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Characterizing the burden of disease of particulate matter for life cycle impact assessment

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Abstract Fine particulate air pollution ($PM_{2.5}$) is a major environmental contributor to human burden of disease and therefore an important component of life cycle impact assessments. An accurate $PM_{2.5}$ characterization factor, i.e., the impact per kilogram of $PM_{2.5}$ emitted, is critical to estimating "cradle-to-grave" human health impacts of products and processes. We developed and assessed new characterization factors (disability-adjusted life years (DALY)/$kg_{PM2.5\ emitted}$), or the products of dose-response factors (deaths/$kg_{PM2.5\ inhaled}$), severity factors (DALY/death), and intake fractions ($kg_{PM2.5\ inhaled}/kg_{PM2.5\ emitted}$). In contrast to previous health burden estimates, we calculated age-specific concentration- and dose-response factors using baseline data, from 63 US metropolitan areas, consistent with the US study population used to derive the relative risk. We also calculated severity factors using 2010 Global Burden of Disease data. Multiplying the revised $PM_{2.5}$ dose responses, severity factors, and intake fractions yielded new $PM_{2.5}$ characterization factors that are higher than previous factors for primary $PM_{2.5}$ but lower for secondary $PM_{2.5}$ due to NO_x. Multiplying the concentration-response and severity factors by 2005 ambient $PM_{2.5}$ concentrations yielded an annual US burden of 2,000,000 DALY, slightly lower than previous US estimates. The annual US health burden estimated from PM emissions and characterization factors was 2.2 times higher.

Keywords Particulate matter · Life cycle impact assessment · Characterization factor · Burden of disease

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Introduction

Life cycle impact assessment (LCIA) evaluates the impact of a product or process from "cradle to grave"—from the extraction of the natural resources used to make the product to its disposal. A product or process usually generates particulate matter (PM) air pollution, either through the vehicular transport of the product or through the use of electricity from fossil-fueled power plants in the manufacture or use of the product. PM is classified as "primary" when it is emitted directly and "secondary" when it forms in the atmosphere due to secondary chemical reactions between other airborne substances. Whether primary or secondary, an accurate characterization factor for PM, defined as the disability-adjusted life years (DALY, or years of healthy life lost) per kilogram of particulate emitted, is critical to the analysis of health impacts in LCIAs.

Long-term epidemiologic cohort studies examining the association between PM and mortality in the US provide effect estimates preferable to those derived from animal studies for use in calculating a PM characterization factor. Many such studies, including the Harvard Six Cities Study (Laden et al. 2006) and the American Cancer Society Study of Particulate Air Pollution and Mortality (Pope et al. 2002), found increased mortality with increasing concentrations of PM (Hoek et al. 2013). These two studies in particular have informed the US Environmental Protection Agency's National Ambient Air Quality Standards for PM, which are intended to protect population health.

In the specific LCIA context, Hofstetter (1998) provided an initial set of characterization factors for the primary and secondary PM impacts per kilogram emitted, in terms of DALY.

The definition of a characterization factor was further formalized as the product of an intake fraction (Jolliet et al. 2003) multiplied by an effect factor that consists of a dose-response and severity factor, enabling the comparison of PM impacts with other organic and inorganic pollutants. Van Zelm et al. (2008) updated this framework, calculating a particulate characterization factor using results from epidemiologic studies of PM less than 10 μm in aerodynamic diameter (PM_{10})-associated health effects, some conducted in the US (Dockery et al. 1993; Pope et al. 1995), in conjunction with models of particulate exposure and demographic data from Europe.

Although that study significantly improved the quality of characterization factors used in LCIA, we have developed a new approach addressing three issues related to the key inputs to the characterization factor: the dose-response and severity factor (which together are basis of the effect factor), and the intake fraction. These three issues are:

1. Van Zelm et al.'s use of local European background mortality was not necessarily consistent with the US population background mortality from which the dose-responses were derived.
2. A new effect factor (the product of dose-response and severity factor) can be calculated using alternative methods of calculating the severity factor (DALY/death) that draw on the revised 2010 Global Burden of Disease Study (GBD) disease-specific DALY.
3. A set of new intake fractions, accounting for both the emissions source height and the "archetypal" emissions environment (urban, rural, or remote locations) and covering both primary and secondary particulates, calculated by Humbert et al. (2011), are now available.

This paper addresses these issues by developing updated components to the characterization factor, calculating a new characterization factor for PM, and assessing its performance, through the following five objectives:

1. Determine new $PM_{2.5}$ dose-response factors (deaths/kg $PM_{2.5}$ inhaled) by using age- and cause-of-death-specific results from Reanalysis of the American Cancer Society Study of Particulate Air Pollution and Mortality (the ACS Study (Pope et al. 2002)) and $PM_{2.5}$ concentrations in 63 US Standard Metropolitan Statistical Areas (SMSAs). Calculate the dose-response factors in terms of $PM_{2.5}$ (as opposed to PM_{10}), which is the fraction of PM_{10} with sufficient evidence to support a likely causal relationship with health endpoints (Humbert et al. 2011; U.S. Environmental Protection Agency 2010).
2. Calculate new severity factors based on 2010 GBD disease-specific DALY and total effect factors for PM-related effects, in term of years of life lost (YLL) and DALY.
3. Combine the new dose-response and severity factors with the new intake fractions (Humbert et al. 2011) to calculate and recommend $PM_{2.5}$ characterization factors that can be used for LCIA in different world regions.
4. Calculate the overall burden of disease attributable to PM in the US based on (a) ambient levels of $PM_{2.5}$ and (b) emissions of primary and secondary $PM_{2.5}$ using the revised intake fraction, and compare the results from these two methods to assess the new characterization factors.
5. Compare our estimate of the US burden of disease associated with PM to other estimates in the literature.

Materials and methods

Figure 1 provides an outline of our methods for estimating a characterization factor as well as the burden of disease for $PM_{2.5}$ both within and outside the LCIA framework. We first used the intake fractions ($kg_{inhaled}/kg_{emitted}$) estimated by Humbert et al. (2011) to assess exposures. Building on those fractions, we then estimated dose-response factors (cases/$kg_{inhaled}$) and severity factors (DALY/case), and multiplied these two quantities to estimate an age-adjusted effect factor (DALY/$kg_{inhaled}$). Next, we multiplied the effect factor with intake fractions to estimate the characterization factor (DALY/$kg_{emitted}$). Finally, we estimated a US $PM_{2.5}$ burden of disease based on these characterization factors and compared it to a $PM_{2.5}$ disease burden based on directly monitored $PM_{2.5}$ levels. The details of this process are provided below, and further details and derivations of the methods are provided in Appendix A.

Concentration- and dose-response factors

We calculated concentration–response factors (CRF, $PM_{2.5}$-associated annual mortality rate per $\mu g/m^3$ $PM_{2.5}$ inhaled) for mortality, for each age group and cause of death (cardiopulmonary disease, lung cancer, and all causes), as the

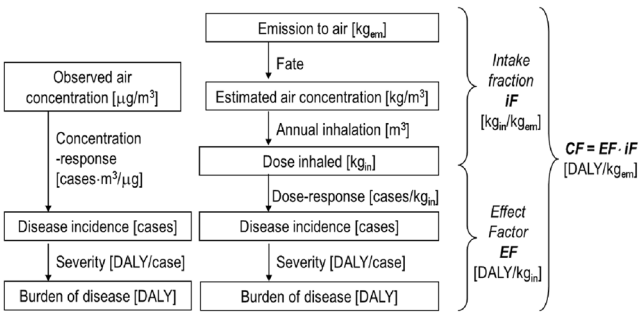

Fig. 1 Processes for estimating (1) a characterization factor (CF), (2) burden of disease based on observed air concentrations, and (3) burden of disease based on emissions for $PM_{2.5}$

population-weighted average of the CRF in each metropolitan area *i*:

$$\mathrm{CRF} = (\mathrm{RR}-1)\sum_{i=1}^{63\ \mathrm{SMSAs}}\left[\frac{\mathrm{MR}_{\mathrm{total},i}}{(\mathrm{RR}-1)\mathrm{C}_i+1}\cdot\frac{\mathrm{POP}_i}{\sum_{i=1}^{63\ \mathrm{SMSAs}}\mathrm{POP}_i}\right] \quad (1)$$

where $MR_{total,i}$ is the annual mortality rate for metropolitan area *i* in deaths/person/year, POP_i is the population size of metropolitan area *i* in persons, C_i is the $PM_{2.5}$ concentration in area *i*_ENREF_11, and *RR* is the increased risk of mortality per unit increase in C_i. We obtained RRs for four age groups (30 years and older, 30–59, 60–69, and 70 and older) (Appendix B Table 4) from the ACS Study (Pope et al. 2002). The ACS Study estimated the increased risk of death among adults 30 years of age and older due to all causes, cardiopulmonary diseases, and lung cancer associated with levels of ambient $PM_{2.5}$ in cities across the USA. We based our final characterization factor on the cardiopulmonary and lung cancer mortality results, rather than the all-cause results, because of their plausibility of association with $PM_{2.5}$, although we considered estimates using the all-causes RRs as well.

RRs are not very "portable" or generalizable from one population to another (Steenland and Armstrong 2006). Therefore, to provide the best estimate for our CRF, we used age-specific mortality rates from a population similar to that of the ACS study—a white, US population from the same time period as the ACS study (Intercensal Population Estimates by Age, Sex, and Race: 1980–1989 2009; National Center for Health Statistics 2010). Average $PM_{2.5}$ concentrations for 63 SMSAs from 1979 to 1983 were obtained from Appendix D of Part II of the ACS Study (Krewski et al. 2000). It is important to note that several other cohort studies relate mortality to $PM_{2.5}$ (Hoek et al. 2013). We chose to use the ACS study because of its large sample size, its broad distribution of cities across the USA, rigorous control for multiple confounders, and the availability of mortality rate and exposure data consistent with the study. Furthermore, in a meta-analysis of long-term air pollution exposure and cardiopulmonary mortality studies, the weighted mean of the RRs in the meta-analyses were similar to those of the individual ACS study *RRs* (Hoek et al. 2013), further validating our choice to use the ACS study RRs rather than RRs pooled across studies.

The dose-response factor ($PM_{2.5}$-associated deaths per kilogram of $PM_{2.5}$ inhaled) was then calculated as the CRF divided by the IH, where IH was the annual inhalation rate of an average individual, which was estimated at 4,745 m^3/person (U.S. Environmental Protection Agency 1997).

Severity factors and effect factors

Severity factors relate the cases of death attributed to PM, determined by the above-described dose-response, to the corresponding number of disability-adjusted life years (DALY) (Murray and Lopez 1996) and are expressed in terms of DALY/death. The severity factors for cardiopulmonary and lung cancer deaths were calculated from the Global Burden of Disease Study 2010, for the high-income North America region (Global Burden of Disease Collaborators 2013). We determined DALY/death and YLL/death based on the simplifying assumption that, e.g., the ratio of DALY/death for all causes (not just $PM_{2.5}$) of cardiopulmonary mortality is equivalent to the ratio of DALY/death for $PM_{2.5}$-associated cardiopulmonary mortality (Steenland and Armstrong 2006). For the all-cause severity factors, we used cardiopulmonary and lung cancer outcomes, assuming that these causes would more accurately reflect the severity of PM-associated disease.

Effect factors were then calculated within each age group as the product of the dose-response factors and the severity factors. The overall effect factor for 30 years and older was calculated as the population-weighted mean of the age-specific factors (assuming an effect factor of 0 for ages 0–29) using either the US population as weights or the WHO World Standard Population for 2000–2025 (http://www.who.int/whosis/indicators/compendium/2008/1mst/en/index.html). As a sensitivity analysis, these effect factors were compared to the effect factors we calculated using available dose-response relationships in the literature for specific morbidity outcomes associated with PM, although we were skeptical of the breadth and quality of these available dose-response relationships (see Appendix C for further discussion).

Characterization factors—impact per kilogram emitted

Intake fractions from Table 3 in Humbert et al. (2011) were combined with the newly calculated dose-response factor and severity factors (described in Sections 2.1 and 2.2) to calculate updated characterization factors for primary and secondary $PM_{2.5}$. The uncertainty around our characterization factors was then estimated as follows. Assuming uncertainties in intake fraction, dose-response, and severity factors were uncorrelated and assuming characterization factors have a log-normal distribution, the square of the geometric standard deviation (GSD^2) of the characterization factor was estimated as:

$$\mathrm{GSD}^2_{\mathrm{CF}} = e^{\sqrt{\left(\ln\mathrm{GSD}^2_{\mathrm{iF}}\right)^2+\left(\ln\mathrm{GSD}^2_{\mathrm{DR}}\right)^2+\left(\ln\mathrm{GSD}^2_{\mathrm{SF}}\right)^2}} \quad (2)$$

Our 95 % confidence intervals around our point estimates were then (estimate/GSD^2, estimate · GSD^2), and our 90 %

confidence intervals were (estimate/$GSD^{1.6}$, estimate · $GSD^{1.6}$).

Burden of disease—impact per year

Estimate using ambient concentrations

The county-specific $PM_{2.5}$ concentrations were obtained from the EPA's BenMAP 3.0 (Abt Associates Inc. 2008), and average annual population data for each county and age group (years 2005–2009) were obtained from the American Community Survey (U.S. Census Bureau 2010).

We calculated the burden of disease (DALY/year) in 2005 as the product of the concentration response factor (CRF, Eq. 1), the severity factor (SF), the population (POP), and the $PM_{2.5}$ concentration (C), summed over each combination of cause of death (d), county (i), and age group (a).

$$B_{\text{disease}} = \sum_{d=1}^{\text{cods}} \sum_{i=1}^{\text{counties}} \sum_{a=1}^{\text{ages}} \text{CRF}_{da} \times \text{SF}_{da} \times \text{POP}_{ai} \times \text{C}_i \quad (3)$$

This method differs from previous burden-of-disease estimates in that a constant baseline mortality rate is assumed in each county (for each age group and cause of death). An absolute increase in disease burden is then calculated using a concentration–response factor and population counts rather than multiplying the total mortality rate in that county by the attributable fraction. We therefore avoid misestimating the burden of disease as can occur when an attributable fraction is multiplied by a mortality rate that may be substantially elevated or diminished for reasons other than ambient particulate exposure, e.g., smoking. For comparison, we also estimated the burden of disease using an attributable fraction instead of a CRF:

$$B_{\text{disease}} = \sum_{d=1}^{\text{cods}} \sum_{i=1}^{\text{counties}} \sum_{a=1}^{\text{ages}} \left(1 - \frac{1}{e^{ln(\text{RR}_{ad})\cdot \text{C}_i}}\right) \cdot \text{MR}_{aid} \cdot \text{POP}_{ia} \quad (4)$$

where MR was the annual mortality rate based on 1980–1988 mortality and POP was the annual population from 2005 to 2009.

Estimate using emissions inventory

We obtained county-specific emissions of primary $PM_{2.5}$, SO_2, NO_x, and NH_3 from the EPA's 2005 National Emissions Inventory (U.S. Environmental Protection Agency 2005). US-specific characterization factors were then calculated using intake fractions from Table S1 and equations S11–S16 in Humbert et al. (2011).

In LCIA, where ambient pollutant concentrations resulting from a specific product or process are usually not known, the impact or total burden can be calculated by multiplying the characterization factor and the emission mass due to a functional unit of this product over its life cycle. Likewise, using characterization factors, the overall national burden of disease from PM due to all products and processes can be calculated by multiplying the emissions by the effect factor and the intake fraction (the characterization factor: $iF \cdot EF$), and summing over each combination of county (i), pollutant (j), and stack height (h).

$$B_{\text{disease}} = \sum_{i=1}^{\text{counties}} \sum_{j=1}^{\text{pollutants}} \sum_{h=1}^{\text{stack heights}} \left[\text{iF}_{hj} \cdot \text{EF}_i \cdot \text{M}_{\text{emitted }hji}\right] \quad (5)$$

where $M_{\text{emitted }hji}$ is the yearly mass of pollutant j emitted from stack height h in county i ($\text{kg}_{\text{emitted}}$ year^{-1}). For this calculation, an effect factor weighted by the US population in each age group was generated for each county.

The emission-based burden of disease of Eq. 5 can be directly compared to the concentration-based burden of disease of Eq. 3 to test its validity. Because we used identical effect factors (DALY per kg $PM_{2.5}$ inhaled) for each county, this method essentially compares the intake (kg $PM_{2.5}$ inhaled) calculated using emissions to the intake calculated using ambient concentrations.

Results

$PM_{2.5}$ dose-response factor

The population-weighted average of $PM_{2.5}$ concentration across the 63 SMSAs was 21.2 μg/m^3 (Table 1). Age-adjusted cardiopulmonary and lung cancer mortality rates ranged widely across SMSAs, from 580 to 870 deaths per 100,000 population. The mean age-adjusted attributable fraction for $PM_{2.5}$ ranged between 6.6 and 19 % for cardiopulmonary causes of mortality, with an average of 12 % among individuals aged 30 years and older. The attributable fraction was as high as 23 % for lung cancer mortality in the 60–69 age group (Appendix B Table 5), with an average of 8.6 % for 30 years and older (Table 1).

We estimated a combined dose-response factor of 4.2 deaths per kg $PM_{2.5}$ inhaled for cardiopulmonary and lung cancer mortality and an equivalent concentration-response factor of 2.0 deaths per 100,000 population per μg/m^3 $PM_{2.5}$ inhaled (Table 1). The concentration-response factor may be used when ambient concentrations are known.

Table 1 Characterization factor inputs: means and ranges (minimum, maximum) for $PM_{2.5}$ concentration; total, non-PM, and $PM_{2.5}$-attributable mortality rates; attributable fractions; concentration–response factors; dose–response factors; severity factors and effect factors standardized to the World Health Organization (WHO) World Standard Population

	Cardiopulmonary	Lung cancer	Cardiopulmonary and lung cancer	All causes
$PM_{2.5}$ concentration ($\mu g/m^3$), 1979–1983[a]	21.2 (10.3–37.8)			
Total annual mortality rates, 1982–1988[a,b,c]	640 (520–770)	82 (40–110)	720 (580–870)	1,200 (1,100–1,400)
Attributable fractions[a,b]	0.12 (0.064–0.19)	0.086 (0.042–0.15)	0.12 (0.063–0.19)	0.079 (0.041–0.13)
Non-PM mortality rates[a,b,c]	560 (480–660)	75 (37–100)	640 (540–740)	1,100 (980–1,300)
$PM_{2.5}$-attributable mortality rates[a,b,c]	78 (39–150)	7.1 (2.6–15)	86 (42–160)	96 (47–180)
Concentration-response factor (mortality rate per $\mu g/m^3$ $PM_{2.5}$ inhaled)[a,c,d]	1.8 (1.6–2.1)	0.17 (0.085–0.24)	2.0 (1.7–2.3)	2.2 (2.0–2.6)
Dose–response factor (deaths per kg $PM_{2.5}$ inhaled)[a,d]	3.9 (3.3–4.4)	0.35 (0.18–0.50)	4.2 (3.6–4.8)	4.7 (4.2–5.5)
Severity factors[e]				
DALY/death	17	28	19	23[f]
YLL/death	13	27	15	17[f]
Effect factors[d]				
DALY per kg $PM_{2.5}$ inhaled	65	9.7	78	110
YLL per kg $PM_{2.5}$ inhaled	50	9.6	64	82

[a] Weighted by the total population of the Standard Metropolitan Statistical Area (N=63)

[b] Among individuals aged 30 years and older

[c] Per 100,000 population

[d] Denominator is population among or quantity of $PM_{2.5}$ inhaled by all ages, not just individuals 30 and older

[e] Based on the Global Burden of Disease 2010 Estimates of Deaths, DALY and YLL for the High-Income North America region. YLD=DALY−YLL

[f] The age-specific severity factors for the "cardiopulmonary and lung cancer" category were used to calculate the all-cause severity factors. The all-causes severity factors differ from the "cardiopulmonary and lung cancer" severity factors in this table because of the difference in age distribution of all-cause deaths vs. cardiopulmonary and lung cancer deaths

See Appendix B Tables 5 and 6 for age-group specific results

$PM_{2.5}$ severity factors and total effect factors

For cardiopulmonary and lung cancer, the severity factor of 19 DALY is dominated by YLL as opposed to YLDs which account for only four additional DALY (Table 1).

Our default factor for LCIA of 78 DALY per kg $PM_{2.5}$ inhaled (Table 1), based on the total WHO World Standard Population, is lower than our US-specific effect factor (110 DALY per kg $PM_{2.5}$ inhaled) due to the fact that the WHO standard population is younger than the US population. Our WHO-population effect factor based on the RR for all-cause mortality is higher (110 DALY per kg $PM_{2.5}$ inhaled).

Combining effect and intake factors to determine characterization factors

Table 2 combines the effect factor of 78 DALY per kg $PM_{2.5}$ inhaled with the set of default intake fractions provided for various conditions by Humbert et al. (2011). Most life cycle inventories and LCIAs are still performed without knowledge of the source type and location of PM emissions. In these cases, a default, emission-weighted average characterization factor of 1.2E−03 DALY/kg primary $PM_{2.5}$ emitted would be used (gray cells in Table 3). When the type of emission source and its location are known for foreground processes (i.e., the processes directly evaluated in the LCIA), the characterization factor for the respective source and location should be used.

The uncertainty around our characterization factor estimates was great. The GSD^2 of the emission-weighted intake fraction has been evaluated to be 5.3 (Humbert et al. 2011). Given that the majority of the uncertainty in the dose response factor is due to the (RR−1) term in the numerator, the GSD^2 of the dose response factor was roughly estimated as 2.2 based on RR point estimates as low as 1.03 and as high as 1.13 per 10 $\mu g/m^3$ increase in $PM_{2.5}$ for all causes of mortality in sensitivity analyses of the ACS Study conducted by Krewski et al. (2009). The GSD^2 of the severity factor was qualitatively estimated as 1.4 given de Hollander et al.'s earlier estimate of 10 DALY per death (de Hollander et al. 1999) compared to our estimate of 19 DALY per death. According to Eq. 2, the world primary $PM_{2.5}$ emissions-weighted characterization factor of 1.2E−03 therefore has a GSD^2 of 6.5 and a 95 % CI of (1.8E−04, 7.6E−3).

Table 2 Characterization factors for primary $PM_{2.5}$ and secondary $PM_{2.5}$ (precursor pollutants are SO_2, NO_x, and NH_3) for the World Health Organization World Standard Population based on the cardiopulmonary and lung cancer effect factor

Emission source	Characterization factor for the respective location of emission:				Unit
	Urban	Rural	Remote	Population-weighted average	
Primary $PM_{2.5}$					
High-stack	8.6E−04	1.2E−04	7.8E−06	5.3E−04	DALY/kg $PM_{2.5}$ emitted
Low-stack	1.2E−03	1.6E−04	7.8E−06	6.9E−04	
Ground-level	3.4E−03	3.0E−04	7.8E−06	2.0E−03	
Emission-weighted average	2.0E−03	2.0E−04	7.8E−06	1.2E−03	
SO_2	7.7E−05	6.2E−05	3.9E−06	6.9E−05	DALY/kg SO_2 emitted
NO_x	1.6E−05	1.3E−05	7.8E−07	1.4E−05	DALY/kg NO_x emitted
NH_3	1.3E−04	1.3E−04	7.8E−06	1.3E−04	DALY/kg NH_3 emitted

See Appendix B Table 7 for world-region-specific primary $PM_{2.5}$ characterization factors

Contribution of $PM_{2.5}$ to annual US burden of disease

Human health damage based on annual US ambient PM concentrations

Comparison with Global Burden of Disease US estimates and EPA estimates We estimated a PM burden of disease for the entire US in 2005 of 130,000 deaths and 2 million DALY, without considering any minimum threshold concentration, and we compared our burden of disease estimates to those of the GBD and the US EPA for two different minimum thresholds of $PM_{2.5}$ (Table 3). We used methods similar to those used by the US EPA in estimating the US burden of disease—RRs from the ACS Study and county-level air quality inputs. When using attributable fractions (as the EPA did) in conjunction with background mortality from an earlier time period (1982–1988), we estimated a higher burden of disease of 150,000 deaths compared to the 110,000 annual deaths estimated by the EPA. Estimates based on cardiopulmonary and lung cancer mortality were almost identical to estimates based on all-cause mortality. When we used our age-specific concentration-response functions instead of attributable fractions, our estimate dropped to 130,000 deaths annually.

Above a background concentration of 4 $\mu g/m^3$, our estimates of 52,000 deaths and 960,000 DALY, adjusted to the World Health Organization Standard Population, were much lower than the Global Burden of Disease US estimates of 103,000 deaths and 1,800,000 DALY.

Comparison of our estimate of $PM_{2.5}$ effects on life expectancy to Pope et al.(2009) Based on a regression of national mortality statistics and PM concentrations for 51 US metropolitan areas, Pope et al. (2009) have estimated the increase in life expectancy for each 10 $\mu g/m^3$ decrease in $PM_{2.5}$ to be 0.61 (95 % CI, 0.22–1.0) years. For adults over 30 years of age, we estimated 0.00037 deaths (all-cause) per person per 10 $\mu g/m^3$ inhaled per year (concentration-response factor adjusted to the US 2000 population) and a severity factor of 17 YLL per death (Table 1). Multiplying these two factors by a healthy life expectancy of an additional 52 years after age 30 per person (Mathers et al. 2006b), we estimate an increase in life expectancy of 0.33 years per person for each 10 $\mu g/m^3$ decrease in $PM_{2.5}$. Our result falls within Pope et al.'s 95 % confidence interval.

Human health damage based on annual US primary and secondary pollutant emissions and characterization factors

Using 2005 US emissions and urban and rural characterization factors, we estimated the annual intake of $PM_{2.5}$ as 38,000 kg $PM_{2.5}$, which was 2.2 times higher than the estimate based on actual ambient concentrations. Figure 2 plots the logarithm of the $PM_{2.5}$ intake estimated using ambient concentrations vs. the intake estimated using the emissions inventories and characterization factors in order to examine the concordance between the intake and burden of disease calculations by county. We would not expect the two values to match for each county because the "emissions" intake is based on the intake due to the county emissions without regard to the location of the affected population (not just the population within that county). The S-shaped scatterplot reflects this, where the emissions-based intake is lower than the ambient concentration-based intake in counties with low emissions and higher than the ambient concentration based intake in counties with high emissions. Nevertheless, the two estimates are within a factor of 10 for 91 % of the counties, with closer agreement among the counties with a population density less than the median of 15 persons per km^2 (for the log-transformed values, $t=-24$, $p<0.0001$).

Table 3 Comparison of US PM burden of disease estimates

Estimate	Mortality rates	Age grouping	Population for age adjustment	Method[a]	Health outcomes considered	Background $PM_{2.5}$ level	PM-associated deaths (in thousands)	PM-associated DALY (in thousands)
GBD 2010 (US Burden of Disease Collaborators 2013)	2010 National Estimates	25+ in 5-year groups	WHO Standard	AF · MR · POP	Mortality due to COPD, IHD, LRIs (ages 0–4), lung cancer, and stroke[b]	4 μg/m^3	103 (95 % UI, 84.7–122.4)	1,820.4 (95 % UI, 1,552.6–2,111.1) [c]
Ours	1982–1988	30+ in 5-year groups	WHO Standard	CRF · PM · POP	Cardiopulmonary and lung cancer mortality	4 μg/m^3	52 (90 % CI, 27–98) [c]	960 (90 % CI, 500–1,800)
US EPA 2010 (U.S. EPA 2010)	1996–1998	30+	USA 2000	AF · MR · POP	All-cause mortality	5.8 μg/m^3	63 (90 % CI, 39–87)[d,e]	
ours	1982–1988	30+ in 5-year groups	U.S. 2000	CRF · PM · POP	Cardiopulmonary and lung cancer mortality	5.8 μg/m^3	71 (90 % CI, 37–140)[c]	
US EPA 2010 (U.S. EPA 2010)	1996–1998	30+	USA 2000	AF · MR · POP	All-cause mortality	Policy Relevant Background	110 (90 % CI, 68–150)[d,e]	
Ours	1982–1988	30+ in 5-year groups	USA 2000	AF · MR · POP	All-cause mortality	0 μg/m^3	150 (90 % UI, 78–280)	
Ours	1982–1988	30+ in 5-year groups	USA 2000	AF · MR · POP	Cardiopulmonary and lung cancer mortality	0 μg/m^3	150 (90 % UI, 77–280)	
Ours	1982–1988	30+ in 5-year groups	USA 2000	CRF · PM · POP	Cardiopulmonary and lung cancer mortality	0 μg/m^3	130 (90 % UI, 69–250) [c]	2,000 (90 % UI, 1,000–3,800) [c]

[a] *AF* attributable fraction, *MR* mortality rate, *CRF* concentration–response factor, *PM* $PM_{2.5}$ concentration, *POP* population

[b] *COPD* chronic obstructive pulmonary disease, *IHD* ischemic heart disease, *LRI* lower respiratory infection

[c] In calculating 90 % uncertainty intervals (UIs), we estimate a $GSD^{1.6}$ of 1.9 for the (RR−1) term we use (see Section 2.3)

[d] Based on RR for all-cause mortality of 1.044 per 10 μg/m^3 increase in $PM_{2.5}$ from ACS Study Table 33 (Krewski et al. 2009)

[e] County $PM_{2.5}$ concentrations modeled as a fusion of 2005 monitored $PM_{2.5}$ concentrations with 2005 CMAQ-modeled air quality levels

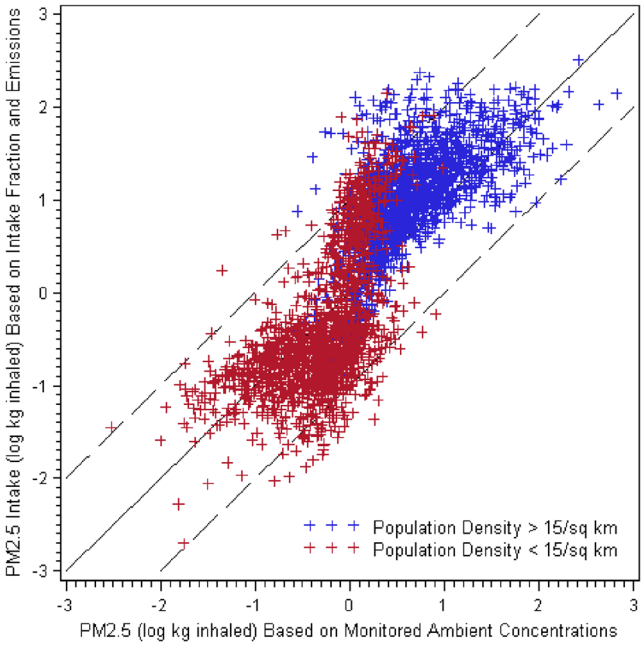

Fig. 2 Intake of $PM_{2.5}$ (kg) based on emissions vs. intake based on ambient concentrations for each county plotted on a log scale to visualize the associations in the counties with lower burdens. The *solid line* is the 1:1 line, and the *dotted lines* are 10:1 lines

Discussion

We present here an updated methodology to characterize the disease burden associated with particulate air pollution exposure for application in LCIA that addresses several issues in previous work. The newly calculated characterization factors are based on age- and cause-of-death-specific data and are based on the US population background mortality from which the $PM_{2.5}$ dose-responses were derived as well as the revised disease-specific severity factors from the 2010 GBD. By combining our updated effect factors with the new intake fractions of Humbert et al. (2011), we calculated revised impact per kilogram of primary and secondary particulate emitted for a default region as well as for specific world regions. Below, we compare our dose-response factor, effect factor, severity factor, and characterization factor calculations as well as our burden of disease calculations to previous calculations.

Assuming that $PM_{2.5}$ is approximately 1.67 times more toxic than PM_{10} (European Commission 2005), our dose-response factor is 27 % lower than the PM dose-response factor calculated by Van Zelm et al. (2008) who estimated a dose-response factor of 5.76 deaths per kg PM_{10} inhaled (Appendix C Table 8). Consequently, in term of YLL, the estimate of 64 YLL per kg $PM_{2.5}$ inhaled we obtained is also 33 % lower than the 96 YLL due to $PM_{2.5}$, obtained by Van Zelm et al. (2008). However, our estimate of morbidity due to $PM_{2.5}$ of 14 YLDs per kg $PM_{2.5}$ inhaled (Table 1) is much higher than that estimated by Van Zelm et al. of 0.055 YLDs per kg $PM_{2.5}$ inhaled (Appendix C Table 8). Therefore, our resulting effect factor of 78 DALY per kg $PM_{2.5}$ inhaled is only 19 % lower than the Van Zelm et al. effect factor of 96 DALY per kg $PM_{2.5}$ inhaled (after converting from PM_{10} to $PM_{2.5}$, Appendix C Table 8).

Our severity factors are higher than a previous estimate by De Hollander et al., who calculated the environmental burden of disease in the Netherlands and estimated weight factors for various causes of morbidity attributed to PM (de Hollander et al. 1999). De Hollander et al. estimated 10.1 YLL per death. The YLL severity factor in the present study is higher (15 YLL/death). The additional 5 YLDs/death in the present DALY severity factor represent morbidity due to cardiopulmonary diseases including chronic bronchitis, which accounts for 37 % of the cardiopulmonary YLDs in the high income North America region (Global Burden of Disease Collaborators 2013). Nevertheless, our severity factor remains dominated by death, yielding an overall factor of 19 DALY per death due to $PM_{2.5}$.

For primary $PM_{2.5}$, our characterization factor of 1.2E−03 (Table 2) is 2.7 times higher than Van Zelm's factor of 4.3E−4, as converted from the PM_{10} estimates accounting for the respective proportion of PM_{10} emitted as $PM_{2.5}$ for the primary and secondary PM (Humbert et al. 2011). This results from higher intake fractions (3.1 times higher) multiplied by lower effect factors (27 % lower as discussed above), obtained using US mortality rate inputs, age-group specific RRs, and chronic morbidity due to $PM_{2.5}$. Our characterization factors for secondary $PM_{2.5}$ due to SO_2 and NH_3 of 6.9E−5 and 1.3E−4 are virtually the same as Van Zelm's factors of 6.9E−5 and 1.4E−4, while our characterization factor for secondary $PM_{2.5}$ of 1.4E−5 due to NO_x is lower than Van Zelm's factor of 6.2E−5 due to our use of a lower intake fraction.

The factors suggested in this paper update the factors provided by Humbert (2009) which are currently recommended by the European Commission in its Production and Organization Environmental Footprint methodologies (Wolf et al. 2012). The factors suggested in this paper are about a third lower than the factors provided by Humbert (2009) for primary $PM_{2.5}$ and secondary PM from SO_2 and are about the same for secondary PM from NO_x and NH_3. The correspondence between our revised characterization factors and previous ones suggests that conclusions used in previous LCIA using those methods would still be largely valid.

Our estimate based on ambient concentrations is 2.2 times lower than our estimate based on $PM_{2.5}$ emissions inventories and characterization factors in the US, and this difference is due to the additional assumptions about intake required when using emissions inventories and characterization factors instead of known ambient concentrations. In county-by-county comparisons, the two intake estimates differed by less than a factor of 10 for 91 % of the counties. The intake fraction is heavily dependent on assumptions about the density of the

population near the emissions source, so it was not surprising that the uncertainty in the intake fraction is larger for counties with higher population densities. The general concordance of the emissions and characterization factor-derived results with the ambient concentration results supports the use of these characterization factors for LCIA.

Our US burden of disease estimates based on ambient levels of $PM_{2.5}$ fall in the range the estimates derived by the US EPA in 2010. When using a CRF, which provides an estimate of risk on an absolute scale, as opposed to the direct use of an attributable fraction for each county which estimates risk relative to the baseline mortality in that county, our results did not change substantially. This is not surprising given that we made this comparison using a population similar to that used to derive the RRs. Pope et al.'s (2009) life expectancy estimate was also similar to ours, which again, is not surprising considering that a similar study population was used. Our results reinforce that US health-based air quality standards that are based on previous burden of disease and life expectancy estimates are robust to the methodologic differences we addressed here. The advantage of using a CRF is that it produces results for other countries and regions that are less dependent on background mortality, and this method is more relevant to instances when epidemiologic evidence from one population is applied to another, as in LCIA.

Our burden of disease estimate falls below the GBD estimate, likely due to the higher RR*s* used in the GBD, which were modeled from multiple cohort studies of ambient air pollution as well as secondhand tobacco smoke, indoor solid cooking fuel and active smoking (US Burden of Disease Collaborators 2013). The RRs in the GBD modeling were allowed to vary by exposure concentration (Burnett et al. 2014), although any possible biological mechanisms explaining the resulting non-linear dose-response relationships were not proposed. For the purposes of LCIA a linear, no-lower-threshold dose-response curve is often assumed. Although the health effects of $PM_{2.5}$ may actually be attenuated in countries with higher background $PM_{2.5}$ levels (Ostro 2004), a linearity assumption may still be appropriate in LCIA applications in which one would not want an inter-regional comparison of the additional adverse health effects due to a process to be influenced by the background $PM_{2.5}$ levels in those regions. On the other hand, in indoor settings, our characterization factor would possibly overestimate PM-attributable health effects considering much higher concentrations of PM are often observed indoors, and more research is needed to quantify the health effects of indoor PM (Smith and Peel 2010). Even solely among studies of mortality and ambient air pollution, considerable heterogeneity in the effect estimates exists (Hoek et al. 2013), and some of this heterogeneity may be due to differences in baseline mortality risk from study to study. Additional research needs in refining estimates of PM-related health effects as well as limitations of the present study are listed below.

PM regulations and epidemiology studies typically focus on PM mass. If and when PM number, surface area and composition are shown to be important and robustly quantifiable in dose-response relationships, it will be necessary to reevaluate results presented here.

The ACS Study did not control for other air pollutants or noise pollution. There is a demand for improved health effects modeling of multiple, correlated air pollutants simultaneously, and techniques for addressing this challenge are being developed (Dominici et al. 2010). In the meantime, the effects of PM reported in this paper should be applied cautiously considering that some of the effect attributed to PM may be due to other air pollutants. In LCIA, because PM health effects are often much higher than the effects of other air pollutants, the PM health effects serve as a proxy for the environmental health effects of air pollutants generated by that product or process.

Our simplified severity factor calculation attempts to avoid the gaps in knowledge regarding PM-associated morbidity. However, Appendix C Table 8 suggests that our severity factor could still be underestimating PM-associated morbidity due to chronic bronchitis. Also, the effect factor recommended here uses only dose-response information based on adults. The influence of PM inhalation on low birth weight (Bell et al. 2008) and asthma among children and expressing this influence in terms of DALY also deserves further attention.

We have updated the PM characterization factor so that future LCIAs may have more precise comparisons of the health burden of PM from various products or processes. However, our uncertainty intervals are still relatively wide, and advances in modeling the intake fraction are needed. For both LCIA and estimates of the burden of disease due to PM, a better understanding of the mechanisms behind a potentially non-linear dose-response relationship and sources of heterogeneity in effect estimates is needed. Nevertheless, the $PM_{2.5}$-attributable fractions for cardiopulmonary disease as well as lung cancer were high (9–12 % on average) indicating that $PM_{2.5}$ represents an important exposure contributing to mortality in the US and is fundamental to our understanding of the health impacts of PM throughout a product or process's life cycle.

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Appendix A: Additional materials and methods

Concentration- and dose-response factors

Data

The RRs in the ACS Study (Pope et al. 2002) accounted for confounding by several individual risk factors (age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure, and diet) and spatial autocorrelation. The ACS Study evaluated differences in mortality associated with chronic (multi-year) $PM_{2.5}$ exposure, but some of the short-term effects of $PM_{2.5}$ are likely captured in this study type. Although time series studies of mortality and morbidity associated with only short-term exposure to $PM_{2.5}$ have been conducted, characterization factors based on these have been estimated to be two to four orders of magnitude lower than the characterization factor for mortality due to chronic exposure in past calculations (van Zelm et al. 2008). Therefore, short-term effects are not addressed separately here.

To elaborate on the point that RRs are not very "portable" or generalizable from one population to another (Steenland and Armstrong 2006), RRs estimate health effects *relative to* the baseline levels of that health effect. For example, a country may have a higher rate of mortality than the US among individuals aged 55–59, due to causes other than outdoor pollutants, such as tobacco smoke. Therefore, the fraction of deaths attributable to PM would be overestimated in that country if a US-based study were used for the calculation. Thus, in estimating *absolute* increases in a particular health effect per unit of pollutant, it is better to be consistent between the study population used to derive the RR and the corresponding health effect data. Because the RRs were derived from the ACS Study cohort, to calculate a $PM_{2.5}$-attributable fraction, we obtained mortality and population data for US SMSAs by age group. We used this US-based data because the distribution of population factors that may modify the association between $PM_{2.5}$ and mortality, such as tobacco smoking, would be more likely comparable with those in the ACS study population than data from another country. Also, because the ACS Study population was 94 % white, dose-response factors were calculated using mortality rates among whites only to ensure consistency.

US mortality data were obtained from the US Centers for Disease Control and Prevention's National Center for Health Statistics (National Center for Health Statistics 2010). After 1988, one-year-age-specific mortality data was made available only for counties and cities with populations greater than 100,000 persons (Data Release Policy, http://www.cdc.gov/nchs/nvss/dvs_data_release.htm), so counts of deaths by five-year age group and cause of death for each SMSA were calculated for the years 1982–1988, within the follow-up period for the ACS Study (1982–1998). The ACS Study only enrolled individuals aged 30 years and older, so only mortality data for decedents 30 years and older were considered. Annual mortality rates for each cause of death were calculated by dividing the deaths by annual population estimates for each age group, which were obtained from the US Census (Intercensal Population Estimates by Age, Sex, and Race: 1980–1989 2009). The seven years were then averaged by five-year age group, cause of death and SMSA.

Calculation

The concentration–response factors (CRF, $PM_{2.5}$-associated annual mortality rate per $\mu g/m^3$ $PM_{2.5}$ inhaled) for mortality, for each cause of death (cardiopulmonary disease, lung cancer, and all causes) and age group, were defined as the population-weighted increase in mortality rate attributed to $PM_{2.5}$ in the US SMSAs divided by the average $PM_{2.5}$ concentration:

$$\mathrm{CRF}_i = \frac{\mathrm{MR}_{\mathrm{PM2.5},i}}{\mathrm{C}_i} \cdot 10^9 \tag{6}$$

where $MR_{PM2.5,i}$ is the $PM_{2.5}$-associated annual mortality rate for metropolitan area i in deaths/person/year, and C_i is the $PM_{2.5}$ concentration (in $\mu g/m^3 = 10^{-9}$ kg/m^3) in area i.

From Cox proportional hazards models (and other log-linear models commonly used in epidemiology studies), the *RR* (unitless) for each unit increase in $PM_{2.5}$ concentration (C in $\mu g/m^3$) is equivalent to e^{β}, where β is the increase in ln(deaths) per 1 $\mu g/m^3$ increase in $PM_{2.5}$. Considering that in the range of applicable $PM_{2.5}$ concentrations and RRs in the US, the association between mortality and PM is approximately linear, the attributable fraction for metropolitan area i, or the proportion of total cases attributable to $PM_{2.5}$ in that metropolitan area, is

$$\mathrm{AF}_{\mathrm{PM2.5},i} = \frac{\mathrm{MR}_{\mathrm{PM2.5},i}}{\mathrm{MR}_{\mathrm{total},i}} = 1 - \frac{1}{e^{\beta \mathrm{C}_i}} \approx \frac{(RR-1)\mathrm{C}_i}{(RR-1)\mathrm{C}_i + 1} \tag{7}$$

The concentration-response factor for metropolitan area i therefore becomes

$$\mathrm{CRF}_i = \frac{MR_{\mathrm{total},i} \cdot \mathrm{AF}_{\mathrm{PM2.5},i}}{\mathrm{C}_i} \cdot 10^9 = \frac{\mathrm{MR}_{\mathrm{total},i} \cdot (\mathrm{RR}-1)}{(\mathrm{RR}-1)\mathrm{C}_i + 1} \cdot 10^9 \quad (8)$$

$\mathrm{PM}_{2.5}$ concentration and mortality rate vary by location within the US, but the RRs presented in the ACS Study were not specific to any one metropolitan area. Therefore, the recommended concentration-response factor (for each cause of death and age group) was calculated as a population-weighted average of the concentration-response factors of individual metropolitan areas. This can also be represented as the increase in risk multiplied by a population-weighted non-PM mortality rate (last term of Eq. 9):

$$\mathrm{CRF} = (\mathrm{RR}-1) \sum_{i=1}^{63\ \mathrm{SMSAs}} \left[\frac{\mathrm{MR}_{\mathrm{total},i}}{(RR-1)\mathrm{C}_i + 1} \cdot \frac{\mathrm{POP}_i}{\sum_{i=1}^{63\ \mathrm{SMSAs}} \mathrm{POP}_i} \right] = (RR-1) \sum_{i=1}^{63\ \mathrm{SMSAs}} \left[\mathrm{MR}_{\mathrm{non\text{-}PM},i} \cdot \frac{\mathrm{POP}_i}{\sum_{i=1}^{63\ \mathrm{SMSAs}} \mathrm{POP}_i} \right] \quad (9)$$

where POP_i is the population size of metropolitan area i in persons.

Severity factors and effect factors

The human health burden of disease due to the emission of an atmospheric pollutant can be expressed using disability-adjusted life years (DALY) (Murray and Lopez 1996). DALY are the sum of years of life lost (YLL) and years of life lost due to disability (YLDs) for a disease. YLDs are the product of the incidence, duration, and weight factor (on a scale of 0 (perfect health) to 1 (death)) for that disease (Murray and Lopez 1996). Severity factors relate the cases of death attributed to PM, determined by the above-described dose-response, to the corresponding number of DALY. Severity factors are expressed in terms of DALY/death, where "death" in the denominator refers to the PM-attributed cases of cardiopulmonary or lung cancer mortality calculated using the DRFs.

We used DALY and YLL which do not include age weights or 3 % discounting; these have been taken as the standard for LCIA (Crettaz et al. 2002; Hofstetter 1998; Pennington et al. 2002; van Zelm et al. 2008). Users interested in a value-of-statistical-life quantity (VSL) may convert the $\mathrm{PM}_{2.5}$-associated mortality rate to a VSL.

Effect factors for secondary $\mathrm{PM}_{2.5}$ were assumed to be equivalent to effect factors from primary $\mathrm{PM}_{2.5}$ since the effect factor was derived from monitors capturing a mixture of primary and secondary $\mathrm{PM}_{2.5}$.

Characterization factors—impact per kilogram emitted

The human health impact per kilogram of a given atmospheric emission, called the characterization factor (CF, DALY $\mathrm{kg_{emitted}}^{-1}$), is the product of four parameters:

$$\mathrm{CF} = \mathrm{SF} \cdot \mathrm{DRF} \cdot \mathrm{XF} \cdot \mathrm{FF} = \mathrm{EF} \cdot \mathrm{iF} \quad (10)$$

The fate factor (FF, $\mathrm{kg_{air}}$ per [$\mathrm{kg_{emitted}}$ year^{-1}]) relates the emission rate ($\mathrm{kg_{emitted}}$ year^{-1}) to the mass in the exposure medium ($\mathrm{kg_{air}}$); the exposure factor determines the change in intake rate per change in mass in the environment (XF, [$\mathrm{kg_{inhaled}}$ year^{-1}] per $\mathrm{kg_{air}}$), and the dose-response factor indicates the change in morbidity or mortality attributable to a change in intake (DRF, cases per $\mathrm{kg_{inhaled}}$). The emitted pollutant can be a single chemical or a group of chemicals, and it can be a primary pollutant or a contributor to a secondary pollutant (Rosenbaum et al. 2007). The product of SF and DRF is the effect factor (EF, DALY $\mathrm{kg_{inhaled}}^{-1}$) and the product of XF and FF is the intake fraction (iF, $\mathrm{kg_{inhaled}}$ per $\mathrm{kg_{emitted}}$). The intake fraction for primary pollutants indicates the fraction of the emission taken in (inhaled) by the overall population (Bennett et al. 2002). The intake fraction for secondary pollutants is the inhaled mass of the pollutant attributable to a specific precursor per mass emission of the precursor.

Since coarse (between 2.5 and 10 μm in aerodynamic diameter, $\mathrm{PM}_{10-2.5}$) particles are likely removed faster from the atmosphere than fine particles ($iF(\mathrm{PM}_{10-2.5}) < iF(\mathrm{PM}_{2.5}$

(Lai et al. 2000; Liu and Nazaroff 2003)) and the effect factor of coarse particles is lower ($EF(PM_{10-2.5}) << EF(PM_{2.5})$ (Brunekreef and Forsberg 2005; Cooke et al. 2007; Dockery et al. 1993; European Commission 2005; Hofstetter 1998; U.S. Environmental Protection Agency 2010)), the overall characterization factor is therefore dominated by $PM_{2.5}$:

$$\begin{aligned} CF(PM_{10}) &= iF(PM_{2.5})\cdot EF(PM_{2.5})\cdot f_{PM2.5} + iF(PM_{10-2.5})\cdot EF(PM_{10-2.5})(1-f_{PM2.5}) \approx iF(PM_{2.5})\cdot EF(PM_{2.5})\cdot f_{PM2.5} \\ &= CF(PM_{2.5})\cdot f_{PM2.5} \end{aligned} \quad (11)$$

where $f_{PM2.5}$ is the fraction of PM_{10} which is emitted as $PM_{2.5}$.

Burden of disease—impact per year

Estimate using ambient concentrations

$PM_{2.5}$ concentrations for each county in the nation were estimated using Voronoi neighborhood averaging of 2005 ambient monitor data from the EPA's BenMAP 3.0 software which estimates health benefits from reductions in air pollutants (Abt Associates Inc. 2008).

Estimate using emissions inventory

Stack-height specific characterization factors were assigned to each emissions source according to Table S2 in Humbert et al. (2011). Emissions with uncategorized stack heights were assigned to the low stack height category. The characterization factors were weighted according to the proportion of the population that was considered urban vs. rural in the US 2000 Census. The characterization factors for remote sources were applied in counties with population densities less than 10 persons/km^2.

Appendix B: age- or location-specific data and results

Table 4 Mortality risk ratios (RRs) associated with $PM_{2.5}$ exposure derived from the ACS Study (Pope et al. 2002) (Fig. 4) by cause of death and age group

Age group (years)	RR per 10 μg/m^3 $PM_{2.5}$ (95 % confidence interval)
All-cause mortality	
30 and older	1.04 (1.01, 1.08)
30–59	1.04 (1.00, 1.09)
60–69	1.02 (0.97, 1.06)
70 and older	1.05 (1.00, 1.09)
Cardiopulmonary mortality (ICD-9 codes 401–440 and 460–519)	
30 and older	1.06 (1.02, 1.10)
30–59	1.05 (0.98, 1.14)
60–69	1.02 (0.97, 1.07)
70 and older	1.08 (1.03, 1.15)
Lung cancer mortality (ICD-9 codes 162)	
30 and older	1.08 (1.01, 1.16)
30–59	1.04 (0.95, 1.14)
60–69	1.14 (1.03, 1.27)
70 and older	0.98 (0.84, 1.16)

Table 5 Population-weighted means for annual total mortality rates, non-PM mortality rates, and $PM_{2.5}$-attributable mortality rates per 100,000 population and attributable fractions by each mortality-cause category and age group (n=63 SMSAs)

	Ages 30–34	Ages 35–39	Ages 40–44	Ages 45–49	Ages 50–54	Ages 55–59	Ages 60–64	Ages 65–69	Ages 70–74	Ages 75–79	Ages 80 and older
Total mortality rates, 1982–1988											
Cardiopulmonary	13	28	59	120	230	390	680	1,100	1,900	3,100	8,100
Lung cancer	0.98	3.6	12	31	65	120	180	240	290	300	250
All causes	120	160	230	360	590	950	1,500	2,300	3,500	5,200	12,000
Attributable fractions											
Cardiopulmonary	0.094	0.094	0.094	0.095	0.095	0.096	0.035	0.035	0.15	0.15	0.15
Lung cancer	0.075	0.075	0.075	0.076	0.076	0.076	0.23	0.23	−0.034	−0.034	−0.034
All causes	0.077	0.077	0.077	0.077	0.078	0.078	0.041	0.041	0.095	0.095	0.095
Non-PM mortality rates											
Cardiopulmonary	13	25	53	110	200	360	660	1,100	1,600	2,600	6,900
Lung cancer	0.91	3.3	11	28	60	110	140	180	300	310	260
All causes	110	150	210	330	540	880	1,400	2,200	3,100	4,700	10,000
$PM_{2.5}$-attributable mortality rates											
Cardiopulmonary	1.3	2.7	5.6	11	22	38	24	39	290	470	1,200
Lung cancer	0.073	0.27	0.89	2.3	5.0	9.0	41	54	−9.8	−10	−8.3
All causes	9.6	12	17	28	46	75	61	93	330	500	1,100

Table 6 Population-weighted means for dose-response and concentration–response factors and final severity and effect factors for specific age groups

	Ages 30–34	Ages 35–39	Ages 40–44	Ages 45–49	Ages 50–54	Ages 55–59	Ages 60–64	Ages 65–69	Ages 70–74	Ages 75–79	Ages 80 and older
Concentration-response factor (mortality rate (per 100,000 population) per μg/m^3 $PM_{2.5}$)[a]											
Cardiopulmonary	0.063	0.13	0.27	0.54	1.0	1.8	1.1	1.8	14	22	58
Lung cancer	0.0040	0.013	0.043	0.11	0.24	0.42	1.9	2.6	−0.46	−0.48	−0.39
All causes	0.46	0.59	0.83	1.3	2.2	3.5	2.9	4.3	16	23	52
Dose–response factor (deaths per kg $PM_{2.5}$ inhaled)[a]											
Cardiopulmonary	0.13	0.27	0.56	1.1	2.1	3.8	2.4	3.9	29	47	120
Lung cancer	0.0070	0.027	0.090	0.23	0.50	0.89	4.0	5.4	−0.97	−1.0	−0.83
All causes	0.96	1.2	1.7	2.8	4.6	7.4	6.1	9.2	33	49	110
Severity factors											
DALY/death											
Cardiopulmonary	120	92	68	55	47	40	33	27	21	16	6.9
Lung cancer	54	49	44	40	35	30	26	21	17	13	6.2
All causes[c]	120	88	65	52	44	37	31	25	20	15	6.9
YLL/death											
Cardiopulmonary	54	49	44	39	35	30	25	21	17	13	5.8
Lung cancer	54	49	44	39	35	30	25	21	17	13	6.0
All causes[c]	54	49	44	39	35	30	25	21	17	13	5.8
Effect factors											
DALY/death											
Cardiopulmonary	16	24	38	63	100	150	77	100	600	750	850
Lung cancer	0.40	1.4	4.0	9.3	17	27	100	120	−17	−13	−5.1
All causes	120	110	110	150	200	280	190	230	660	760	760
YLL/death											
Cardiopulmonary	7.1	13	25	44	74	110	60	82	480	600	720
Lung cancer	0.40	1.3	4.0	9.2	17	27	100	110	−16	−13	−4.9
All causes	52	61	77	110	160	220	150	190	550	630	640

[a] N=63 US Standard Metropolitan Statistical Areas

[b] Based on the Global Burden of Disease 2010 Estimates of Deaths, DALY and YLL for the High-Income North America region

[c] The all-causes severity factors are actually the cardiopulmonary + lung cancer severity factors as these are more likely to reflect the severity of PM-associated disease

YLD=DALY−YLL

Table 7 Emission-weighted average world region-specific characterization factors for primary $PM_{2.5}$

	Urban	Rural	Remote	Population-weighted average
World	2.0E−03	2.0E−04	7.8E−06	1.2E−03
Generic continent	1.2E−03	7.2E−05	7.8E−06	6.6E−04
US+Latin America	2.3E−03	5.9E−05	7.8E−06	9.4E−04
Europe	1.4E−03	1.6E−04	7.8E−06	7.8E−04
Africa+Middle East	2.0E−03	8.6E−05	7.8E−06	6.2E−04
Central Asia	1.6E−03	1.0E−04	7.8E−06	5.1E−04
South East Asia	2.3E−03	3.6E−04	7.8E−06	1.6E−03
Arctic	7.3E−04	3.3E−05	7.8E−06	1.4E−04
Oceania	9.4E−04	2.3E−05	7.8E−06	3.8E−04
Antarctica	0.0E+00	0.0E+00	7.8E−06	7.8E−06

Appendix C: alternate severity and effect factors

Estimates of disability due to PM in Appendix C Table 8 do not make the assumption that morbidity due to $PM_{2.5}$ is equivalent to morbidity due to other causes of that disease. Most of these estimates are small compared to the estimate of chronic mortality with the exception of disability due to chronic bronchitis. Künzli et al. (2000) estimated a high burden of chronic bronchitis due to PM using both incidence rates and risk ratios from the Seventh-Day Adventist Cohort Study (Abbey et al. 1993). The incidence rates of chronic bronchitis presented in the Seventh-Day Adventist Cohort Study (approximately 6 per 1,000 annually among *non-smokers* among individuals over the age of 25) (Abbey et al. 1995) are much higher than the COPD incidence estimated for industrialized nations for the WHO (approximately 2 per 1,000 annually among *all* individuals over the age of 30) (Lopez et al. 2006; Shibuya et al. 2001) considering that most COPD is attributed to smoking (Hnizdo et al. 2002; Salvi and Barnes 2009). Hofstetter (1998) proposes a very conservative disability weight to assign to chronic bronchitis—0.05 per incident case (over a 40-year duration)—compared to that used by the WHO in the 2000 Global Burden of Disease (0.17 for mild/moderate COPD and 0.53 for severe COPD) (Mathers et al. 2006a). In Appendix Table 8, we applied the more conservative severity factor to the Seventh-Day Adventist Cohort Study effect estimate associated with the high chronic bronchitis incidence rate among non-smokers. The 41 additional YLDs due to PM estimated in the Appendix Table 8 effect factor are higher than the 4 YLDs estimated in Table 1 which used our simplified severity factor calculation. The burden of chronic bronchitis due to PM may be *higher* than we account for in our simplified severity factor calculation, but the uncertainty in directly attempting to estimate the PM-associated burden of chronic bronchitis from the Seventh-Day Adventist Cohort Study is large, so we do not use the effect factors in Appendix Table 8 in our final characterization factor.

Table 8 Alternate evaluations of dose-response, severity and effect factors of PM_{10}, and conversion to $PM_{2.5}$

Type of endpoint	Dose-response factor (case/kg_{inh} PM_{10})	Source/comment	Severity factor (DALY/case)	Source/comment	Effect factor (DALY/kg_{inh} PM_{10})	Source/comment	Effect factor (DALY/kg_{inh} $PM_{2.5}$)[a]
Chronic mortality	5.76	Van Zelm et al. (2008) (RR based on Künzli et al. (2000) and F_{inc} on European Commission (2007))	10	Van Zelm et al. (2008) (based on Künzli et al. (2000) and Pye and Watkiss (2005)) Note that Bare et al. (2003) uses 10.9 DALY/case based on De Hollander et al. (1999)	57.6	Van Zelm et al. (2008) (As matter of comparison, Torfs et al. (2007) uses 82.2 DALY/kg_{inh})	96.0
Acute respiratory morbidity	0.73	Van Zelm et al. (2008) (RR based on Medina et al. (2005) and F_{inc} on Knol and Staatsen (2005))	0.025	Van Zelm et al. (2008) (based on Knol and Staatsen (2005))	0.018	Van Zelm et al. (2008)	0.030
Acute cardiovascular morbidity	0.55	Van Zelm et al. (2008) (RR based on Le Tertre et al. (2002) and F_{inc} on Knol and Staatsen (2005))	0.027	Van Zelm et al. (2008) (based on Knol and Staatsen (2005))	0.015	Van Zelm et al. (2008)	0.025
Chronic bronchitis (adults)	9.5	Künzli et al. (2000)	2	Hofstetter (1998)	19		31.7
Chronic bronchitis (children)	140	Künzli et al. (2000)	0.025	Hofstetter (1998)	3.6		6.0
Restricted activity days	6100	Künzli et al. (2000)	2.7E−4	Hofstetter (1998)	1.7		2.8
Asthmatics: asthma attacks (children)	56	Künzli et al. (2000)	2.7E-4	Hofstetter (1998)	0.015		0.025
Asthmatics: asthma attacks (adults)	140	Künzli et al. (2000)	2.7E−4	Hofstetter (1998)	0.037		0.062

[a] $PM_{2.5}$ was assumed to be 1.67 times as toxic as PM_{10} (European Commission 2005)

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