Environ Model Assess (2012) 17:613–622
DOI 10.1007/s10666-012-9317-3

Environmental Modeling and Methods for Estimation of the Global Health Impacts of Air Pollution

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Received: 7 December 2011 / Accepted: 9 March 2012 / Published online: 14 April 2012

Abstract Air pollution is increasingly recognized as a significant contributor to global health outcomes. A methodological framework for evaluating the global health-related outcomes of outdoor and indoor (household) air pollution is presented and validated for the year 2005. Ambient concentrations of PM2.5 are estimated with a combination of energy and atmospheric models, with detailed representation of urban and rural spatial exposures. Populations dependent on solid fuels are established with household survey data. Health impacts for outdoor and household air pollution are independently calculated using the fractions of disease that can be attributed to ambient air pollution exposure and solid fuel use. Estimated ambient pollution concentrations indicate that more than 80% of the population exceeds the WHO Air Quality Guidelines in 2005. In addition, 3.26 billion people were found to use solid fuel for cooking in three regions of Sub Saharan Africa, South Asia and Pacific Asia in 2005. Outdoor air pollution results in 2.7 million deaths or 23 million disability adjusted life years (DALYs) while household air pollution from solid fuel use and related indoor smoke results in 2.1 million deaths or 41.6 million DALYs. The higher morbidity from household air pollution can be attributed to children below the age of 5 in Sub Saharan Africa and South Asia. The burden of disease from air pollution is found to be significant, thus indicating the importance of policy interventions.

Keywords Air pollution · Atmospheric PM2.5 · Health impact methodology · Solid fuels · Household health

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1 Introduction

The relation between ambient air pollution and health has been well discussed (see [1] for a detailed literature survey of the health impacts of outdoor air pollution) and a number of epidemiological studies (including, for example, [2–4]) have reported significant effects of exposure to fine particles (particulate matter with aerodynamic diameter smaller than 2.5 μm) on long-term mortality due to cardiopulmonary disease and lung cancer in adults, while controlling for smoking, diet, occupation and other factors. There is also evidence of significant mortality and morbidity losses associated with household air pollution caused by the inefficient combustion of solid fuels [5].

This has led to increasing recognition of the need for policies that can sufficiently control for the health impacts from air pollution. An integrated air quality policy approach

will require adequate knowledge base and analytical tools that combine information on expected trends in anthropogenic activities that relate to air pollution and information on atmospheric dispersion of emissions including representation of urban areas (see [6] for discussion). Limited measurement data for air pollution and the absence of dispersed and advanced air pollution sensors makes it difficult to obtain accurate measurements of air pollutants in general. Recent advances in satellite measurements are helping to improve the availability of information on air pollutants, in particular fine particulate matter (see, for example, [7]). In addition, atmospheric models are increasingly being deployed to understand the spatial distribution of air pollutants (see [8]) and additionally compute health impacts (see [9]). Finally, integrated assessment models have also recently been updated to include more information on air pollutants to examine in particular the implications for a range of radiative forcing implications [10].

Growing concern for the serious health and environmental impacts of enduring dependence on dirty cooking fuels is also driving efforts to better understand household fuel choices, to set new targets for access to modern fuels, and design policies that facilitate a swifter transition to cleaner fuels and stoves [11, 12, 13, 14]. Undertaking consistent measurements of pollution concentrations and direct exposure levels within households at a global scale requires a much larger effort and has still not been attempted. In the absence of consistent household exposure datasets, information on populations dependent on biomass and other solid fuels is being used as a proxy for exposure. Recently, there have been more regular efforts to provide globally comprehensive estimates of the numbers of populations dependent on solid fuels [15, 14, 16].

Based on these recent developments, this paper describes a methodological basis that can be applied to specifically evaluate the atmospheric implications and health impacts of energy policies. Based on state-of-the-art modeling tools and an assessment of methodologies, it provides a template for quantifying the global health impacts of ambient and household air pollution. The results are validated for 2005. The health impact assessment approach used is similar to recent studies like [9] but updates include the link to an energy model for detailed sector based estimation of emissions and an accounting of urban and rural exposures at a spatial level.

2 Methodology

The Model for Energy Supply Strategy Alternatives and their General Environmental Impact (MESSAGE; [17–19]) is used for representing the underlying global energy system (see Fig. 1 for regional definitions in MESSAGE) and resulting greenhouse gas (GHG) and air pollutant emissions. In addition to the energy system, the model covers all GHG-emitting sectors, including agriculture, forestry, energy, and industrial sources for a full basket of greenhouse gases and other radiatively active gases (see [19–21]).

A similar set-up was used as in [20] in terms of representation of air pollutants and emissions for 2005 including open burning are consistent with [22]. Global spatially explicit emissions at a sector level (at a $1° \times 1°$ resolution) for 2005 were derived based on data described in [23].

In order to estimate the impacts of the spatially explicit emissions, atmospheric concentrations of PM, and aerosols were derived using the TM5 model. The TM5 model is an

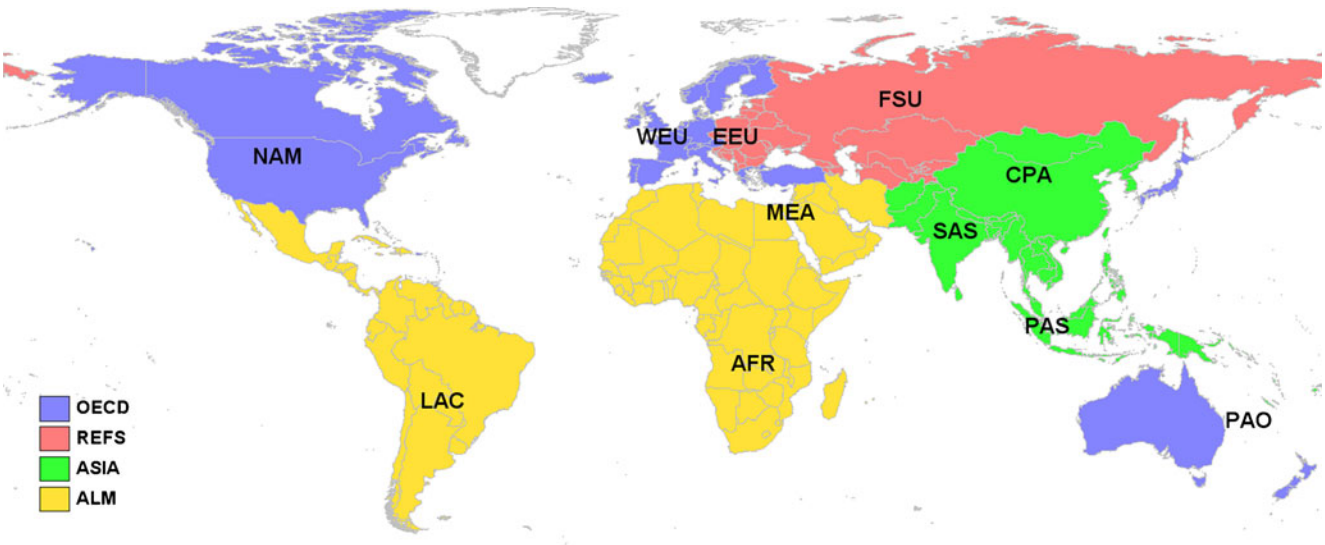


Fig. 1 Illustration of world regions in MESSAGE

off-line global transport chemistry model [24] that uses meteorological fields, including large-scale and convective precipitation and cloud data, from the European Centre for Medium Range Weather Forecast. For this work, a similar set-up in terms of model resolution has been selected as used [25]. The model has been used in a number of recent inter model comparisons [24, 26–29]. For PM2.5, TM5 includes contributions from (a) primary PM2.5 released from anthropogenic sources, (b) secondary inorganic aerosols formed from anthropogenic emissions of SO_2, NO_x, and NH_3 (including water vapor), (c) particulate matter from natural sources (soil dust, sea salt, biogenic sources). The spatial resolution of 1°× 1° used is state-of-the art for capturing the global features of long-range transported pollutants for the current mega regional scale analysis at which we calculate health impacts. However, given that ambient concentrations of some air pollutants may show strong variability at a much finer scales (e.g., in urban areas, at hot spots close to industrial point sources of emission, etc.), and could thus result in variable impacts on populations, we also separately estimate for all regions, an urban increment at the grid cell according to population density and the area over which they are emitted. The urban and rural population fractions are estimated by setting a threshold on the population density in high-resolution sub-grids (see Appendix I for details).

Household solid fuel dependence was independently estimated for the five MESSAGE regions of Sub Saharan Africa (AFR), Pacific Asia (PAS), South Asia (SAS), Centrally Planned Asia (CPA) and Latin America (LAM) in 2005 using nationally representative health and socio-economic surveys from key countries [30, 31, 32] and comparing these with other existing estimates of solid fuel dependence from [16] and the [33].

Health impacts from outdoor and household air pollution based on mortality and disability adjusted life years (DALYs) were further estimated using available World Health Organization (WHO) Comparative Risk Assessment methodologies [34] as described below:

Outdoor air pollution (OAP) The population-attributable fraction (PAF) approach based on the gradient of risk between the theoretical minimum level of air pollution exposure and the estimated observed exposure as detailed in [34] is used. This involved the estimation of attributable fractions (see Appendix II for details) which were further combined with population weighted average PM2.5 concentrations for the MESSAGE regions (2005 population estimates are based on [35]). Health impacts are estimated based on total PM2.5 concentrations. We do not estimate the health-related impacts of ozone, although recent evidence suggests that this could be significant (see, for example, [36]). We use cause-specific risk rates globally for selected risk categories based on [37] and as applied in [38] as regionally specific RRs are not available. We limit the analysis to adults over 30 years of age as detailed in Table 1 and use a concentration threshold range of 7.5–50 $\mu g/m^3$ based on [38] and later discussed in [39]. However, as discussed in many studies (including [38, 39]), whether or not there is a threshold makes a large difference to the estimate of attributed deaths, and the linearity or otherwise of the dose-response association is important and will have a significant impact on the results. There have been some recent studies suggesting a nonlinear relationship between estimated inhaled doses of PM2.5 (at higher levels) from ambient air pollution exposure. To-date, however, systematic non linear concentration response functions have not been published (see [40] for discussion on the implications of non-linearity and existing gaps).

Household air pollution (HAP) Health impacts attributable to solid fuel use in homes are estimated using methodology described in [41] and is described in detail in Appendix II. We use household dependence on solid fuels (biomass and coal) as a proxy for actual exposure to household air pollution. We are cognizant of the fact that this method neglects the large variability of exposures within households using solid fuels (e.g., due to differences in ventilation levels, etc.). However, the lack of comparable national or regional quantitative data on exposures within households, made the use of this method necessary. Estimates of relative risks for household air pollution as obtained from [41] and [42] and summarized in Table 2 were used to estimate the burden of those diseases with strong epidemiological evidence for an enhanced risk due to solid fuel use. While there is some evidence of increased incidence of cataracts and other eye diseases and perinatal effects as a consequence of exposure

Table 1 Relative risk rates for outdoor air pollution

Health outcome	GBD Category, WHO 2009	Group (sex, age in years)	Relative risk (per 10 $\mu g/m^3$)	Confidence Interval (CI)
Cardiopulmonary (infectious and chronic respiratory diseases and selected cardiovascular outcomes for adults)	39, 40, 106–109, 111	Men and women ≥30	1.059	1.015–1.105
Lung cancer	333	Men and women ≥30	1.082	1.011–1.158

Table 2 Relative risks for household air pollution

Health outcome	GBD category, WHO 2009	Group (sex, age in years)	Mean relative risk	Confidence interval (CI)
ALRI	39	Children <5	2.3	1.9–2.7
COPD	112	Women ≥30	3.2	2.3–4.8
Lung cancer (from exposure to coal smoke)	333	Women ≥30	1.9	1.1–3.5
Ischemic Heart Disease (IHD)	107	Women ≥30	1.2	n.a
COPD	112	Men ≥30	1.8	1.0–3.2
Lung cancer (from exposure to coal smoke)	333	Men ≥30	1.5	1.0–2.5

to smoke from solid fuel combustion, we do not include these in our analysis. In addition to adult-related diseases, we include here acute lower respiratory infections (ALRI) in children for which household air pollution from solid fuel use is a significant risk factor.

As seen in Tables 1, 2, considerable overlap exists between the underlying disease categories and populations at risk for outdoor and indoor air pollution. As discussed in [38], human exposure to air pollution occurs both indoors and outdoors and an individual's exposure to ambient urban air pollution depends on the relative amounts of time spent indoors and outdoors, the proximity to sources of ambient air pollution, and on the indoor concentration of outdoor pollutants. We cannot estimate the exact extent of the overlap in terms of the resulting impacts, but expect that in some developing nations it could be significant. This implies that the outdoor air pollution health impacts and household health impact estimates are not additive. We do not correct for this. There is also recent literature which suggests that the a more detailed component-wise estimation of PM2.5 could potentially have implications for the magnitude of health impacts (see, for example, [43, 44]) but we do not examine this issue in detail here.

We use baseline data from [45] on mortality and DALYs. This data is available at (http://www.who.int/healthinfo/global_burden_disease/projections/en/index.html) and was sampled to the MESSAGE regions based on underlying population shares of the countries. We base our estimates for 2005 on the 2004 and 2008 data which is available.

3 Results

Estimates of global emissions of SO_2, NO_x and PM2.5 are shown in Fig. 2. The power, industry, and transportation sectors are major emission sources globally. In addition, the residential sector is a large contributor to energy related PM emissions, especially in Asia and Africa due to the use of biomass and coal in cooking. In some regions like Africa and Latin America, non-energy sources, in particular open biomass burning are a dominant source of PM emissions.

Table 3 presents the resulting population weighted average annual PM2.5 concentration for the year 2005 aggregated from the gridded values to MESSAGE regions. The calculations were performed with a near-final version of the emissions. In order to ensure that these concentrations are completely consistent with emissions corresponding to the RCP inventories, some amount of rescaling was necessary. Appendix III shows the differences in PM2.5 concentrations

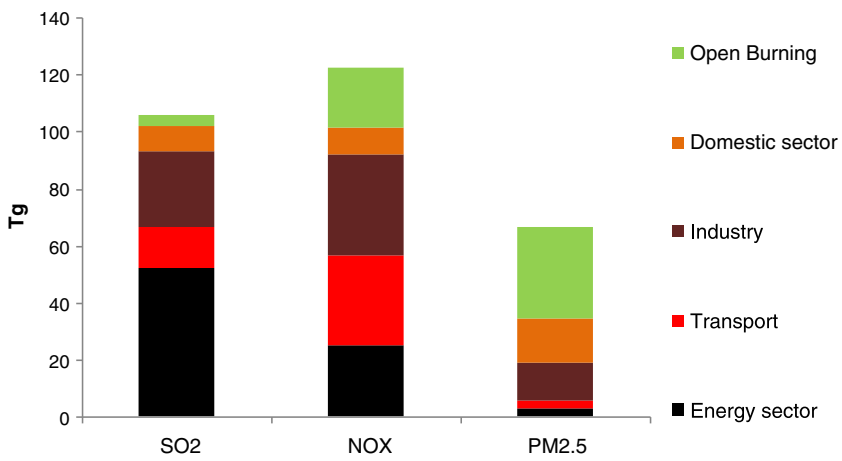


Fig. 2 Global emissions of SO_2 (T_g SO_2), NO_x (T_g NO_x) and PM2.5 (Tg PM2.5). Open burning includes agricultural waste burning, savannah and deforestation related emissions

Table 3 Regional average population weighted mean PM2.5 concentrations (including dust, sea salt and secondary organic aerosols, SOA), 2005, in micrograms per cubic meter

Region	Total	Comparison with other available studies
World	31.4	27 [7]
Europe (includes WEU, EEU and FSU)	21.8	16–17 [46]; 15–17 [7]
North America (NAM)	15.6	11–13 [7]; 13.8 (estimate for Eastern US; [47])
Pacific OECD (PAO)	21.2	
Centrally Planned Asia (CPA)	60.9	
South Asia (SAS)	31.5	
Pacific Asia (PAS)	19.5	
Latin America (LAM)	9.9	7 (estimate for South America, [7])
Sub Saharan Africa (AFR)	15.6	
Middle East and North Africa (MEA)	18.4	26 (estimate for North Africa [7])

before and after the scaling. Global PM2.5 concentration was estimated at 31.4 μg/m^3. Our estimates are quite comparable to a recent study by [7] who determined global estimates of population weighted PM2.5 concentrations of 20–27 μg/m^3 using a combination of total column aerosol optical depths from satellite instruments and models.

We compare the resulting PM2.5 concentrations with WHO Air Quality Guidelines (AQGs) and the three interim targets (IT 1–3) set for long-term exposure to PM2.5 [48]. As seen in Fig. 3 more than 80% of the world's population is estimated to exceed the WHO AQG for PM2.5 of 10 μg/m^3 while more than 30% also exceed the WHO Interim Target-1 of 35 μg/m^3.

We estimate the populations dependent on solid fuels in 2005 based on national level household survey data in three regions—around 3.26 billion, specifically in Sub Saharan Africa, South Asia, and Pacific Asia. Our estimates of populations dependent on solid fuels are slightly higher for all regions than other recent estimates including for example [16, 33]. This is mainly because of the inclusion of multiple fuels as our estimates are based on national level household survey data assuming all households that report some positive consumption of any of the solid fuels (unprocessed biomass, charcoal and coal) as dependent on solid fuels, even if they use these only as secondary or tertiary sources of cooking energy or are using these for other thermal purposes such as heating. Table 4 presents our estimates of the share of population using solid fuels in rural and urban areas.

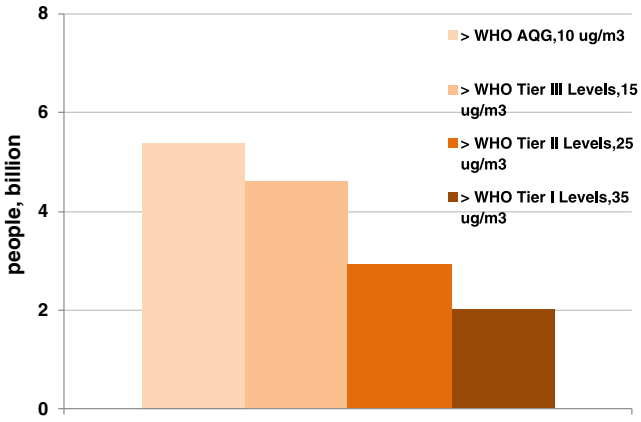


Fig. 3 Global population exposed to ambient concentrations of PM2.5 exceeding long-term WHO AQG and three IT Levels in 2005

Table 4 Fractions of population dependent on solid fuels, 2005, %

Region	Rural		Urban	
	Coal	Biomass	Coal	Biomass
SAS	0.5	97.8	4.5	53
PAS	0	82.4	0	31
AFR	0	97.5	0	88
CPA	30	50	28	10
LAM	2	60	1	6

We estimate that outdoor air pollution results in 2.7 million annual deaths or 23 million annual (DALYs) worldwide in 2005 as seen in Table 5 (also indicated are the ranges based on uncertainties in RRs). This represents around 5% of all deaths, 2% of all DALYs and around 12% of the total burden that can be attributed to cardiovascular, respiratory and lung cancer related causes. More than 70% of this burden is felt in Asia (CPA+SAS+PAS) alone. These results can be compared to other recent studies, including [9] who estimate 2.4–3.7 million deaths globally from exposure to PM2.5. Reasons for the higher estimates from our analysis as compared to for instance that estimated by previous GBD studies (see, for example, [34]

Table 5 Annual mortality and DALYs from outdoor air pollution, 2005 (in parenthesis are the ranges of impacts from low and high confidence intervals of risk rates)

	Total population, million>30 years	Annual mortality (millions)	Annual DALYs (millions)
OECD	616	0.37 (0.07–0.58)	2.4 (0.44–3.68)
REFS	238	0.26 (0.07–0.42)	1.97 (0.52–3.18)
CPA	782	1.05 (0.29–1.57)	7.98 (2.2–11.8)
SAS	585	0.69 (0.19–1.09)	6.93 (1.94–10.91)
PAS	230	0.12 (0.03–0.19)	1.12 (0.29–1.84)
LAM	244	0.04 (0.01–0.07)	0.38 (0.1–0.64)
AFR	208	0.14 (0.04–0.23)	1.56 (0.42–1.58)
MEA	142	0.05 (0.01–0.08)	0.48 (0.13–0.18)
World	3,061	2.7 (0.72–4.23)	22.83 (6–35.5)

Table 6 a Health impacts of household air pollution (HAP) based on mean RRs, mortality, Millions (in parenthesis are the ranges of impacts from the low and high confidence intervals of risk rates). b Health impacts of household air pollution based on mean RRs, DALYs, millions (in parenthesis are the ranges of impacts from low and high confidence intervals)

Disease, sex and age	SAS	PAS	AFR	CPA	LAM
a Annual HAP-related mortality (million)					
ALRI, children <5	0.22 (0.18–0.25)	0.05 (0.04–0.06)	0.50 (0.42–0.56)	0.03 (0.02–0.03)	0.01 (0.00–0.01)
COPD, women >30	0.19 (0.16–0.23)	0.1 (0.08–0.12)	0.03 (0.02–0.03)	0.26 (0.18–0.34)	0.02 (0.01–0.03)
Lung cancer, women >30	0	0	0	0.02	0
COPD, men >30	0.16 (0.00–0.25)	0.06 (0.00–0.11)	0.03 (0.00–0.05)	0.12 (0.00–0.25)	0.01 (0.00–0.02)
Lung cancer, men >30	0	0	0	0.03	0
Ischemic heart disease, women >30	0.11	0.03	0.02	0.02	0.01
Ischemic heart disease, men >30	0.08	0.02	0.02	0.01	0.01
b Annual HAP-related DALYs (million)					
ALRI, children <5	7.94 (6.56–8.92)	1.83 (1.46–2.12)	17.58 (14.65–19.65)	0.98 (0.79–1.13)	0.28 (0.21–0.35)
COPD, women >30	2.23 (1.80–2.62)	0.90 (0.69–1.10)	0.27 (0.22–0.31)	1.6 (1.14–2.10)	0.27 (0.18–0.38)
Lung cancer, women >30	0.005	0.00	0.00	0.22	0.005
COPD, men >30	1.76 (0.00–2.83)	0.67 (0.00–1.19)	0.37 (0.00–0.58)	1.19 (0.00–2.37)	0.14 (0.00–0.30)
Lung cancer, men >30	0.007	0	0	0.3	0.004
Ischemic heart disease, women >30	1.05	0.26	0.21	0.16	0.06
Ischemic heart disease, men >30	0.82	0.2	0.17	0.11	0.05

and [38]).include the representation of both urban and rural exposures (thus including effects of industrial sources and other hot spots typically located outside urban areas) and the increase in global population since previous estimations. However, it is important to stress that health impact estimations from ambient air pollution exposures are subject to a number of uncertainties. The upcoming Global burden of Disease report [49] is expected to review a number of the underlying uncertainties based on latest epidemiological evidence.

Our estimates in Table 6 indicate that more than 2.1 annual million deaths or alternatively the loss of 41.6 annual million DALYs could be attributed to solid fuel use and related indoor smoke in 2005. In terms of shares, these results correspond to 23% of deaths and 35% of DALYs from combined causes (ALRI, COPD, lung cancer, and IHD). The HAP DALY estimates are higher than those from OAP due to the very high incidence of the morbidity burden among children less than 5 years of age which accounts for more than 68% of the total, with the largest fraction of these occurring in Sub Saharan Africa. HAP related premature child deaths are seen to exceed those due to HIV/AIDS and malaria [45].

We can compare these estimates to that of [50] who estimate globally 1.6 million deaths and 38.5 million DALYs were lost in the year 2000 as a result of exposure to indoor smoke from SFU. Two main reasons for the increased impacts are the higher estimates of populations dependent on solid fuels and the inclusion of ischemic heart disease, a risk category, which has not been included in household (indoor) impact estimates to date.

4 Summary

This paper provides a framework that combines energy and atmospheric models and uses available methodologies to estimate the global health impacts from outdoor and household air pollution. Global population weighted mean average ambient PM2.5 concentration for the year 2005 was estimated at 31–35 μg/m^3. More than 80% of the world's population is seen to currently exceed the WHO AQG for PM2.5 of 10 μg/m^3 while more than 30% also exceed the WHO Interim Target-Tier 1 level of 35 μg/m^3. Ambient concentrations in developing countries, particularly in Asia, are seen to be high due to large populations and significant emissions from the industrial and transportation sectors. In addition, 3.26 billion people were estimated to use solid fuel for cooking in 2005 in Sub Saharan Africa, South Asia, and Pacific Asia, leading to high exposures to household air pollution.

We estimate health impacts of 2.7 million annual deaths and 23 million annual DALYs from outdoor air pollution in 2005. This represents around 5% of all deaths, 2% of all DALYs and around 12% of the total burden that can be attributed to cardiovascular, respiratory and lung cancer related causes. We also estimate 2.1 million annual deaths and 41.6 million annual DALYs lost due to solid fuel use and related indoor smoke in developing countries. The

significantly higher morbidity impacts of HAP as compared to OAP are primarily due to large populations of children below the age of 5 who are at a large risk from indoor cooking, especially in Sub Saharan Africa and South Asia.

Our estimates are consistent with recent studies that suggest that air pollution is a more significant contributor to the global burden of disease than previously estimated. This can be explained by high ambient concentrations of combined urban and rural outdoor air pollution especially in Asia and the increases in population since previous estimates. Additionally, given regional disparities in fuel use and development, while household air pollution is the primary problem for instance in Sub Saharan Africa, regions in Asia face high levels of exposure due to both outdoor and household air pollution.

Pollution-related impacts are found to be significant when compared to other major causes of disease and death in developing countries. This indicates the need for effective air pollution-related policies that can improve health and wellbeing in such regions. This paper provides a methodological basis that can be used for assessing future policy impacts in terms of exposures and health related impacts of OAP and HAP.

Expert assessments from the upcoming Global Burden of Disease study are expected to evaluate and significantly update the most recent information on health impacts from a range of causes-including indoor and outdoor air pollution. Future analysis will need to take this into account.

Appendix I: Representing Urban/Rural Fractions of PM2.5 in TM5

TM5 model simulations were performed at a spatial resolution of 1°×1° longitude–latitude, corresponding to a nominal longitudinal resolution of ca. 111 km at 0° latitude (tropics), 79 km at 45° latitude, and 56 km at 60° latitude (latitudinal resolution is always 111 km). Ambient concentrations of some air pollutants may show strong variability at a much finer scales (e.g., in urban areas, at hot spots close to industrial point sources of emission, etc.), and could thus result in variable impacts on populations. We also separately estimate for all regions, an urban increment at the grid cell resulting from anthropogenic primary aerosol emissions, assuming that the model calculations are sufficient to cover aerosols from natural and secondary sources. The sub-grid increment parameterization attributes calculated primary aerosol concentrations according to population density and the area over which they are emitted. Population density is derived from the high (0.1°×0.1°) resolution CIESIN population dataset provided by Columbia University (http://www.ciesin.org/). The urban increment of primary aerosol concentration at the 1°×1° grid cell is calculated according to population density and the area over which they are emitted.

Assuming that the concentration of Primary PM in each 1°×1° grid cell of the model is given by

$$\mathrm{CTM5} = \frac{\mathrm{E}}{\lambda} \tag{1}$$

With E=in-cell emission intensity of BC+PPOM (primary emissions of black carbon and particulate organic matter), λ=in-cell mixing rate, including dilution.

If we distinguish rural from urban emissions, we can define the rural concentration as

$$C_{\mathrm{RUR}} = \frac{E_{\mathrm{RUR}}}{\lambda} = \frac{1 - f_{\mathrm{up}}}{1 - f_{\mathrm{ua}}} \frac{E}{\lambda} \tag{2}$$

With f_{up}=urban population fraction in the 1°×1° grid cell derived from 0.1°×0.1° population statistics, f_{ua}=urban area fraction in the grid cell.

The urban and rural population fractions are estimated by setting a threshold on the population density in high-resolution sub-grids. To conserve the grid-average concentration, after the calculation of C_{RUR}, the urban concentration must fulfill the requirement that:

$$f_{\mathrm{ua}} C_{\mathrm{URB}} + (1 - f_{\mathrm{ua}}) C_{\mathrm{RUR}} = \mathrm{CTM5} \tag{3}$$

where according to Equations 1 and 2,

$$C_{\mathrm{RUR}} = \frac{1 - f_{\mathrm{up}}}{1 - f_{\mathrm{ua}}} \mathrm{CTM5} \tag{4}$$

C_{URB} follows immediately from Eq. (3)

Equation 4 basically rescales the sub-grid concentration of primary emitted components according to population density and the area over which they are emitted.

In order to avoid very spiky artifacts associated with a small fraction of the grid occupied by a densely populated sub-area, we introduce empirical limitations to the ratio $C_{\mathrm{RUR}}/C_{\mathrm{URB}}$ and to $\mathrm{CTM5}/C_{\mathrm{RUR}}$:

1. *Primary BC and POM (C_{RUR})* should not be lower than 0.5 times the TM5 grid average. This is based on observations in Europe [51, 52]
2. Urban primary BC and POM should not exceed the rural concentration by a factor 5.

Finally, the concentration edges between urban and rural areas are smoothed numerically (linear interpolation over the 0.1°×0.1° sub-grid cells at the rural–urban border to avoid artificial gradients).

Appendix II: Methodology for Estimation of Health Impacts from Outdoor and Household Air Pollution

We estimate health impacts from ambient air pollution using the PAF approach based on the gradient of risk between the theoretical minimum level of air pollution exposure and the estimated observed exposure [34]. We apply an approach similar to that detailed in [50] which involved: (1) estimating total population exposures to PM2.5; (2) choosing appropriate exposure-response factors for PM2.5 as discussed earlier in the text; (3) determining the current rates of morbidity and mortality in the population of concern using data from [45] and (4) estimating the attributable number of deaths and diseases.

The population-attributable fraction to exposure is calculated based on [53] and is estimated as:

$$\mathrm{PAF} = \frac{P \times (\mathrm{RR} - 1)}{[P \times (\mathrm{RR} - 1) + 1]} \tag{5}$$

where P=exposure expressed in PM2.5 concentrations, and RR = relative risk for exposed versus non-exposed populations. Once the fraction of a disease that is attributed to a risk factor has been established, the attributed mortality or burden is simply the product of the total death or DALY estimates for the disease and the attributed fraction.

We estimate the effects by combining information on the exposed population and the fraction of current disease levels attributable to solid fuel use. The approach utilizes relative risk estimates for health outcomes that have been associated with exposures to household pollution due to indoor smoke from solid fuel use and uses the population dependent on solid fuels as an exposure surrogate. In contrast to the pollutant based approach, which focuses on PM2.5 concentrations from combustion, the fuel-based approach takes advantage of the large number of epidemiological investigations conducted primarily in rural areas of developed countries that treat exposure to household air pollution from SFU as a single category of exposure and appears to be the most reliable method for assessing the environmental burden of diseases from SFU in developing countries [50].

The attributable fraction to SFU, $\mathrm{AF_{sfu}}$, can be estimated as:

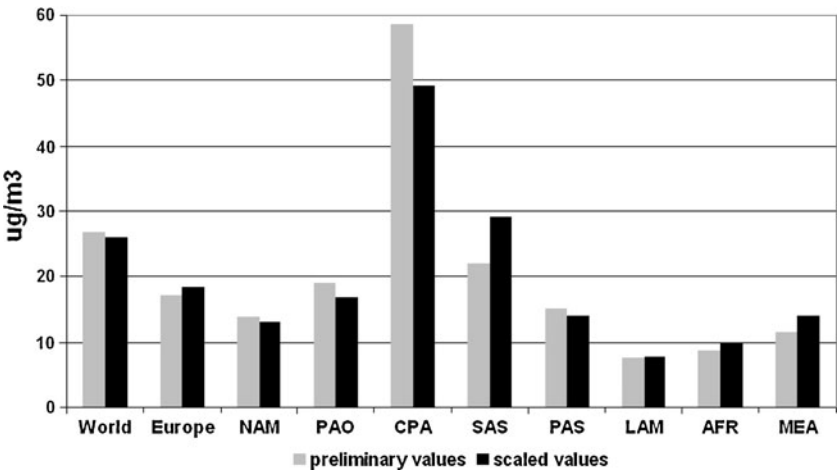
$$\mathrm{AF_{sfu}} = \left[\frac{P_\mathrm{e}(r_\mathrm{r} - 1)}{P_\mathrm{e}(r_\mathrm{r} - 1) + 1}\right] \tag{6}$$

where p_e represents the population exposed to the solid fuels and r_r the relative risk due to SFU.

Similarly, attributable burden due to the solid fuel, $\mathrm{AB_{sfu}}$ use can be estimated as

$$\mathrm{AB_{sfu}} = \mathrm{AF_{sfu}CDL} = \left[\frac{P_\mathrm{e}(r_\mathrm{r} - 1)}{P_\mathrm{e}(r_\mathrm{r} - 1) + 1}\right]\mathrm{CDL} \tag{7}$$

Appendix III: Comparison of Preliminary and Scaled Values of Average PM2.5 Concentrations (Neglecting the Effects of Dust, Sea Salt and SOA, Without Urban Increment)



Rescaling involved calculating for each grid cell, the ratio of change in concentrations to changes in emissions for each component separately and scaling for the change in emissions. This assumes no regional transfer of emissions but assuming that emission changes are not at the grid level but rather at country/state/province level, the relative change in emissions within the cell is similar to the relative changes of the surrounding cells. Shown above are the comparisons of PM2.5 estimates before and after scaling. The differences were found not to impact the health impacts significantly due to the further truncation of the response above 50 $\mu g/m^3$.

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