Estimates of global mortality attributable to particulate air pollution using satellite imagery

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ABSTRACT

Background: Epidemiological studies of the health effects of air pollution have traditionally relied upon ground-monitoring stations to measure ambient concentrations. Satellite derived air pollution measures offer the advantage of providing global coverage.
Objective: To undertake a global assessment of mortality associated with long-term exposure to fine particulate air pollution using remote sensing data.
Methods: Global $PM_{2.5}$ exposure levels were derived from the MODIS and MISR satellite instruments. Relative risks and attributable fractions of mortality were modeled using previously developed concentration–response functions for the association between $PM_{2.5}$ and mortality.
Results: The global fraction of adult mortality attributable to the anthropogenic component of $PM_{2.5}$ (95% CI) was 8.0% (5.3–10.5) for cardiopulmonary disease, 12.8% (5.9–18.5) for lung cancer, and 9.4% (6.6–11.8) for ischemic heart disease.
Conclusion: This study demonstrates the feasibility of using satellite derived pollution concentrations in assessing the population health impacts of air pollution at the global scale. This approach leads to global estimates of mortality attributable to $PM_{2.5}$ that are greater than those based on fixed site ground-level measures of urban $PM_{2.5}$, but more similar to estimates based on global chemical transport model simulations of anthropogenic $PM_{2.5}$.

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1. Introduction

Increased mortality due to exposure to fine particular matter ($PM_{2.5}$) has been documented in a number of cohort studies, including the Harvard Six Cities Study (Dockery et al., 1993; Krewski et al., 2000), the American Cancer Society (ACS) cohort study (Krewski et al., 2009; Pope et al., 2002), the Veterans Aging Cohort Study (Lipfert et al., 2006), the Medicare cohort studies (Zeger et al., 2008), and the Nurses' Health Study cohort (Puett et al., 2008). Significant associations between $PM_{2.5}$ and mortality from all causes, cardiovascular disease, cardiopulmonary disease, and lung cancer have been reported (Pope and Dockery, 2006).

Air pollution was estimated to represent 1.4% of the total mortality attributable to 26 risk factors assessed in the previous World Health Organization's (WHO) global burden of disease project (GBD) (Ezzati et al., 2002). The previous WHO GBD study estimated that urban $PM_{2.5}$ exposure was responsible for approximately 712,000 cardiopulmonary and 62,000 lung cancer deaths in 2000 (Cohen et al., 2004). More recent estimates extending beyond urban areas are far greater with 3.5 million cardiopulmonary and 220,000 lung cancer annual deaths being attributed to the anthropogenic component of $PM_{2.5}$ (Anenberg et al., 2010). Comparison of the previous and current GBD studies indicates much higher exposure assessment due to the inclusion of satellite remote sensing and chemical transport models to provide global estimates (Brauer et al., 2011).

Aerosol dispersion varies spatially and temporally, and can display both local and regional patterns, depending on their source (Chu et al., 2003). These characteristics of aerosols limit the ability of fixed site ground-based PM monitors to capture large-scale, regional and global PM distributions (Gupta et al., 2006). Satellite-mounted sensors can account for spatial variability and long-range transport of air

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pollution, and thus provide global coverage of aerosols by measuring concentration gradients between stationary monitors, as well as in regions that lack ground monitoring capabilities all together (Engel-Cox et al., 2004).

The Moderate Resolution Imaging Spectroradiometer (MODIS) and the Multi-angle Imaging Spectroradiometer (MISR), two instruments onboard the National Aeronautics and Space Administration's Terra satellite, were designed specifically for the purpose of retrieving aerosol data (King et al., 1999). Both MISR and MODIS derived aerosol pollution measures have been found to be well correlated with ground-level aerosol pollution measures under certain conditions (Chu et al., 2003; Gupta et al., 2006; Hutchison et al., 2005; Kahn et al., 2005; Liu et al., 2004; van Donkelaar et al., 2006). For instance, MODIS aerosol optical depth (AOD) and daily ground-based PM_{10} measures were linearly correlated, $r = 0.82$ at a site in northern Italy (Chu et al., 2003); MODIS AOD and average binned ground-based $PM_{2.5}$ concentrations were linearly correlated, $r = 0.96$, across major urban areas (Gupta et al., 2006); and, annual mean MISR versus surface-level $PM_{2.5}$ concentrations were linearly correlated, $r = 0.78–0.81$, over the US (Liu et al., 2004). In another study, annual mean $PM_{2.5}$ concentrations obtained from MODIS and MISR in conjunction with a global atmospheric chemical transport model were linearly correlated with surface $PM_{2.5}$ measurements in Canada and the United States, $r = 0.58$ for MISR and $r = 0.69$ for MODIS (van Donkelaar et al., 2006). The agreement between satellite derived and ground based measures of $PM_{2.5}$ concentration improves when data from MISR, MODIS, and chemical transport models are combined (van Donkelaar et al., 2010).

The present study used $PM_{2.5}$ data generated from both MODIS and MISR instruments to assess global mortality attributable to the total as well as the anthropogenic components of $PM_{2.5}$.

2. Materials and methods

2.1. Study design overview

This cross-sectional study estimated the global population fractions of adult mortality and expected number of deaths that can be attributed to chronic $PM_{2.5}$ exposure. Satellite imagery was used to derive $PM_{2.5}$ concentration estimates, and previously developed concentration–response (C–R) functions were used to calculate relative risks of associations between $PM_{2.5}$ and four causes of mortality. Risk coefficients for mortality from all causes, cardiopulmonary disease (ICD-9 400-440; 460-519), lung cancer (ICD-9 162), and ischemic heart disease (ICD-9 410-416) in relation to ambient $PM_{2.5}$ were based on the ACS cohort study. Ischemic heart disease (IHD), a subset of cardiopulmonary disease (CPD), was assessed as it is one of the chronic CPD causes of mortality most strongly associated with PM (U.S. EPA, 2009). Relative risks and mortality data were used to estimate the population fractions of mortality attributable to chronic $PM_{2.5}$ exposure.

Our methodology is based on the WHO's 2000 GBD assessment for urban air pollution (Cohen et al., 2004). The satellite-derived $PM_{2.5}$ measures were compared with estimates presented in the 2000 GBD and studies using similar methods.

2.2. Global assessment of mortality attributable to $PM_{2.5}$

2.2.1. Base case scenario

The following C–R function approximating chronic $PM_{2.5}$ exposure to risk of mortality was employed as the base case:

$$RR_{X-X_0} = \frac{\exp(\alpha + \beta X)}{\exp(\alpha + \beta X_0)} = \exp[\beta(X - X_0)] \qquad (1)$$

where RR_{X-X_0} is the relative risk at exposure 'X', compared to the reference exposure 'X_0'; β is the parameter estimate for the association between chronic $PM_{2.5}$ exposure and cause of mortality (Krewski et al., 2009); X is the mean $PM_{2.5}$ ambient concentration; and, X_0 is the reference exposure level for $PM_{2.5}$. While Eq. (1) is an exponential model, it will be referred to as a 'linear' since risk appears to be increasing linearly with $PM_{2.5}$ within the lower concentration ranges observed in this study. This was previously demonstrated in analyses of the ACS cohort, where the shape of the C–R function was found to be linear within the ranges of $PM_{2.5}$ observed (Krewski et al., 2000; Pope et al., 2002).

In 1982, the American Cancer Society (ACS) initiated a prospective cohort study and enrolled 1.2 million adults 30 years of age and older who were members of households with at least one individual 45 years of age or older. Participants completed a confidential questionnaire that included demographic, smoking history, alcohol use, diet, and education information. Associations between $PM_{2.5}$ and mortality have been reported for three follow-up periods—a 7-year follow-up (Pope et al., 1995), a 16-year follow-up (Pope et al., 2002), and an 18-year follow-up (Krewski et al., 2009). Risk coefficients in the current study were derived from relative risk estimates published in the most recent analysis of the ACS cohort study, where the confounding and modifying effects of ecological covariates on the air pollution–mortality association was examined at various scales with adjustment for 44 individual-level covariates as well as seven ecological covariates, and multiple-level spatial autocorrelation was assessed using a random effects Cox survival model (Krewski et al., 2009). For the base case scenario, the reference exposure level was set to 5.8 $\mu g/m^3$, the lowest concentration observed in 116 cities in the United States during the 1999–2000 $PM_{2.5}$ exposure collection period (Krewski et al., 2009). Thus, for the base case, it was assumed that 5.8 $\mu g/m^3$ is the theoretical minimum threshold concentration, below which $PM_{2.5}$ has no impact on mortality.

2.2.2. Derivation of fractions of mortality attributable to $PM_{2.5}$

The fraction of mortality attributable to $PM_{2.5}$ at the country-level (Ostro 2004) was derived as follows:

$$AF = \frac{RR_{X-X_0} - 1}{RR_{X-X_0}} \qquad (2)$$

The expected number of deaths attributable to $PM_{2.5}$ for each country was calculated by multiplying the number of country-level adult deaths due to a particular cause by the attributable fraction (AF) for that country. Subregional and global AFs and expected mortality estimates were derived from country-level relative risks (based on country-level $PM_{2.5}$ pollution) as well as country mortality inputs. Attributable numbers of country-level deaths were aggregated to the subregional and global levels and were divided by the total number of subregional and global deaths to obtain subregional and global AFs respectively.

2.2.3. Derivation of subregional relative risks

While country-level relative risk data were used to develop subregional and global AF and expected mortality estimates, subregional relative risks were calculated to enable regional comparison of risk associated with $PM_{2.5}$. Linear relative risks for outcome 'i' in subregion 'k' were determined by subtracting the reference exposure (X_0) from country-level mean weighted $PM_{2.5}$ concentrations (X_c), and then multiplying each country-level $X_c - X_0$ value by the proportion of the subregion exposed at that level (P_c), where P_c = (country 'c' population within subregion 'k')/(subregion 'k' population). Country-level $P_c * (X_c - X_0)$ values were then summed at the subregional level, giving a weighted subregional exposure average, termed C_k; in addition, C_k values were used in the linear risk function to obtain the subregional relative risk $RR_k = \exp(\beta_i * C_k)$.

2.3. Fine particulate matter ($PM_{2.5}$) concentrations

Detailed information on the satellite-derived $PM_{2.5}$ concentrations employed in this work has been published elsewhere (van Donkelaar et al., 2010). $PM_{2.5}$ concentrations were estimated by combining total-columnar aerosol optical depth (AOD) measures with the GEOS-Chem chemical transport model (www.geos-chem.org; Bey et al., 2001) to account for factors that affect the relationship between AOD and $PM_{2.5}$. Satellite-derived $PM_{2.5}$ concentrations were standardized to 35% relative humidity for consistency with ground-based monitors. GEOS-Chem simulations were used to determine the fraction of the $PM_{2.5}$ data from natural sources such as dust, and to a smaller extent, sea salt. Ambient $PM_{2.5}$ concentrations were produced in formats that included the dust and sea salt components, $PM_{2.5\_Total}$, as well as formats that excluded the natural dust components of $PM_{2.5}$, $PM_{2.5\_No\ Dust}$, based upon simulated aerosol speciation. Removal of the dust component of $PM_{2.5}$ resulted in concentrations that approximated the anthropogenic components of $PM_{2.5}$.

As described in van Donkelaar et al. (2010), a global $0.1° \times 0.1°$ map of ambient country-level $PM_{2.5}$ concentrations was developed based on a composite average of MODIS and MISR total columnar aerosol measurements taken during 2001–2006. To have been included, a given pixel must have had a total of at least 50 valid measurements over the 6-year observation period. The data were averaged within country boundaries and weighted according to population distributions using a $2.5' \times 2.5'$ global map of 2005 population density cells from the Socioeconomic Data and Applications Center (Gridded Population of the World version 3; http://sedac.ciesin.columbia.edu/). The $PM_{2.5}$ concentrations derived by these methods were shown to significantly agree with global ground based measurements, with spatial correlation coefficients ranging from 0.77 to 0.83 (van Donkelaar et al., 2010).

2.4. Mortality data

Country-level mortality estimates were required for the subregional and global attributable fraction calculations. Detailed country-level mortality data for adults aged 20 years and older was obtained from the WHO Mortality Database for 132 countries; data from 2005 or the next closest year of available data was obtained (Mortality, ICD-7-10; http://www.who.int/whosis/mort/download/en/index.html). Mortality data from the 2004 WHO GBD update, which is modeled for 192 countries, was used to calculate the expected numbers of deaths attributable to $PM_{2.5}$ (Mortality and morbidity; http://www.who.int/healthinfo/global_burden_disease/estimates_regional/en/index.html).

2.5. World Health Organization subregions

Data were structured to respect WHO subregional categories. In the WHO's GBD analyses, 192 member states are divided into five mortality strata: (A) very low child mortality, low adult mortality; (B) low child mortality, low adult mortality; (C) low child mortality, high adult mortality; (D) high child mortality, high adult mortality; and (E) high child mortality, very high adult mortality (WHO, 2004). These five strata are applied to the six WHO regions: Africa (AFR), Americas (AMR), South-East Asia (SEAR), Europe (EUR), Eastern Mediterranean (EMR), and Western Pacific (WPR).

3. Results

3.1. Fine particulate matter concentrations

Fig. 1 shows a global comparison of satellite derived $PM_{2.5\_Total}$ and $PM_{2.5\_No\ Dust}$ concentrations for 231 countries. Country-level geographic and population coverage of the $PM_{2.5}$ data from 132 countries used in the study was generally good, with median coverage of 97% and 99% respectively. Mean weighted $PM_{2.5\_Total}$ country concentrations ranged from 2.8 $\mu g/m^3$ to 51.5 $\mu g/m^3$, while the range of mean weighted $PM_{2.5\_No\ Dust}$ country concentrations was 1.0–47.4 $\mu g/m^3$. Table 1 reveals the estimated mean weighted subregional total and anthropogenic $PM_{2.5}$ concentrations along with country-level mortality estimates by WHO subregion.

3.2. Global assessment of mortality attributable to $PM_{2.5}$

Subregional relative risks for total $PM_{2.5}$ calculated under the base case scenario are presented in Table 2 for all four causes of mortality of interest. Countries in the Mediterranean regions (EMR) had $PM_{2.5}$ concentrations with large natural dust components. Subregions WPR-B, EMR-D, and EMR-B respectively had the highest relative risk estimates for all four causes of death for $PM_{2.5\_Total}$. However, once the natural dust component of $PM_{2.5}$ was removed, the relative risks for EMR-B and EMR-D became comparable to estimates for other subregions, while those of WPR-B remain large.

The global fraction of mortality attributable to $PM_{2.5\_Total}$ (95% CI) was 7.1% (4.1–9.8) for all causes, 12.1% (8.0–15.8) for cardiopulmonary disease, 16.8% (7.8–24.1) for lung cancer, and 17.5% (12.3–21.8) for ischemic heart disease, under the base case. The global fraction of mortality attributable to $PM_{2.5\_No\ Dust}$ was 4.5% (2.6–6.2) for all causes, 8.0% (5.3–10.5) for cardiopulmonary disease, 12.8% (5.9–18.5) for lung cancer, and 9.4% (6.6–11.8) for ischemic heart disease.
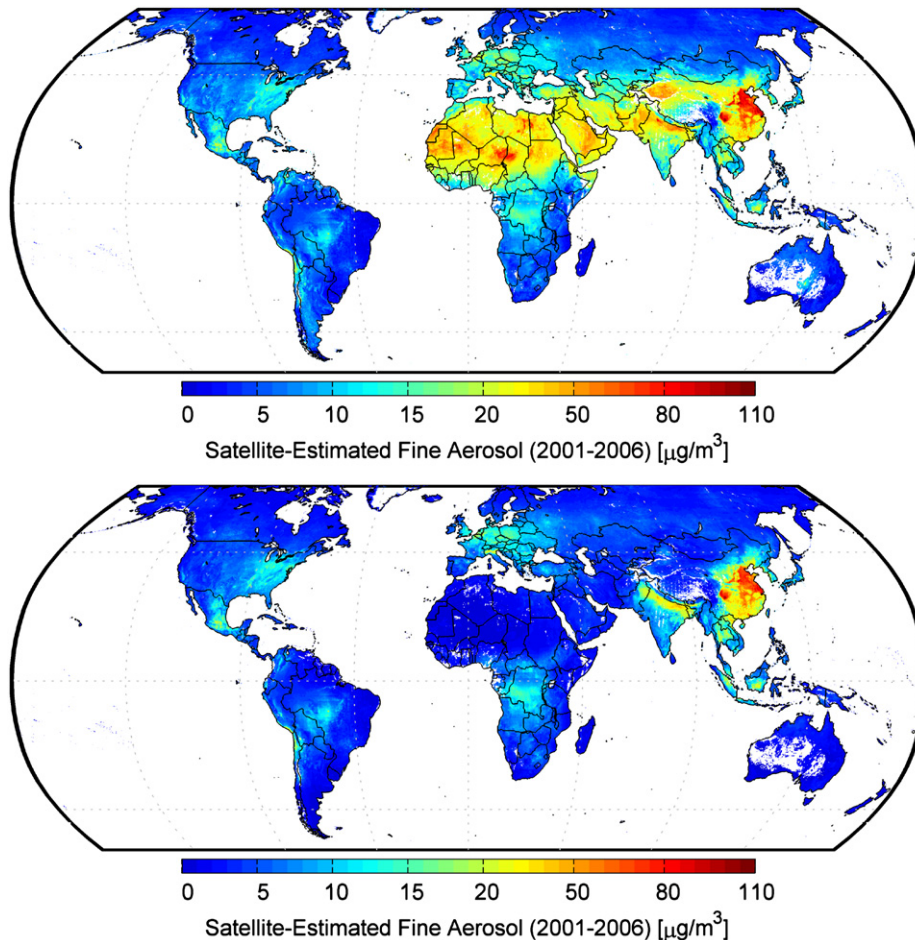
Fig.1. Global satellite-derived $PM_{2.5\_Total}$ (top) and $PM_{2.5\_No\ Dust}$ (bottom) measures averaged over 2001–2006. Data was standardized to 35% relative humidity, and adjusted for sampling to better represent a true annual sample (adapted from van Donkelaar et al., 2010).

Table 1
Satellite derived mean weighted PM$_{2.5}$ concentrations and all cause (AC), lung cancer (LC), cardiopulmonary disease (CPD) and ischemic heart disease (IHD) mortality data for 132 countries summarized by WHO subregion.

WHO region	WHO subregion	Number of countries represented	2005 population (millions)	PM$_{2.5\_Total}$ ($\mu g/m^3$)[a]	PM$_{2.5\_No\ Dust}$ ($\mu g/m^3$)[a]	Number of adults deaths (000s)[b]			
						AC	CPD	LC	IHD
Africa	AFR-D	5	2	7.0	1.3	17	7	[c]	2
	AFR-E	2	59	6.2	5.3	780	202	6	16
Americas	AMR-A	3	339	11.3	10.1	2762	1202	182	502
	AMR-B	41	462	7.9	5.9	2864	1035	53	280
	AMR-D	5	68	9.5	8.2	301	89	3	13
Eastern Mediterranean	EMR-B	5	96	24.4	4.7	128	31	1	6
	EMR-D	2	232	38.2	11.4	456	184	2	12
Europe	EUR-A	26	413	15.5	11.5	4016	1809	209	577
	EUR-B	16	223	16.4	8.8	1574	922	53	286
	EUR-C	9	235	10.3	8.1	3704	2241	88	1134
South-East Asia	SEAR-B	2	85	15.1	12.9	566	99	8	16
	SEAR-D	1	0.4	6.6	3.2	1	[c]	[c]	[c]
Western Pacific	WPR-A	5	155	14.8	11.4	1264	560	72	112
	WPR-B	10	1,450	47.3	41.0	1519	672	65	119
World		**132**	**3820**	**26.3**	**21.3**	**19,953**	**9051**	**742**	**3075**

[a] Mean subregional PM$_{2.5}$ levels were weighted to 2005 country populations.
[b] Data from 2005 or next closest available; adults ages ≥ 20 years (http://www.who.int/whosis/mort/download/en/index.html).
[c] Estimates < 1000.

Table 2
Relative risk of mortality associated with PM$_{2.5\_Total}$ under the base case scenario (95% confidence intervals).

WHO region	WHO subregion	All cause	Cardiopulmonary disease	Lung cancer	Ischemic heart disease
Africa	AFR-D	1.02 (1.01–1.04)	1.04 (1.02–1.06)	1.04 (1.02–1.07)	1.08 (1.05–1.12)
	AFR-E	1.01 (1.00–1.01)	1.01 (1.01–1.01)	1.01 (1.00–1.01)	1.02 (1.01–1.02)
Americas	AMR-A	1.04 (1.02–1.06)	1.07 (1.04–1.10)	1.08 (1.03–1.12)	1.15 (1.09–1.20)
	AMR-B	1.02 (1.01–1.03)	1.03 (1.02–1.04)	1.04 (1.01–1.06)	1.07 (1.04–1.09)
	AMR-D	1.03 (1.02–1.04)	1.05 (1.03–1.06)	1.05 (1.02–1.08)	1.10 (1.06–1.14)
Eastern Mediterranean	EMR-B	1.15 (1.08–1.23)	1.25 (1.15–1.37)	1.28 (1.11–1.48)	1.60 (1.36–1.89)
	EMR-D	1.28 (1.15–1.42)	1.48 (1.27–1.72)	1.54 (1.20–1.98)	2.27 (1.70–3.02)
Europe	EUR-A	1.08 (1.04–1.11)	1.12 (1.07–1.18)	1.14 (1.06–1.23)	1.28 (1.17–1.39)
	EUR-B	1.08 (1.05–1.12)	1.14 (1.08–1.19)	1.15 (1.06–1.25)	1.31 (1.19–1.43)
	EUR-C	1.03 (1.02–1.05)	1.06 (1.03–1.08)	1.06 (1.03–1.10)	1.12 (1.08–1.17)
South-East Asia	SEAR-B	1.07 (1.04–1.11)	1.12 (1.07–1.17)	1.13 (1.05–1.22)	1.26 (1.16–1.37)
	SEAR-D	1.01 (1.00–1.01)	1.01 (1.01–1.01)	1.01 (1.00–1.02)	1.02 (1.01–1.03)
Western Pacific	WPR-A	1.07 (1.04–1.11)	1.12 (1.07–1.17)	1.13 (1.05–1.22)	1.26 (1.16–1.37)
	WPR-B	1.37 (1.19–1.57)	1.65 (1.36–2.00)	1.73 (1.26–2.39)	2.85 (1.97–4.12)

The fractions of mortality attributable to PM$_{2.5\_No\ Dust}$ were 23–46% lower than those associated with PM$_{2.5\_Total}$. Eastern Mediterranean (EMR) and Western Pacific (WPR) subregions had the largest AFs of mortality associated with PM$_{2.5}$, while the African (AFR) subregions had the smallest. The mortality attributable to PM$_{2.5}$ for the EMR subregions was predominately related to the non-anthropogenic component of PM$_{2.5}$, while the AF of mortality associated with PM$_{2.5}$ in the WPR subregions was predominantly associated with the anthropogenic component. The WPR-B subregion, which includes several countries with large populations and high PM$_{2.5}$ pollution levels such as China, South Korea, and Taiwan, contributed greatly to global estimates of mortality associated with PM$_{2.5}$. Removal of this subregion from the analysis decreased base case global estimates of mortality by 50% for all cause mortality, 59% for cardiopulmonary disease mortality, 64% for lung cancer mortality, and 31% for ischemic heart disease mortality.

The numbers of adult deaths attributable to PM$_{2.5}$ in 2004 was over 3.3 million for all causes, 2.5 million for cardiopulmonary disease, 1.3 million for ischemic heart disease, and 222,000 for lung cancer, under the base case. Attributable mortality estimates decreased with removal of the natural dust component, with 2.1 million all cause, 1.6 million cardiopulmonary disease, 677,000 ischemic heart disease, and 170,000 lung cancer deaths estimated to be attributable to the PM$_{2.5\_No\ Dust}$.

3.3. Sensitivity analysis

The impact of three analytic choices – the shape of the C–R function as well as lower and upper threshold effects – was explored through sensitivity analyses. Table 3 summarizes global all cause, lung cancer, cardiopulmonary disease and ischemic heart disease deaths for adults attributable to PM$_{2.5}$ under all nine scenarios considered.

3.3.1. Concentration–response function

When extrapolating beyond the highest PM$_{2.5}$ concentrations observed in a cohort study, 30 $\mu g/m^3$ (Dockery et al., 1993; Krewski et al., 2009), it has been suggested that use of a linear function may be inappropriate since it could overestimate mortality; in this regard, the use of a log-linear exposure function has been recommended such that its slope would flatten at higher PM$_{2.5}$ concentrations (Ostro 2004).

Table 3
Attributable number of global adult all cause (AC), lung cancer (LC), cardiopulmonary disease (CPD) and ischemic heart disease (IHD) deaths (000s) in 2004 due to PM$_{2.5}$, under nine scenarios.

Sensitivity scenario	Fraction of PM$_{2.5}$	Shape of C–R curve	Reference exposure (µg/m^3)	Attributable deaths in 2004 (000s)[a] (95% confidence interval) {% change}[b]			
				AC	CPD	LC	IHD
Base case	Total	Linear[c]	5.8	3313 (1943–4593)	2477 (1631–3224)	222 (103–318)	1256 (887–1566)
	No dust	Linear[c]	5.8	2093 (1225–2907) {-37}	1645 (1081–2146) {-34}	170 (78–245) {-23}	677 (476–849) {-46}
Choice of C–R function	Total	Log-linear[d]	5.8	3591 (2278–4867) {8}	2551 (1738–3292) {3}	235 (128–328) {6}	1487 (1087–1825) {18}
	No dust	Log-linear[d]	5.8	2303 (1460–3124) {-30}	1694 (1156–2186) {-32}	186 (101–260) {-16}	840 (608–104) {-33}
Minimum exposure threshold	Total	Linear[c]	2.8	4220 (2470–5859) {27}	3080 (2024–4019) {24}	264 (123–378) {19}	1669 (1175–2087) {33}
	No dust	Linear[c]	1.0	3335 (1943–4652) {1}	2472 (1615–3244) {0}	234 (108–338) {5}	1254 (871–1590) {0}
	Total	Log-linear[d]	2.8	6333 (4045–8525) {91}	4319 (2974–5525) {74}	357 (199–490) {61}	2606 (1938–314)6 {107}
	No dust	Log-linear[d]	1.0	7390 (4748–9892) {123}	4947 (3433–6282) {100}	427 (242–577) {92}	2896 (2175–3468) {131}
Maximum exposure threshold (30 µg/m^3)	Total	Linear[c]	5.8	2640 (3722–5859) {-20}	1924 (1232–2569) {-22}	176 (78–262) {-21}	1123 (772–1431) {-11}

[a] Attributable deaths were calculated using modeled global mortality data from the 2004 WHO GBD update (Mortality and morbidity; http://www.who.int/healthinfo/global_burden_disease/estimates_regional/en/index.html).

[b] Percent change compared to the base case for PM$_{2.5\_Total}$.

[c] Linear risk coefficients (95% CI): AC—0.00751 (0.00421–0.01089); CPD—0.01205 (0.00742–0.01672); LC—0.01328 (0.00554–0.02103); IHD—0.02523 (0.01630–0.03415) (Krewski et al., 2009).

[d] Log-linear risk coefficients (95% CI): AC—0.12019 (0.07407–0.16776); CPD—0.18878 (0.12343–0.25385); LC—0.21136 (0.10807–0.31562); IHD—0.39392 (0.26995–0.51239) (Krewski et al., 2009).
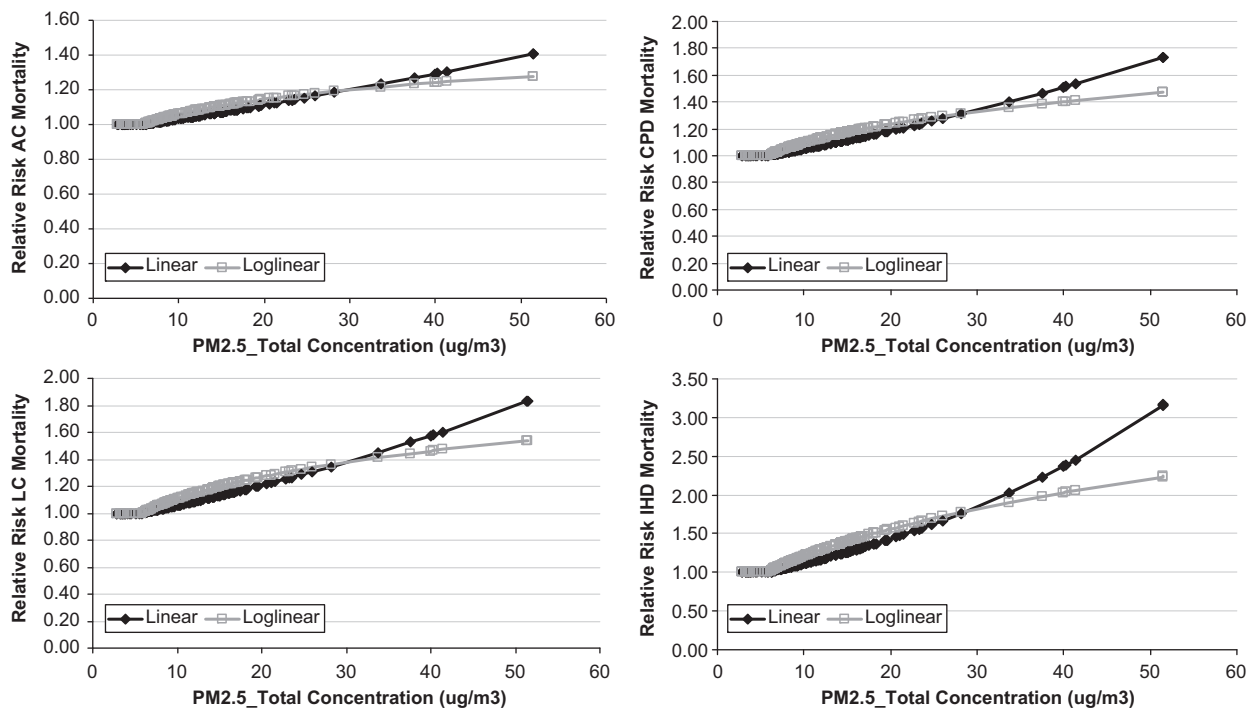
Fig. 2. Comparison of linear and log-linear concentration–response functions for the relative risk of all cause (AC), cardiopulmonary disease (CPD), lung cancer (LC), and ischemic heart disease (IHD) mortality associated with PM$_{2.5\_Total}$.

The following log-linear C–R function was explored:

$$RR_{X-X_0} = \frac{\exp[\alpha + \gamma \ln(X+1)]}{\exp[\alpha + \gamma \ln(X_0+1)]} = \left[\frac{(X+1)}{(X_0+1)}\right]^{\gamma} \quad (3)$$

where the parameter γ represents the log-linear association between PM$_{2.5}$ exposure and mortality and was based on ACS cohort (Krewski et al., 2009).

Log-linear models, compared to their linear counterparts, produced higher world attributable fraction estimates. A comparison of the linear and log-linear concentration–response functions

are shown for all four causes of death in Fig. 2. The log-linear C–R functions for PM$_{2.5\_Total}$ increased the attributable numbers of deaths across all four causes by 3–18% when the reference value was the same as the base case, and by 61–107% when the reference value was set to 2.8 µg/m^3.

3.3.2. Minimum exposure threshold

Since the base case reference value assumes a theoretical minimum threshold effect and ignores any potential health benefits that

may arise when reducing $PM_{2.5}$ concentrations below 5.8 $\mu g/m^3$, several sensitivity scenarios were explored where the reference value was set to the lowest levels observed for $PM_{2.5\_Total}$ and $PM_{2.5\_No\ Dust}$ concentrations, 2.8 $\mu g/m^3$ and 1.0 $\mu g/m^3$, respectively. This was explored for the total and anthropogenic components of $PM_{2.5}$, for both the linear and log-linear models.

Decreasing the theoretical minimum reference value from 5.8 $\mu g/m^3$ to 2.8 $\mu g/m^3$ for $PM_{2.5\_Total}$, and from 5.8 $\mu g/m^3$ to 1.0 $\mu g/m^3$ for $PM_{2.5\_No\ Dust}$ resulted in higher attributable fractions. Decreasing the reference value for $PM_{2.5\_Total}$ to 2.8 $\mu g/m^3$ produced increases of 19–33% in attributable mortality estimates.

3.3.3. Maximum exposure threshold

To assess the impact of extrapolating risk beyond 30 $\mu g/m^3$, the highest ambient $PM_{2.5}$ exposure level observed in both the ACS cohort (1979–1983) and the Harvard Six Cities study (1979–1985), a scenario was run where excess risk was constrained to be no greater than that associated with a concentration of 30 $\mu g/m^3$. Maximizing concentrations to 30 $\mu g/m^3$ resulted in an 11–22% decrease of in the expected number of global deaths due to $PM_{2.5}$. However, these decreases were only observed for three subregions, EMR-B/D and WPR-B, where country $PM_{2.5}$ concentrations of above 30 $\mu g/m^3$ were observed.

Overall, the range of attributable mortality was large across all nine scenarios. Attributable mortality estimates ranged from 2.1 to 7.4 million for all causes, 1.6–4.9 million for cardiopulmonary disease, 677,000–2.9 million for ischemic heart disease, and 170,000–427,000 for lung cancer. Subregions with the highest $PM_{2.5}$ concentrations were most affected in the sensitivity scenarios.

3.4. Uncertainty analysis

Paciorek and Liu (EHP, 2009) discuss the uncertainty in satellite-based $PM_{2.5}$ estimates. To quantify additional uncertainty related to $PM_{2.5}$ measurements, systematic error estimates were developed by combining sampling induced and retrieval-inherent uncertainties for each pixel and developing a population-weighted mean of those errors for each country (modified Figs. 6 and 7, van Donkelaar et al. 2010). The systematic error values were used to develop country-level upper and lower concentration estimates for the total fraction of $PM_{2.5}$. Table 4 shows the propagation of pollutant concentration ranges into uncertainty estimates associated with global attributable fractions due to systematic satellite measurement error under the base case. Uncertainty associated with risk coefficient variability and satellite measurement error were then imputed by combining upper

and lower limits for risk coefficients with upper and lower values for $PM_{2.5}$ concentrations in the linear concentration–response function (Table 4). The absolute magnitude of uncertainty associated with global AF mortality estimates increased by approximately 70% for all causes and cardiopulmonary disease, 40% for lung cancer, and 100% for ischemic heart disease when comparing uncertainty due to risk coefficient variability versus uncertainty due to both risk coefficient variability and systematic satellite measurement error.

3.5. Use of model parameters from the 2000 GBD for urban air pollution

The attributable fractions of mortality derived in our study were larger than those derived in the 2000 GBD for urban air pollution. Table 5 compares global and subregional AFs using the different input factors employed in this study, relative to the 2000 GBD analysis. When the risk coefficients as well as the reference and maximum exposures from the 2000 GBD for urban air pollution are applied to the $PM_{2.5}$ concentration estimates employed in the present analysis, AF estimates decrease by roughly 43% for lung cancer and 55% for cardiopulmonary disease (Table 5, column 2).

4. Discussion

This work represents one of the first comprehensive attempts to estimate global mortality attributable to measured PM, and specifically, that attributable to the anthropogenic component of $PM_{2.5}$. In comparison with previous studies aiming to estimate global mortality attributable to $PM_{2.5}$, which were limited to assessing the effects of urban air pollution due to the lack of monitors in rural regions (Cohen et al., 2004) or which were based on simulated $PM_{2.5}$ concentrations (Anenberg et al., 2010), the current assessment employed global $PM_{2.5}$ exposure measures from two satellite instruments, representing the most comprehensive observationally based $PM_{2.5}$ data currently available at a global scale.

4.1. Total versus anthropogenic components of $PM_{2.5}$

Total concentrations of $PM_{2.5}$ were modeled as the base case and served as the basis for sensitivity analysis for comparison purposes with other important work in this area, namely the WHO GBD for urban air pollution, thus allowing for commentary on the use of satellite imagery in such an analysis. However, the risk coefficients employed in this study more accurately represent the association between mortality and the anthropogenic versus

Table 4
Uncertainty ranges for global attributable fractions (%) of mortality associated with fine particulate matter under the base case.[a]

Cause of mortality	Uncertainty ranges for global attributable fractions (%)			
	Global attributable fraction (%)	Uncertainty due to variability in risk coefficient estimates[b]	Uncertainty due to systematic satellite measurement error[c]	Uncertainty due to variability in risk coefficients and systematic satellite measurement error
All cause	7.1	4.1 – 9.8	5.2 – 9.1	3.0 – 12.5
Cardiopulmonary disease	12.1	8.0 – 15.8	9.3 – 15.2	6.1 – 19.6
Lung cancer	6.8	7.8 – 24.1	13.5 – 20.1	6.2 – 28.5
Ischemic heart disease	17.5	12.3 – 21.8	12.4 – 22.6	8.7 – 28.0

[d] Uncertainty ranges were imputed by combining upper and lower limits for risk coefficients with upper and lower values for $PM_{2.5}$ concentrations (calculated based on systematic error) in the linear concentration–response function.

[a] AF estimates for the total fraction of $PM_{2.5}$ using a linear concentration–response function and reference $PM_{2.5}$ concentration of 5.8 $\mu g/m^3$.

[b] Risk coefficient variability is represented by the 95% confidence interval associated with the linear risk coefficient estimates (Krewski et al., 2009).

[c] Systematic satellite measurement error estimates were developed by combining sampling induced and retrieval-inherent uncertainties for each pixel; estimates were population weighted at the country-level (modified Figs. 6 and 7, van Donkelaar et al., 2010).

Table 5
Attributable fractions (AF) of lung cancer and cardiopulmonary disease mortality associated with $PM_{2.5}$ under various scenarios.

	AF lung cancer mortality			AF cardiopulmonary disease mortality		
	2000 GBD for urban air pollution[a]	Satellite PM_2.combined 2000 GBD model parameters[b]	Current analysis	2000 GBD for urban air pollution[a]	Satellite $PM_{2.5}$ combined with 2000 GBD model parameters[b]	Current analysis
World	**5.0**	**9.5**	**16.8**	**3.0**	**5.5**	**12.1**
AFR-D	5.0	0.4	0.8	2.0	0.5	1.2
AFR-E	2.0	0.0	1.1	1.0	0.0	1.0
AMR-A	3.0	2.9	7.0	2.0	2.2	6.4
AMR-B	4.0	0.9	2.5	3.0	0.7	2.3
AMR-D	6.0	1.9	5.3	3.0	1.2	4.3
EMR-B	4.0	13.4	23.2	3.0	10.0	21.3
EMR-D	8.0	18.7	31.0	4.0	14.0	28.6
EUR-A	2.0	6.2	12.2	1.0	4.5	11.0
EUR-B	5.0	6.3	12.4	3.0	4.8	11.6
EUR-C	3.0	2.3	6.0	2.0	1.6	5.3
SEAR-B	6.0	7.1	13.6	4.0	3.7	9.1
SEAR-D	4.0	0.0	1.1	3.0	0.0	1.0
WPR-A	4.0	6.2	11.9	3.0	4.5	10.8
WPR-B	10.0	20.3	33.3	6.0	14.1	28.4
Risk coefficients (per 1 $\mu g/m^3$)	0.00789[a]	0.00789[a]	0.01328[c]	0.00575[a]	0.00575[a]	0.01204[c]
Reference exposure ($\mu g/m^3$)	7.5	7.5	5.8	7.5	7.5	5.8
Maximum exposure ($\mu g/m^3$)	50.0	50.0	None	50.0	50.0	None

[a] AF estimates and risk coefficients (linear concentration–response functions) from Cohen et al. (2004) (*note*: confidence intervals not reported in this work).
[b] Estimates based on risk coefficients, reference exposure, and maximum exposure threshold from the 2000 GBD while using satellite derived $PM_{2.5\_Total}$ concentrations from the current study.
[c] Risk coefficients from Krewski et al. (2009) (linear concentration–response functions).

natural dust components of $PM_{2.5}$, such that the coefficients were based on $PM_{2.5}$ measures from urban areas in the United States where natural dust comprises only a small fraction of the total particle mass and is primarily derived from human activities. Thus, due to composition differences in $PM_{2.5}$ from anthropogenic versus natural sources the risk coefficients, the risk coefficients may not be representative of the risks associated with the natural dust component of $PM_{2.5}$. While the natural dust component of $PM_{2.5}$ is associated with health effects, pollution control policies would be intrinsically different for natural versus anthropogenic components of $PM_{2.5}$.

4.2. Sensitivity analyses

4.2.1. Concentration–response function

The log-linear model was introduced into the 2000 GBD for urban air pollution because it was believed to be a reasonable way to characterize excess risk at higher concentrations, such that the rate of increase, while still increasing monotonically with $PM_{2.5}$, decreases at higher concentrations (Cohen et al., 2004; Pope et al., 2002), as has been observed for daily mortality in time-series studies (Schwartz et al., 2002). The log-linear function was proposed as an alternative to the linear model when modeling health risk beyond the highest air pollution levels observed in epidemiological studies in which, up to those levels, risk was observed to increase approximately linearly with increasing $PM_{2.5}$ concentrations. In the 2000 GBD for urban air pollution, the burden estimates for sub-regions with low exposure levels were 63% higher in log-linear versus linear scenarios, while burden estimates for subregions with high exposures remained unchanged or were slightly reduced (Cohen et al., 2004). Similar results were found in this study, where AFs of mortality were substantially greater under the log-linear versus linear scenarios for subregions with relatively low concentrations of $PM_{2.5}$. However, global AFs of mortality were only slightly greater with the log-linear compared to linear models since those subregions that contributed most to global AF estimates were those with large populations and high pollution levels that had lower AFs under the log-linear scenario.

4.2.2. Minimum exposure threshold

Selection of the theoretical minimum reference exposure was a critical part of this assessment as previous estimates of mortality attributable to $PM_{2.5}$ have been sensitive to this variable (Cohen et al., 2004). As would be expected, decreasing the minimum exposure threshold from 5.8 $\mu g/m^3$, to 2.8 $\mu g/m^3$ for $PM_{2.5\_Total}$ and 1.0 $\mu g/m^3$ for $PM_{2.5\_No\ Dust}$ resulted in higher attributable fraction estimates. When modeling risk for concentrations below 5.8 $\mu g/m^3$, we assumed that the relationship between $PM_{2.5}$ and mortality was either linear or log-linear. However, given the limited evidence regarding the relationship between $PM_{2.5}$ risk of mortality at low $PM_{2.5}$ concentrations, it is difficult to comment on the accuracy of these increased AF estimates with lower reference values. The base case avoided extrapolating the C–R function below the concentrations observed in the epidemiological study from which the risk coefficients were derived.

4.2.3. Maximum exposure threshold

Extrapolation of risk beyond the highest $PM_{2.5}$ exposure levels observed in the ACS cohort and Harvard Six Cities study, 30 $\mu g/m^3$, involved supposition about the shape of the C-R function. The base case assumed that risk increases exponentially with ambient $PM_{2.5}$ concentrations for the entire range of country-level concentrations observed in this work. Overall, maximizing risk at 30 $\mu g/m^3$ decreased AF estimates for three subregions where country-level concentrations above this level were observed (EMR-B/D and WPR-B), resulting in moderate decreases in global AF estimates across all four causes of mortality.

4.3. Comparison with previous estimates of global mortality attributable to $PM_{2.5}$

Two key factors leading to differences between the current study and the 2000 GBD for urban air pollution are related to the risk coefficients and reference value used. The risk coefficients employed in our study were based on a more sophisticated model (a Cox random effects survival model with one level of clustering and adjustment for seven ecological covariates), which resulted in larger risk coefficients. The choice of the reference exposure also

contributed to the differences observed between the studies. In the base case, the 2000 GBD used the lowest observed concentration in the 1979–1983 observation period of the ACS, 7.5 $\mu g/m^3$, while the current study used the lowest concentration from the 1999–2000 period, 5.8 $\mu g/m^3$. When the risk coefficients and reference value from the base case assessment of the 2000 GBD for urban air pollution were applied to the current analysis, the AFs for lung cancer and cardiopulmonary disease decreased to 5.5% and 9.5% for cardiopulmonary disease and lung cancer respectively. While these estimates are greater than those derived in the previous GBD for urban air pollution, 3% and 5% for cardiopulmonary disease and lung cancer respectively (Cohen et al., 2004), the remaining differences in AFs of mortality between the current study and 2000 GBD may have arisen as a result of including rural ambient $PM_{2.5}$ pollution in the burden estimates.

In comparison to more recent estimates of global annual mortality attributable to anthropogenic $PM_{2.5}$ (Anenberg et al., 2010), the estimated numbers of deaths in 2004 attributable to anthropogenic $PM_{2.5}$ from our study were roughly 50% (1.6 versus 3.5 million) and 20% (170,000 versus 222,000) lower for cardiopulmonary disease and lung cancer respectively. Despite using different terms ('linear' in the current study versus 'log-linear' in the Anenberg et al. study), both studies appear to have employed the same exponential C–R function to derive relative risk estimates. Furthermore, both studies appear to have employed the same risk coefficients for the association between $PM_{2.5}$ and mortality, and both included rural areas in the assessment. One source of discrepancy between the two studies relates to assumptions regarding background concentrations. When Anenberg et al. employed the same background concentration that was assumed in our base case (5.8 $\mu g/m^3$), the attributable numbers of deaths in our study were approximately 35% lower for cardiopulmonary disease (1.6 versus 2.5 million), and were negligibly different for lung cancer (170,000 versus 164,000). The remaining differences observed for the number of cardiopulmonary deaths attributable to anthropogenic $PM_{2.5}$ are potentially due to differences in mean weighted anthropogenic $PM_{2.5}$ concentrations in relation to cardiopulmonary mortality rates at the country-level. The satellite derived $PM_{2.5}$ estimates differ considerably and tend to be higher than the simulated estimates based on the MOZART chemical transport model employed by Anenberg et al. Discrepancies in $PM_{2.5}$ estimates based on satellite measures compared to MOZART simulations could in part be due to the finer resolution of exposure data used in this study, whereby the resolution of $PM_{2.5}$ estimates based on MOZART simulation is $2.8° \times 2.8°$ versus $0.1° \times 0.1°$ for the satellite-based estimates. The studies differed as well in the handling of missing mortality data. Following observations that mortality rates can vary substantially within a given WHO subregion, the current study included only countries for which mortality data existed. In comparison, Anenberg et al. applied subregional estimates when country-level data was not available. Thus, the unique interaction between the distribution of $PM_{2.5}$ measures and mortality rates in each study may explain the differences in attributable mortality estimates between the studies. Ultimately, the estimates presented by both studies are associated with a large degree of uncertainty, such that when statistical uncertainty and the uncertainty associated with choice of model parameters are considered, the range of estimates presented by the two studies overlap.

4.4. Uncertainty

Estimating health effects from fine particle air pollution at the global scale is inherently associated with uncertainty. The choice of model parameters provided an array of global attributable fraction estimates that ranged from a half to three quarters below baseline estimates up to double baseline estimates, depending on the cause of mortality. Accounting for uncertainty associated with

systematic satellite measurement error as well as variability associated with risk coefficients increased the absolute uncertainty by 40–100%, as compared to the risk coefficient variability alone.

Additional uncertainty was associated with sampling variation in the concentration–response estimates from the ACS compared to other studies, represented by the variation in risk estimates among cohort studies assessing mortality and PM (U.S. EPA, 2009). Uncertainty is also inherent within country-level mortality rate estimates; however, the degree to which this uncertainty influenced our estimates for $PM_{2.5}$ cannot be completely quantified.

4.5. Study limitations and future work

The distribution of causes of mortality varies substantially throughout the world, and the risk coefficients for the association between mortality and $PM_{2.5}$ employed in this study were based on a cohort from the U.S. population. While the risk of all cause mortality due to $PM_{2.5}$ was found to be significant in the U.S., this association may primarily be the result of the association between cardiopulmonary disease mortality and $PM_{2.5}$, a large cause of mortality in the U.S. population. It is recommended that the AFs of all cause mortality associated with $PM_{2.5}$ at the global level be interpreted with caution.

The ACS cohort was a sample of individuals aged 30 years or older with relatively high socioeconomic status (Krewski et al., 2000). However, vulnerability to PM may vary by age, ethnicity, socioeconomic status (SES), risk behaviors such as smoking, and underlying disease status. For instance, education was shown to modify the effects of long-term PM exposure on mortality in the ACS cohort, such that individuals with lower educational attainment had higher risks of mortality (Krewski et al., 2000, 2009; Pope et al., 2004). The APHENA study also reported effect modification by SES-related variables and age on the association between acute PM exposure and mortality (Samoli et al., 2008). Although the relationship between acute PM exposure and mortality is relatively consistent across various countries and continents (Anderson et al., 2004; Samoli et al., 2008), if the effects of long-term PM exposure on mortality are more severe among populations with lower measures of SES, then these results may underestimate the magnitude of health burden from air pollution since a large portion of the world's population lives below poverty line. We were limited in our ability to undertake a comprehensive analysis of covariates influencing the association between $PM_{2.5}$ and mortality as the previously published risk coefficients for the association between $PM_{2.5}$ and mortality, as well as global mortality data were not available by detailed age, ethnicity, SES, lifestyle, and underlying disease strata. Prospective cohort studies on PM exposure and mortality data in developing countries are lacking, and research is required to better characterize global burden attributable to long-term PM exposure. Despite these limitations, the ACS cohort is one of the longest cohort studies on $PM_{2.5}$ and mortality, and it has undergone numerous sets of independent analysis, and is regarded as one of the best available sources of data on the risk of mortality associated with long term exposure to $PM_{2.5}$ (Industrial Economics Inc., 2006; Krewski, 2009).

A key contribution of future work would be the derivation of more refined $PM_{2.5}$ estimates. Despite improvements in remote sensory technology over the past decade, there is still a need for greater precision and accuracy in satellite measures and modeling (Hoff and Christopher, 2009). As such, sources for $PM_{2.5}$ concentration estimates should include a combination of ground monitors, satellite-based sensors, and advanced modeling techniques to supplement areas with poor satellite coverage, such monsoonal regions with persistent cloud. However, despite more refined satellite derived exposure estimates, the ecological nature of this work will likely continue to be a limitation, particularly at the global scale. Although the use of ecological measures of air pollution exposure

can be expected to underestimate risk (Mallick et al., 2002), the use of finer scales of geographic resolution has been shown to increase estimates of the mortality due to air pollution in Los Angeles (Jerrett et al., 2005), but not in New York (Krewski et al., 2009). Methods such as population weighting, while not equivalent to personal exposure estimates, can help to ensure that concentrations observed in more population dense regions are given more weight.

Aside from differentiating between natural and anthropogenic sources of PM$_{2.5}$ in an attempt to address differing toxicities with PM$_{2.5}$ composition, it was essentially assumed that all PM$_{2.5}$ components pose the same health risks. While it is acknowledged that this is a limitation of this work, unfortunately the epidemiological evidence is not sophisticated enough at this stage to look at risk coefficients specific to anthropogenic and natural dust components (U.S. NRC, 2009). Developing more refined risk estimates for the association between specific PM$_{2.5}$ components and health outcomes based on good cohort data is an important area to be addressed in future work.

Future research should also adjust for factors affecting the association between PM$_{2.5}$ and mortality that differ globally by region. This would involve the development of regional mortality data as well as risk coefficients stratified by socioeconomic covariates of interest. Furthermore, the associations between outdoor PM$_{2.5}$ exposure and mortality should be considered in conjunction with other related causes of mortality, such as smoking and indoor air pollution.

4.6. Study strengths

The PM$_{2.5}$ concentrations employed in this study were obtained from two different satellite instruments, MISR and MODIS, via methods that optimized the strengths of each sensor while minimizing limitations. It is generally acknowledged that combining measures from different sensors can reduce the uncertainty in column retrievals and ultimately PM$_{2.5}$ estimates (Hoff and Christopher, 2009). Global PM$_{2.5}$ estimates were found to have good correlation with ground-level measurements in North America and elsewhere (van Donkelaar et al., 2010)

Spatial autocorrelation due to the complex spatial patterns in the ACS data has been observed (Krewski et al., 2000). The Cox survival model assumes independence of individual survival times over space and time, an assumption that is not met if survival clusters at the ecological level. If longevity varies spatially, even after controlling for the appropriate risk factors, risk estimates and their variances could be biased. Our study addresses this concern to a certain extent through the use of risk coefficients derived from a random effects survival model applied to the ACS cohort (Krewski et al., 2009).

5. Conclusion

Previous studies of the health effects of air pollution have traditionally relied on data derived from ground-level monitors. Techniques for deriving PM$_{2.5}$ from remote sensing data have been evolving quickly, and satellite imagery technology is now at a stage where concentrations for several major pollutants, and specifically PM$_{2.5}$, can be determined with relative accuracy. Data from remote sensing technologies are quickly becoming useful in environmental health research. This study demonstrated the feasibility of using satellite derived pollution concentrations in the global assessment of mortality risk associated with exposure to fine particles. This approach leads to global estimates of mortality attributable to PM$_{2.5}$ that are greater than those based on fixed site measures of urban PM$_{2.5}$, but more similar to than estimates based on global simulations of anthropogenic PM$_{2.5}$,

depending on the cause of mortality assessed. Regardless of the uncertainty inherent with analyses of global mortality in association air pollution, fine particulate air pollution is an important contributor to worldwide variations in rates of mortality.

Competing financial interests

The authors declare they have no actual or potential competing financial interests.

Acknowledgments

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.envres.2012.08.005.

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