrations of total PM2.5 (aerosols with diameter less than 2.5 μm) are highest in East Asia (EA, 30 μg m^{-3}) and lowest in Australia (3.6 μg m^{-3}). Dust is the dominant component of PM2.5 transported between continents. We estimate global annual premature mortalities (for adults age 30 and up) due to inter-continental transport of PM2.5 to be nearly 380 thousand (K) in 2000. Approximately half of these deaths occur in the Indian subcontinent (IN), mostly due to aerosols transported from Africa and the Middle East (ME). Approximately 90K deaths globally are associated with exposure to foreign (i.e., originating outside a receptor region) non-dust PM2.5. More than half of the premature mortalities associated with foreign non-dust aerosols are due to aerosols originating from Europe (20K), ME (18K) and EA (15K); and nearly 60% of the 90K deaths occur in EA (21K), IN (19K) and Southeast Asia (16K). The lower and higher bounds of our estimated 95% confidence interval (considering uncertainties from the concentration–response relationship and simulated aerosol concentrations) are 18% and 240% of the estimated deaths, respectively, and could be larger if additional uncertainties were quantified. We find that in 2000 nearly 6.6K premature deaths in North America (NA) were associated with foreign PM2.5 exposure (5.5K from dust PM2.5). NA is least impacted by foreign PM2.5 compared to receptors on the Eurasian continent. However, the number of premature mortalities associated with foreign aerosols in NA (mostly occurring in the U.S.) is comparable to the reduction in premature mortalities expected to result from tightening the U.S. 8-h O_3 standard from 0.08 ppmv to 0.075 ppmv. International efforts to reduce inter-continental transport of fine aerosol pollution would substantially benefit public health on the Eurasian continent and would also benefit public health in the United States.

1. Introduction

Human exposure to fine particulate matter is associated with adverse impacts on human health (Bell et al., 2004; Zeka et al., 2005; Pope and Dockery, 2006; Schwartz et al., 2008), including lung cancer and cardiopulmonary mortalities (Pope et al., 2002, 2004). Epidemiological studies (both time-series and cohort studies) have shown that both short-term and long-term exposures to fine particulate matter are associated with elevated rates of premature mortality (Pope et al., 2002; Schwartz et al., 2002, 2008; Samoli et al., 2005; Franklin et al., 2007; Kan et al., 2007; Woodruff et al., 2008). Time-series studies usually examine the effect of short-term exposure to aerosols by analyzing the association between total or specific health outcomes and exposure levels over time, based on routinely collected data such as ambient measurements of aerosol concentrations and hospital records. For example, the time-series study of short-term effects by Kan et al. (2007) found that a 10 μg m^{-3} increase in the 2-day moving average concentration of PM2.5 leads to a 0.36% increase of total mortality in Shanghai, China (based on the data collected from March 2004 to December 2005).

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Unlike short-term time-series studies, cohort studies estimate the association of health outcomes with cumulative exposure over many years. Since both exposure concentrations and length of exposure influence adverse health outcomes, the short-term exposure studies capture only a small fraction of the overall health effects of long-term exposure to PM; long-term exposures have larger and more persistent cumulative effects than short-term exposures (Pope, 2007). For instance, Pope et al. (2002) linked the risk factor data for approximately 500,000 adults (from the American Cancer Society (ACS) cohort study) with ambient air pollution data throughout the United States over a 17-year period from 1982 to 1998 and found that each 10 μg m^{-3} elevation in fine particulate air pollution is associated with approximately a 4% increase in all-cause mortality. In addition, Dockery et al. (1993) examined the effect of long-term air pollution exposure on adult mortalities during the 1970s and 1980s based on the Harvard Six Cities Study. The estimated mortality ratio was 1.13 for a 10 μg m^{-3} increase in $PM_{2.5}$ concentrations. The follow-up cohort study conducted through 1998 found the association to persist (Laden et al., 2006).

A number of time-series and cohort studies have found the concentration–response (CR) relationship between PM exposure and mortality response to be close to linear without a "no-effect" threshold (Daniels et al., 2000; Pope, 2000; Schwartz et al., 2002, 2008; Pope and Dockery, 2006; Qian et al., 2007). This is important for our study as it indicates that any incremental increase in PM exposure (e.g. via inter-continental transport) is associated with the same increase in premature mortality regardless of the background PM concentration. In addition, time-series studies have shown similar CR relationships for PM in both the developing countries of Asia and developed countries in EU and NA (HEI, 2004). Fine particulate air pollution is a mixture of solid particles and liquid droplets that vary substantially in chemical composition and emission sources between cities and seasons (Bell et al., 2007). Due to lack of measurements and toxicological evidence, toxicity of specific particulates composing PM2.5 has not been well characterized. Therefore, to regulate particulate air pollution, most nations promulgate air quality standards on the basis of PM mass below a certain size threshold, without regard to the chemical composition of the particles.

Due to rapid economic growth and increasing fossil fuel combustion, particularly of coal, sulfur emissions in many developing countries have increased substantially in the past few decades and are expected to grow further in the future (Streets et al., 2000; Klimont et al., 2001; Pham et al., 2005). In contrast, effective air pollution regulations have reduced SO_2 emissions significantly in Europe (Moldan et al., 2001; Prechtel et al., 2001) and in the United States (Dutkiewicz et al., 2000; Malm et al., 2002) since 1990. Both observational and modeling studies show that aerosol concentrations are influenced by long-range transport as well as by domestic anthropogenic and natural emissions (Jacob et al., 2003; Jaffe et al., 2003; Park et al., 2003; Derwent et al., 2004; Liu and Mauzerall, 2007; Liu et al., 2008). Since evidence suggests no well-defined threshold below which no adverse health impacts from PM occur, incremental increases in PM concentration due to inter-continental transport can increase rates of premature mortality. Efforts to reduce aerosol concentrations through local emission controls alone will be hampered, in some regions more than others, by increasing emissions from upwind regions. For instance, recent studies indicate that trans-Pacific transport of Asian emissions account for nearly 30–40% of background concentrations of sulfate over the US (Park et al., 2004; Chin et al., 2007). Therefore, quantitatively understanding the magnitude of contribution and the related health damages caused by aerosols transported from foreign continents is of increasing interest to policy makers (TF-HTAP, 2007). Our major objective is to estimate the number of premature mortalities in each continental region resulting from inter-continental transport of foreign fine aerosols. Although domestic emissions (i.e., emitted from the source region) are responsible for most PM2.5 over continental source regions, due to the coarse resolution of our global model, the premature mortalities resulting from domestic emissions are not reported in this paper. Detailed descriptions of our model configuration and evaluation are in the companion paper (Liu et al., 2009). We describe our premature mortality calculations in Section 2. Population-weighted (P-W) concentrations and annual premature mortalities are presented in Section 3, and an uncertainty analysis is in Section 4. We summarize our findings in Section 5.

2. Method

In this study, we use the three-dimensional global chemical transport model (MOZART-2, Model of Ozone and Related Tracers, version 2) driven with NCEP/NCAR reanalysis meteorology (1997–2003) to tag and track inter-continental transport (i.e., transport among the ten continental regions defined in Fig. 1a) of fine aerosols and derive P-W PM2.5 concentrations (including sulfate, black carbon, organic carbon, and fine mineral dust) from ten continental source regions. Anthropogenic emissions (include industrial, residential, and road sources) are based on the RAINS Current Legislation Emission scenario (developed at IIASA) for the year 2000 (Dentener et al., 2005, 2006; Stevenson et al., 2006) and biomass burning emissions are from van der Werf et al. (2003, 2004), based on an average of their 1997–2002 emissions. BC and OC emissions are for the year 1996 and are based on Bond et al. (2004). Detailed descriptions of the PM2.5 simulations, including model configuration, meteorological input, aerosol and aerosol precursor emissions, and chemical tracer configurations as well as model evaluations are given in the companion paper (Liu et al., 2009). In this paper we focus on describing the calculation of P-W aerosol concentrations and premature mortality resulting from inter-continental transport of fine aerosols.

2.1. Population-weighted concentrations

Most atmospheric modeling studies use Area-Weighted (A-W, or area-averaged) concentrations to quantify the average air pollution levels over a region. Area-Weighted (A-W) concentration indicates the average concentration of an air pollutant over the whole domain of a receptor region. In contrast, Population-Weighted (P-W) concentration (see equation (1)), $C_{PW}(S,R)$, represents the average concentration of an air pollutant to which people in a receptor region R are exposed as a result of emissions from a source region S. The calculation of P-W concentrations are based on our simulated annual mean surface aerosol concentrations (averaged from simulations using 1997 to 2003 meteorology) tracked from ten source regions (Fig. 1a), weighted by the 2000 distribution of global population (CIESIN, 2005).

$$\Delta C_{\text{p-w}}(S,R) = \frac{\sum_{i=1}^{n} \Delta C_i(S,R)\cdot POP_i(R)}{\sum_{i=1}^{n} POP_i(R)} \quad (1)$$

where $\Delta C_{\text{P-W}}(S,R)$ is the P-W aerosol concentration in R resulting from aerosol (or precursor) emissions in S; $\Delta C_i(S, R)$ represents aerosol concentrations (emitted from S) in grid box i in R; $POP_i(R)$ is the population in grid box i in R.

2.2. Premature mortality calculation

The annual premature mortalities resulting from air pollution exposure are calculated following the work of (Cifuentes et al., 2001). The concentration-mortality function is:

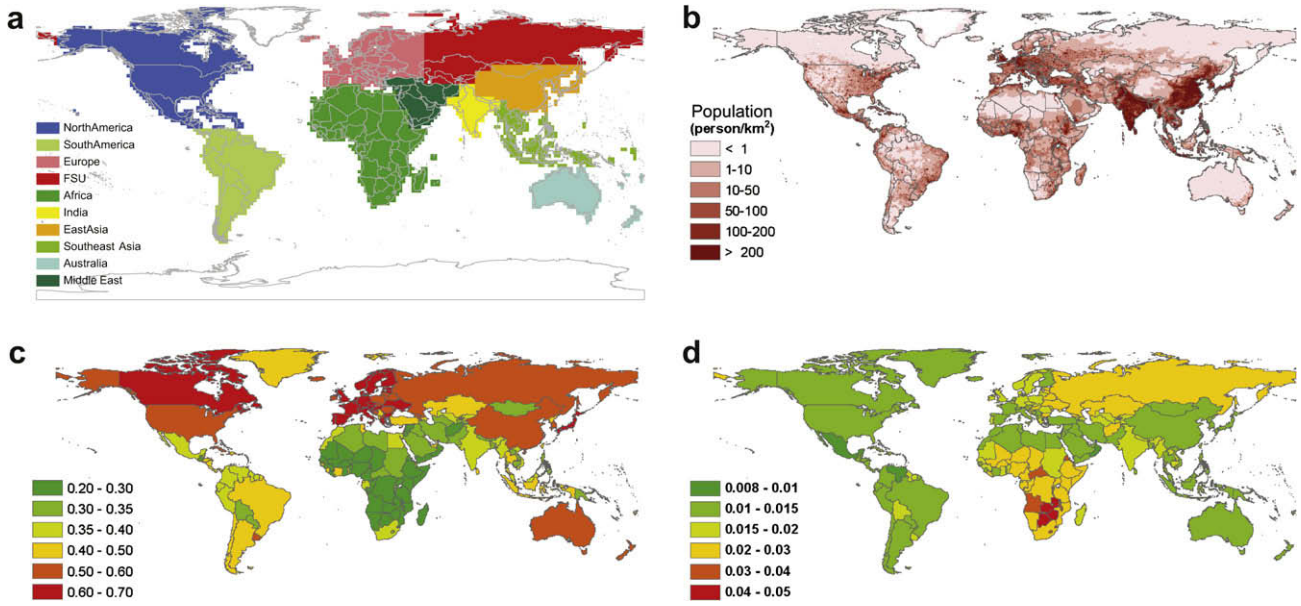

Fig. 1. (a) The ten continental source or receptor regions; (b) global population distribution in 2000 (unit: person km^{-2}, from GWP3); (c) fraction of population age 30 and over in each country in 2000 (from WHO, 2000); (d) baseline mortality rates for adults age 30 and over in each country in 2000 (from WHO, 2000).

$$\Delta Deaths(S,R) = \sum_{i=1}^{n}(POP_i(R)\cdot Mb_i(R)\cdot F_i(S,R)) \quad (2)$$

where $\Delta Deaths(S, R)$ is the number of premature mortalities in region R resulting from aerosol concentrations originating from emissions in region S; $Mb_i(R)$ is the baseline mortality rate in grid box i within R; $F_i(S, R)$ is the fraction of total deaths in grid box i in R contributed by PM2.5 from S (Cifuentes et al., 2001):

$$F_i(S,R) = 1-\exp(-r\cdot\Delta C_i(S,R)) \approx r\cdot\Delta C_i(S,R) \quad (3)$$

In equation (3), r is the increase in the logarithm of relative risk of premature mortality due to a unit increase in air pollution concentrations. Here r corresponds to a 4% increase in mortality rate per 10 μg m^{-3} elevation in PM2.5. This value is based on the findings of the American Cancer Society cohort study conducted in the United States and analyzed by Pope et al. (2002). In our study annual mortalities due to aerosol exposure are estimated only for adults over age 30 because this is the age group included in the ACS study.

Fig. 1b shows the global population distribution for year 2000 (CIESIN, 2005). The fraction (Fig. 1c) and baseline mortalities (Fig. 1d) for adults age 30 and up are calculated based on the age structure in each country. Age structure data are obtained from WHO (2000). We assume the baseline mortality and age structure are uniformly distributed within each country. Table 1 provides the total population, fraction of population age 30 and older, and the baseline mortality rate for those 30+ within each receptor region. We superimpose different layers of population information (see Fig. 1b–d) and spatially join and re-grid them into T62 resolution using ArcGIS version9.2 software. We use the simulated surface aerosol concentrations with population and health data to calculate the global mortality distribution associated with intercontinental transport of fine aerosols.

The relatively coarse resolution of the global model (1.9° × 1.9°) makes it impossible to resolve the high concentrations of particulate pollution in urban areas which also have high population densities. Thus, the premature mortalities resulting from exposure to local emissions would likely be underestimated by our methodology and are not reported in here. However, the coarse resolution should have negligible effect on our estimates of the health impact of intercontinental transport because following long-distance transport aerosol concentrations will not have large gradients within a grid box.

3. Results

3.1. PM2.5 concentrations

In this study, 'domestic' sources indicate aerosols originating from the receptor region itself; 'foreign' sources are aerosols transported from regions outside the receptor region. In addition, we lump aerosols into dust and non-dust PM2.5 because the sources of dust in our model are natural, but the sources of other aerosols are mostly anthropogenic. Note that in this section we confine our attention to population-weighted (P-W) concentrations (eqtn. (1)). Table 2 summarizes the P-W fine aerosol (PM2.5) concentrations over each receptor region due to aerosols originating from each source region.

Table 1
Total population, fraction of age group 30 years and older, and baseline mortality rate in each receptor region (NA, North America; SA, South America; EU, Europe; FSU, the Former Soviet Union; AF, Africa; IN, the Indian subcontinent; EA, East Asia; SE, Southeast Asia; AU, Australia; ME, the Middle East).

	NA	SA	EU	FSU	AF	IN	EA	SE	AU	ME
POP[a]	478	353	671	116	793	1316	1471	538	24	282
F_{30+}[b]	0.53	0.42	0.58	0.50	0.30	0.38	0.51	0.39	0.57	0.33
M_{b30+}[c]	0.0131	0.0123	0.0180	0.0224	0.0213	0.0160	0.0117	0.0139	0.0112	0.0132

[a] Total population in 2000, GWP V3 (unit: millions).
[b] Fraction of population in age group 30 years and older, WHO (2000).
[c] Baseline mortality rate for age group 30 years and older, WHO (2000).

Table 2
Inter-regional source–receptor relationships of annual average population-weighted surface concentrations of fine aerosols (PM2.5) (units: μg m^{-3}). Due to the coarse resolution of the model, italicized values along the diagonal are a conservative estimate of PM2.5 resulting from intra-regional emissions. "Total" in the bottom row indicates simulated total PM2.5 concentrations for a receptor region (ROW, rest of the world, indicates sources from the untagged regions as well as sulfate aerosols produced from DMS).

	Receptor									
	NA	SA	EU	FSU	AF	IN	EA	SE	AU	ME
Source										
NA	*8.73*	0.02	0.14	0.07	0.05	0.06	0.04	0.01	0.00	0.12
SA	0.01	*5.89*	0.00	0.00	0.02	0.01	0.00	0.00	0.04	0.00
EU	0.01	0.00	*7.40*	0.67	0.40	0.05	0.10	0.01	0.00	1.00
FSU	0.02	0.00	0.24	*5.10*	0.03	0.24	0.33	0.02	0.00	1.77
AF	0.38	0.46	0.97	0.92	*21.0*	2.70	0.47	0.37	0.08	5.36
IN	0.01	0.00	0.01	0.13	0.03	*13.9*	0.11	0.33	0.00	0.17
EA	0.09	0.00	0.05	0.41	0.03	0.09	*28.3*	1.24	0.00	0.09
SE	0.00	0.00	0.00	0.00	0.00	0.07	0.12	*5.85*	0.00	0.00
AU	0.00	0.01	0.00	0.00	0.01	0.01	0.00	0.16	*2.96*	0.00
ME	0.05	0.02	0.15	0.95	0.87	2.57	0.21	0.20	0.01	*12.7*
ROW	0.55	0.86	0.56	0.11	0.39	0.18	0.44	0.99	0.53	0.25
Total	9.87	7.28	9.52	8.36	22.9	19.9	30.1	9.16	3.62	21.5

The annual average surface P-W PM2.5 concentrations are relatively high over EA (30 μg m^{-3}), AF (23 μg m^{-3}), ME (22 μg m^{-3}), and IN (20 μg m^{-3}), but are less than 10 μg m^{-3} over other regions. In NA, the P-W PM2.5 concentration (~10 μg m^{-3}) is mostly due to sulfate (~55%) and carbonaceous (~38%) aerosols. Dust accounts for only 7% of total PM2.5 in NA, of which nearly 70% originates from inter-continental transport. Domestic non-dust aerosols are the dominant source (86%) of PM2.5 in NA. Inter-continental transport of foreign non-dust aerosols accounts for less than 0.1 μg m^{-3} in NA, nearly half of which originates from EA. In EU, total PM2.5 is similar to NA, but nearly 22% originates from foreign emissions (AF dust and NA non-dust contribute 0.9 μg m^{-3} and 0.15 μg m^{-3} to PM2.5 in EU, respectively). In EA, most of the PM2.5 originates from domestic sources (~94%), with sulfate, carbonaceous, and dust aerosols contributing 38%, 37% and 25% to PM2.5, respectively. In contrast, in IN approximately 30% of PM2.5 is contributed from foreign sources (mostly from AF and ME dust).

In (Liu et al., 2009), we calculated the A-W surface PM2.5 concentration over each receptor region. The A-W PM2.5 concentrations are generally within a factor of 3 (up to a factor of 4) of P-W PM2.5 concentrations (see Table 3 in (Liu et al., 2009)). Table 3 lists the ratios between P-W and A-W PM2.5 concentrations (RPA). When the RPA value is larger (smaller) than 1, it indicates that the spatial distribution of the air pollution has a higher (lower) coincidence with population and hence influences human health more (less) than is indicated by the surface air quality over the whole domain of a receptor region. The RPA for domestic PM2.5 is usually larger than 1 except in regions with substantial dust emissions (i.e., AF, ME, and AU) because the distribution of anthropogenic emissions are spatially positively correlated with population whereas natural emissions (e.g., mineral dust) are not. The RPA values for domestic emissions over the NA and EA receptors are relatively high. However, over EU, the RPA value is relatively small even though most PM2.5 originates from anthropogenic sources, because the population in EU is more evenly distributed than in NA or EA.

Table 3
Ratios of population-weighted (P-W) annual mean PM2.5 concentrations to Area-weighted (A-W) concentrations for each pair of source–receptor regions.

	Receptor									
	NA	SA	EU	FSU	AF	IN	EA	SE	AU	ME
Source										
NA	*2.4*	1.7	1.1	1.8	0.7	1.0	0.8	1.3	0.5	1.0
SA	1.1	*1.2*	1.1	2.6	0.8	1.0	0.7	0.9	0.9	0.6
EU	0.7	0.9	*1.4*	1.7	1.2	0.8	0.8	1.5	0.4	1.5
FSU	0.9	0.9	0.6	*1.6*	1.0	0.8	0.6	1.3	0.5	1.3
AF	1.1	1.0	0.9	2.7	*0.9*	1.0	0.7	1.2	0.6	0.7
IN	1.2	0.9	1.1	4.3	1.0	*1.4*	0.4	1.4	0.5	0.9
EA	1.0	1.0	1.1	1.6	0.7	0.7	*1.9*	1.7	0.5	1.0
SE	1.3	1.0	1.1	2.5	0.9	1.2	1.3	*2.1*	0.2	0.7
AU	1.5	1.0	1.0	2.4	0.9	1.0	0.8	1.1	*0.6*	0.7
ME	1.2	0.9	0.8	3.8	1.3	0.8	0.6	1.2	0.5	*0.8*
ROW	1.8	1.5	1.0	0.7	0.9	0.9	1.1	1.8	1.4	1.0

The RPA values for inter-continental transport are usually close to 1. However, for the FSU receptor, the RPA for foreign aerosols are much larger than 1, particularly for the ME (3.8) and IN (4.3) sources, indicating the aerosols transported from these two regions are mostly distributed over the populated regions over FSU. In contrast, for AU and EA, the RPA for foreign aerosols are usually less than 1, indicating foreign aerosols are mostly distributed over remote areas of those regions.

3.2. Annual premature deaths

According to equations (1)–(3), the relationship between human exposure and premature death is approximately linear. However, the spatial variations in population density, baseline mortality and age structure as well as variability in emissions and transport patterns results in significant differences in health impacts from inter-continental transport of PM2.5 among these ten regions. Fig. 2a shows the annual premature mortality we calculate in each receptor region as a result of inter-continental transport of fine aerosols from the nine foreign source regions (the corresponding number of deaths for each source–receptor pair is listed in Table 4).

Each year, according to our simulation, nearly 380K premature deaths are associated with inter-continental transport of fine aerosols among these ten continental regions. Approximately half of these deaths (~180K) occurred in IN, mostly caused by fine aerosols transported from AF (~83K) and the ME (~77K). The EA, EU, and ME regions receive the next largest number of premature mortalities (~40–60K deaths each) from foreign aerosols. AF and the FSU are the primary aerosol sources leading to these premature mortalities. Over AF and SE, inter-continental PM2.5 leads to approximately 25K premature deaths in each region, mostly resulting from ME and EA aerosols, respectively. For other regions, due to either small population size or distance from other continents, premature mortalities associated with foreign PM2.5 are relatively small (≤10K). For instance, in 2000 our simulation indicates that nearly 7K adult premature mortalities in NA are due to exposure to foreign aerosols, particularly from AF (4K) and EA (1K).

We find that fine dust dominates inter-continental transport of PM2.5 (not shown). According to our simulation, approximately 80% of the $PM_{2.5}$ from foreign sources in NA, SA, EU, IN and the ME is fine dust. However, little research exists on the health effects of dust exposure (only a few studies found a positive relationship between the East Asian sandstorm events and hospital admissions for stroke and ischemic heart diseases in Taiwan (Yang et al., 2005; Bell et al., 2008)). We therefore separately estimate the premature mortality caused by non-dust aerosols in Fig. 2b. In 2000, we calculate approximately 90K premature mortalities resulted from non-dust aerosols of foreign origin, substantially less than the deaths estimated for total foreign PM2.5. Inter-continental transport of foreign non-dust aerosols potentially leads to 15–20K deaths in each of the EA, IN and SE regions (5–10K deaths each in EU, AF, ME and FSU, and less than 1.2K each in NA, SA and AU, see Table 5). In addition, the non-dust aerosols from EA and EU are the primary sources of premature deaths due to exposure to foreign aerosols in NA and SE, and the ME, AF and FSU regions, respectively.

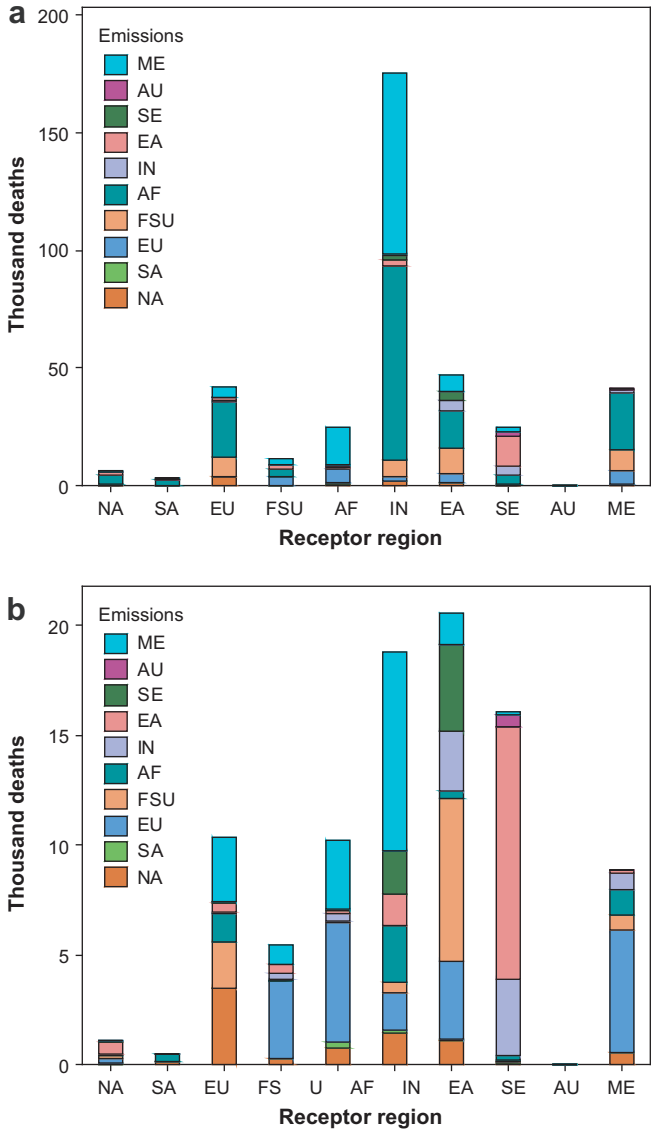

Fig. 2. Annual adult (age 30 and over) premature mortalities in each receptor region associated with inter-continental transport of (a) fine aerosols (PM2.5) (b) non-dust aerosols from the nine other source regions.

Fig. 3 compares the impact on premature mortalities of inter-continental import and export of fine aerosols. 'Import' indicates the total number of premature mortalities in a region caused by aerosol emissions that arrive from the 9 other regions. 'Export' indicates the total number of premature mortalities outside the region resulting from inter-continental transport of the region's emissions. For mortalities from total PM2.5 (including dust, see Fig. 3a), AF and the ME are the two largest "net export" (i.e., export minus import) regions, and IN is the largest "net import" (i.e., import minus export) region. However, for mortalities from non-dust aerosols (Fig. 3b), EU and the ME are the two regions with the largest "net export", and IN and SE are the two regions with the largest "net import". Although non-dust aerosols (and precursor) emissions from IN are much larger than those from the ME (Liu et al., 2009), due to the influence of the Indian monsoon and the effect of the Himalaya mountains, Indian pollutants are rapidly exported to either the Indian ocean or higher altitudes above EA (Liu and Mauzerall, 2005; Liu et al., 2005). As a result, the efficiency with which IN pollutants influence foreign surface air quality and foreign populations is much less than that of emissions from the ME (Liu and Mauzerall, 2007). Therefore, a reduction in anthropogenic emissions from 2000 levels in EU and ME would significantly benefit public health in downwind regions.

4. Uncertainties

Uncertainties exist at every step of our study: emissions, meteorological inputs (i.e., uncertainties in NCEP/NCAR reanalysis data (Smith et al., 2001)), physical transport, chemical reactions, MOZART-2 performance (Horowitz et al., 2003; Tie et al., 2005; Ginoux et al., 2006; Horowitz, 2006; Liu et al., 2009), population data (i.e., population distribution, age structure, and baseline mortality), exposure estimates, application of epidemiological relationships, and attribution of premature mortalities to PM component. Despite these uncertainties, we believe this analysis provides a valuable set of first estimates of the health impacts of global inter-continental transport of aerosols. We highlight some of the key areas of uncertainty below and conservatively quantify the uncertainty range of our calculation.

4.1. Emission inventory and model simulation

The gas-phase and aerosol emissions inventories used in this study are from a variety of sources. However, most of the emission

Table 4
Annual premature mortalities for adults (age 30 and over) as a result of inter-continental transport of PM2.5. Numbers listed below are mean deaths. The corresponding 95% CI[a] is [0.18, 2.4] × deaths. Due to the coarse resolution of the model which would likely lead to an underestimate of exposure resulting from urban emissions, intra-regional premature mortalities are not reported.

	Receptor									
	NA	SA	EU	FSU	AF	IN	EA	SE	AU	ME
Source										
NA		150	3700	310	830	1700	1300	82	0	600
SA	77		11	3	490	170	26	51	21	12
EU	200	5		3500	5400	1600	3500	67	0	5600
FSU	320	11	8100		530	7300	11,000	190	0	8900
AF	4000	2600	24,000	3100		83,000	16,000	4100	46	24,000
IN	130	15	150	380	640		3900	3900	0	1000
EA	1200	19	1400	1500	500	2700		13,000	0	470
SE	31	10	23	5	61	2000	4000		1	20
AU	16	110	15	3	190	160	37	1600		12
ME	590	130	4500	2800	16,000	77,000	7000	2300	4	
ROW	5600	5900	14,000	520	7000	5700	16,000	10,000	290	1300
Total	12,000	8900	56,000	12,000	32,000	180,000	63,000	35,000	370	42,000

[a] CI: confidence interval (details on how we quantify the 95% CI are provided in Section 4).

Table 5
Same as Table 4, but for non-dust PM2.5.

	Receptor									
	NA	SA	EU	FSU	AF	IN	EA	SE	AU	ME
Source										
NA		140	3500	280	720	1400	1100	70	0	520
SA	74		9	2	320	140	21	32	8	10
EU	200	5		3500	5400	1600	3500	67	0	5600
FSU	120	0	2100		44	540	7500	43	0	640
AF	54	320	1300	70		2600	350	160	20	1200
IN	60	3	53	270	370		2700	3500	0	700
EA	530	6	430	380	160	1400		12,000	0	120
SE	31	10	23	5	61	2000	4000		1	20
AU	0	25	0	0	16	3	1	490		0
ME	53	1	3000	930	3100	9000	1400	140	0	
ROW	5600	5800	14,000	520	6900	5600	16,000	10,000	280	1300
Total	6800	6400	24,000	6000	17,000	24,000	37,000	26,000	310	10,000

inventories are developed by applying emission factors from a few countries to the rest of the world. In reality, emission factors for a given source could vary tremendously over time and between countries. For example, up to a factor of 2 uncertainty is associated with BC and OC emissions (Bond et al., 2004). Also, because our inventories approximately represent year 2000 it is likely that emissions and resulting source–receptor relationships today may be different. For example, sulfur dioxide emissions in the United States and Europe have decreased since 2000. In addition, uncertainties result from model parameterizations including meteorological input, resolution, simulations of transport and chemistry (e.g. Horowitz (2006) finds large uncertainties associated with aerosol wet deposition), composition (e.g., internal versus external aerosol mixtures) and size distribution of aerosols.

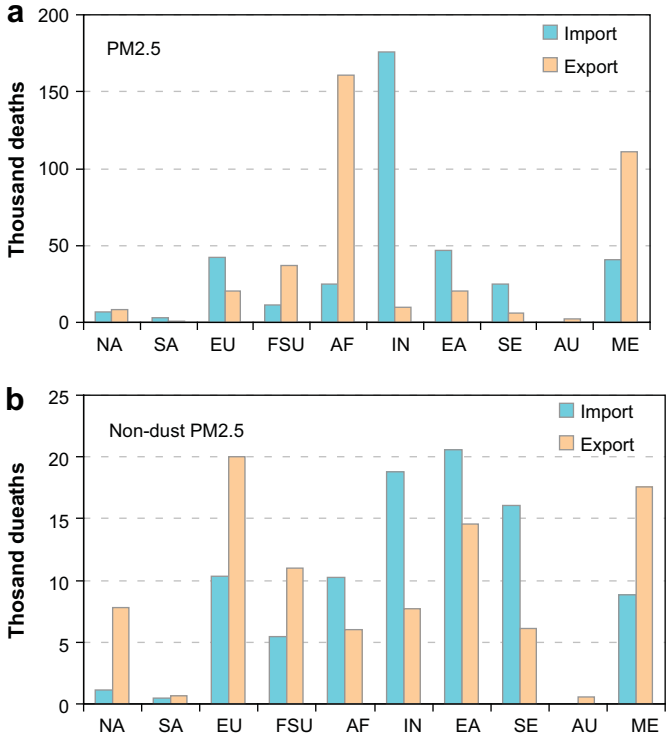

Fig. 3. Annual premature mortalities associated with inter-continental transport of (a) total PM2.5 (b) non-dust PM2.5 in each receptor region (Note scale changes). 'Import' indicates the total number of deaths in a receptor region resulting from emissions from the nine other tagged regions, and 'Export' gives the total number of deaths occurring in the other nine regions as a result of emissions from the given region.

4.2. Concentration–response (CR) relationship

In this study we use the CR relationship from the American Cancer Society (ACS) U.S. cohort study described in Pope et al. (2002) to evaluate the impact of PM2.5 on premature mortality. The actual toxicity-determining characteristics of PM2.5 are uncertain (Bell et al., 2004). The health response from exposure to inter-continental aerosol components could be different from that of the PM2.5 mixture to which the cohorts in the ACS study were exposed. In addition, the harmful components of PM2.5 may differ by health endpoint (i.e., cause of death or illness) or population subgroup (Bell et al., 2004). However, due to a lack of cohort studies conducted in other parts of the world as well as the fact that time-series studies have shown similar CR relationships for PM in developing countries in Asia and developed countries in EU and NA (HEI, 2004), we assume that the CR relationship identified by Pope et al. (2002) is applicable globally. In addition, due to lack of specific information on toxicity, we assume that sulfate, BC, OC, and dust aerosols all have the same effects on human mortality. However, we separately tabulate dust-related mortalities as one way to address this uncertainty. Furthermore, we only estimate the mortalities for adults age 30 and up and do not include infant deaths. In fact, human mortality responses to long-term fine particulate exposure may depend on race, age, health status and aerosol species. In addition, the 95% confidence interval for CR values has a very broad range with the upper bound almost 6 times larger than the lower bound (Pope et al., 2002); this contributes significantly to our total uncertainty. Furthermore, large uncertainties exist in the population data, including population distribution, age structure, baseline mortality, and threshold (i.e., zero-effect level) of aerosols on human health.

4.3. Quantitative uncertainty analysis

We conduct Monte Carlo simulations to quantify the uncertainty range for our mortality calculation. As shown in equation (3), in the linear limit when "$r\cdot\Delta C_i$" is small, F_i is approximately equal to $r\cdot\Delta C_i$. By applying this approximation and equation (1) to equation (2), we obtain:

$$\Delta Deaths(S,R) = \sum_{i=1}^{n}[POP_i(R)\cdot Mb_i(R)\cdot r\cdot\Delta C_i(S,R)]$$
$$= \sum_{i=1}^{n}[D_i(R)\cdot r\cdot\Delta C_i(S,R)] = D(R)\cdot r\cdot\Delta C_{D\text{-}W}(S,R) \quad (4)$$

Where $D_i(R)$ is the all-cause deaths in grid box i in R; $D(R)$ is the total deaths in R; $\Delta C_{D\text{-}W}(S, R)$ is the all-cause death-weighted

aerosol concentration in R resulting from aerosol emissions in S: '$r \cdot \Delta C_{D\text{-}W}(S, R)$' is the fraction of total deaths in R caused by PM2.5 originating from S.

Equation (4) indicates a linear concentration–mortality relationship. If the uncertainties associated with population data (i.e., population distribution and variation of baseline mortality rate within a nation) are ignored, the uncertainty range of premature mortalities, calculated by equation (2), should be proportional to that of '$r \cdot \Delta C_{D\text{-}W}(S, R)$'.

According to Pope et al. (2002), each 10 μm elevation in PM2.5 is associated with approximately a 4% increased risk in all-cause mortality. The corresponding 95% Confidence Interval (CI) is [1%, 8%]. In addition, by comparing the simulated PM2.5 with the observed PM2.5 concentrations over several observational networks (i.e. IMPROVE for the United States, EMEP for Europe, EANET for East Asia as well as the RSMAS global observations from University of Miami), Liu et al. (2009) find that the simulated annual mean aerosol concentrations in general agree within a factor of 2 of these observations. By assuming a normal distribution for r (i.e., concentration–response relationship) and a uniform distribution associated with a factor of 2 of uncertainties for $\Delta C_{D\text{-}W}$ (note the inter-continental component of PM2.5 could be even more uncertain), we conduct Monte Carlo simulations for 1,000,000 trials for the term of $r \cdot \Delta C_{D\text{-}W}$. The resulting 95% CI of premature mortalities (in Tables 4 and 5) is [0.18 × deaths, 2.4 × deaths]. This is a conservative estimate of uncertainty because we ignore the uncertainties in population distribution, age structure, and baseline mortality rates within each receptor region. The true uncertainty range could be broader.

Future epidemiological studies (particularly long-term cohort studies for non-US populations) on the health effects of fine aerosols could significantly reduce the uncertainties of our estimate. In addition, improvements in emission inventories (i.e., annual amount, spatial and temporal distribution, and height of emissions), population data (i.e., spatial distribution of age structure and baseline mortality), model simulations (e.g. detailed simulations of aerosol and cloud microphysics as well as cloud chemistry), additional observational data in developing countries, and higher model resolution would also be beneficial.

5. Conclusions and policy implications

Given increasing concern about inter-continental transport of air pollution, we estimate the impacts of inter-continental transport of aerosols on premature mortality using a simulation of inter-continental transport of fine aerosols (PM2.5) tracked from ten continental source regions. A detailed explanation of the model simulation and evaluation are in the companion paper (Liu et al., 2009). We calculate both the population-weighted (P-W) human exposures and estimate for the first time the adult premature mortalities resulting from inter-continental transport (i.e., the transport among the ten defined continental regions) of sulfate, BC, OC and fine dust aerosols. We find that the population-weighted PM2.5 concentrations for each pair of source–receptor regions differ from the area-weighted PM2.5 concentrations by a factor of 1–4. The annual average P-W fine aerosol concentrations (PM2.5) are largest in EA (30 μg m^{-3}), AF (23 μg m^{-3}), ME (22 μg m^{-3}), and IN (20 μg m^{-3}) due to large SO_2 and BC/OC emissions (from EA, IN) as well as dust emissions (from AF and ME), and are relatively small in other regions (<10 μg m^{-3}). Nearly 90% of the P-W aerosol concentrations in EA, AF and NA occur as a result of emissions from domestic sources. In contrast, less than 75% of aerosol exposure in the ME, FSU, IN and SE come from domestic sources. In NA, inter-continental sources account for only 0.58 μg m^{-3} (6%) of P-W aerosol concentrations, of which 85% is contributed from fine dust, primarily from AF (73%). The foreign influence of EA aerosols is mostly on SE (1.2 μg m^{-3}), while the trans-Pacific transport from EA is relatively small, only accounting for 0.09 μg m^{-3} $PM_{2.5}$ in NA, much lower than the dust contribution from AF (0.38 μg m^{-3}).

Based on the P-W aerosol concentrations, we estimate annual premature mortalities for adults age 30 and up due to inter-continental transport of aerosols. We find that nearly 380K premature deaths per year globally are associated with exposure to foreign fine aerosols. Approximately half of these deaths occur in IN (~180K), mostly caused by fine aerosols transported from AF (~83K) and the ME (~77K). We assume that different PM2.5 components have the same effects on premature mortality since no epidemiological study differentiates between them.

Dust is the dominant component of fine aerosols transported inter-continentally. Inter-continental transport of non-dust aerosols accounts for ~90K deaths globally with more than half of the premature mortalities associated with foreign non-dust aerosols originating from EU (20K), ME (18K) and EA (15K); and nearly 60% of the 90K deaths occurring in Asia (i.e., EA (21K), IN (19K) and SE (16K)).

We conduct Monte Carlo simulations to quantify the uncertainties associated with the mortality calculation. Based on the uncertainty ranges associated with the concentration–response relationship (determined from epidemiological studies) as well as the simulated aerosol concentrations (from MOZART-2 model evaluations), the lumped 95% CI of the mortality results is [0.18 × deaths, 2.4 × deaths]. However, if more sources of uncertainties are considered, the true 95% CI could be broader. Future improvements in epidemiological studies, model simulations, as well as population information will help to narrow the uncertainty range.

The findings in this study have important policy implications. For instance, we find that in 2000 nearly 6.6K premature deaths in North America (~4K in the United States) are associated with PM2.5 from foreign regions. In particular, foreign non-dust aerosols account for 1.1K deaths in North America (~0.9K in the United States), with about half contributed from EA. We find that the number of calculated premature mortalities associated with foreign aerosols in the United States is comparable to the reduction in premature mortalities expected from tightening the U.S. 8-h O_3 standard from 0.08 ppmv to 0.075 ppmv, at a cost of billions of US$ each year (USEPA, 2008). This puts the magnitude of the health consequences of inter-continental transport of PM2.5 in the context of current domestic air quality efforts.

As shown in this study, North America is less impacted by foreign PM2.5 than other regions (e.g., the Indian subcontinent, East Asia, and Europe). In addition, we consider only 10 large continental regions. The premature mortalities associated with actual transboundary transport (i.e., air pollution transported from one nation to another) could be significantly larger. This implies that each year substantial global health damages are likely associated with transboundary transport of PM2.5, especially among the countries on the Eurasian continent. Therefore, given the linear source–receptor relationship for inter-continental transport of aerosols (Liu et al., 2008) and the linear increase of premature mortalities with rising PM2.5 concentrations (Schwartz et al., 2008), international efforts to reduce such transport would benefit public health particularly in countries on the Eurasian continent (i.e., EU, FSU, ME, IN, EA and SE) as well as in the United States.

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