



Estimation of the Global Health Impacts of Air Pollution

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Interim Report

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Shilpa Rao, Vadim Chirkov, Frank Dentener, Rita Van Dingenen, Shonali Pachauri, Pallav Purohit, Markus Amann, Chris Heyes, Patrick Kinney, Peter Kolp, Zbigniew Klimont, Keywan Riahi, Wolfgang Schoepp

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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 PM_{2.5} 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 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 five 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.

About the Authors

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1. Introduction

The relation between ambient air pollution and health has been well discussed (see Curtis et al., 2006) for a detailed literature survey of the health impacts of outdoor air pollution) and a number of epidemiological studies (including, for example, Dockery et al. (1993); Pope et al. (1995); and Schwartz et al. (1996) 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 (WHO, 2009).

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 Jack and Kinney (2010) 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, van Donkelaar et al. (2010)). In addition, atmospheric models are increasingly being deployed to understand the spatial distribution of air pollutants (see Brauer et al. (2012)) and additionally compute health impacts (see Anenberg et al. 2010). 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 (van Vuuren et al., 2011).

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 (AGECC, 2010; Ekholm et al., 2010; International Energy Agency, 2011; WB, 2011). 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 (UNDP and WHO, 2009; International Energy Agency, 2011; GEA, forthcoming).

Based on these recent developments, this report 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 Anenberg et al. (2010) 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) (Messner and Strubegger, 1995; Rao and Riahi, 2006; Riahi et al., 2007) is used for representing the underlying global energy system (see Figure 1 **Error! Reference source not found.** for regional definitions in MESSAGE) and resulting GHG and air pollutant emissions. In addition to the energy system the model covers all greenhouse gas (GHG)-emitting sectors, including agriculture, forestry, energy, and industrial sources for a full basket of greenhouse gases and other radiatively active gases (see Riahi et al., 2007; Riahi et al., 2011a; Riahi et al., 2011b).

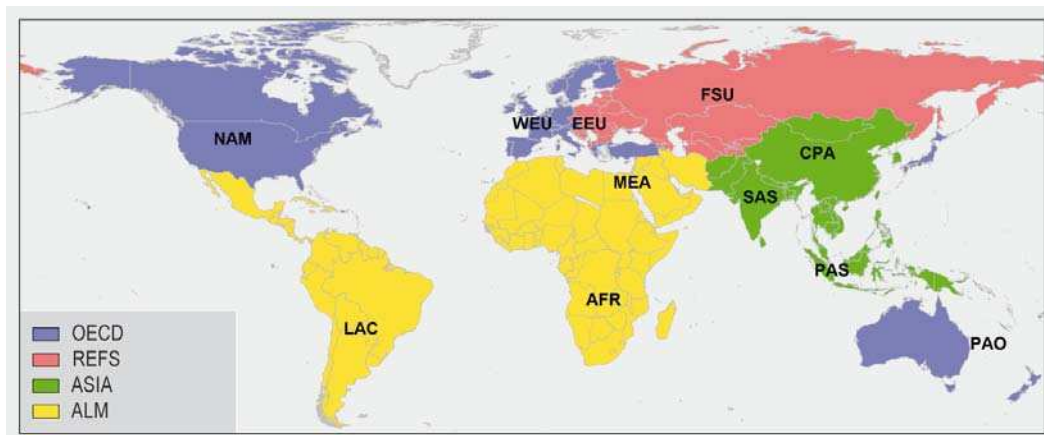


Figure 1: Illustration of World Regions in MESSAGE.

A similar set up was used as in Riahi et al. (2011b) in terms of representation of air pollutants and emissions for 2005 including open burning are consistent with Lemarque et al. (2010). Global spatially explicit emissions at a sector level (at a $1^{\circ} \times 1^{\circ}$ resolution) for 2005 were derived based on data described in Granier et al. (2010).

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 off-line global transport chemistry model (Krol et al., 2005) that uses meteorological fields, including large-scale and convective precipitation and cloud data, from the European Centre for Medium Range Weather Forecast (ECMWF). For this work a similar set-up in terms of model resolution has been selected as used (Van Aardenne et al., 2009). The model has been used in a number of recent inter model comparisons (Dentener et al., 2005; Krol et al., 2005; Dentener et al., 2006; Bergamaschi et al., 2007; Fiore et al., 2009). For PM_{2.5}, TM5 includes contributions from (i) primary PM_{2.5} released from anthropogenic sources, (ii) secondary inorganic aerosols formed from anthropogenic emissions of SO₂, NO_x and NH₃ (including water vapor), (iii) particulate matter from natural sources (soil dust, sea salt, biogenic sources). The spatial resolution of 1°x1° 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 (SUSENAS, 2004; NSSO, 2007; GLSS5, 2008) and comparing these with other existing estimates of solid fuel dependence from UNDP and WHO (2009) and the IEA/UNDP/UNIDO (2010).

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 (CRA) methodologies (WHO, 2002) and are detailed below:

Outdoor air pollution: 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 WHO (2002) is used. This involved the estimation of attributable fractions which were further combined with population weighted average PM_{2.5} concentrations for the MESSAGE regions (2005 population estimates are based on UN (2009)). Health impacts are estimated based on total PM_{2.5} concentrations. We do not estimate the health related impacts of ozone, although recent evidence suggests that this could be significant (see, for example, Jerrett et al. (2009)).

We use cause specific risk rates for selected risk categories based on Pope et al. (2002) and as applied in Cohen et al. (Cohen et al., 2004) globally (regionally specific RRs are not used due to lack of data) and limit the analysis to adults over 30 years of age as detailed in Table 1-a. and concentration threshold (CT) range of 7.5-50 µg/m³ based on Cohen et al. (2004) and later discussed in Krewski et al. (2009). However, as discussed in many studies (including Cohen et al. (2004); Krewski et al. (2009)), 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 PM_{2.5} (at higher levels) from ambient air pollution exposure. To-date however, systematic non linear concentration response functions have not been published (see Smith and Peel (2010) for discussion on the implications of non-linearity and existing gaps).

Household air pollution: Health impacts attributable to solid fuel use in homes are estimated using methodology described in Desai et al. (2004) and 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 Desai et al. (2004) and Wilkinson et al. (2009) and summarized in Table 1b 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 to smoke from solid fuel combustion, we do not include these in our analysis. In addition to adult related diseases, we include here acute respiratory infections (ALRI) in children for which household air pollution from solid fuel use is a significant risk factor.

Table 1a: Relative Risk rates for Outdoor Air Pollution

Health outcome	GBD Category, WHO 2009	Group (sex, age in years)	Relative risk (per 10 µg/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 1b: 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

As seen in Table 1a-1b, considerable overlap exists between the underlying disease categories and populations at risk for outdoor and indoor air pollution. As discussed in Cohen et al. (2004), 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 related impacts presented earlier and household health impact estimates presented here are not additive. We do not correct for this. There is also recent literature which suggests that the composition of PM_{2.5} could potentially have implications this would have for the impacts on health (see, for example, Ostro et al. (2006); Ostro et al. (2009)) but we do not examine this issue in detail.

We use baseline data from WHO (2008) on mortality and disability adjusted life years (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 SO₂, NO_x and PM_{2.5} are shown as in Figure 2. The power and industrial sector (including industrial processes) and transportation sectors are major emission sources globally. In addition, the residential sector is a large contributor to energy related outdoor PM emissions, especially in Asia and Africa, where use of biomass and coal in cooking is associated with correspondingly large emissions. In regions like Africa and Latin America, non-energy sources, in particular savannah burning and forest fires are the dominant source of PM emissions.

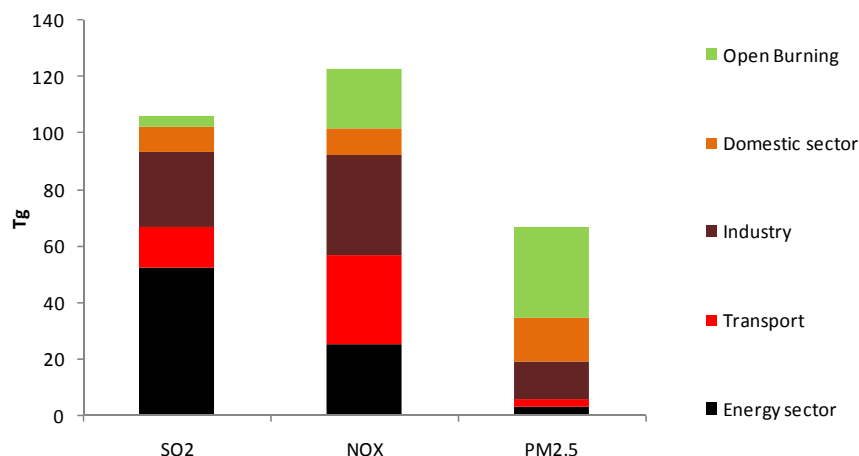


Figure 2: Global Emissions of SO₂ (Tg SO₂), NO_x (Tg NO_x) and PM_{2.5} (Tg PM_{2.5}). Open burning includes agricultural waste burning, savannah and deforestation related emissions.

Table 2 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 before and after the scaling. Global PM2.5 concentration was estimated at 30.3 $\mu\text{g}/\text{m}^3$. Our estimates are quite comparable to a recent study by van Donkelaar et al. (2010) who determined global estimates of population weighted PM2.5 concentrations of 20-27 $\mu\text{g}/\text{m}^3$ using a combination of total column aerosol optical depths from satellite instruments and models.

Table 2: Regional Average Population weighted mean PM2.5 concentrations (including dust, sea salt and secondary organic aerosols, SOA), 2005, $\mu\text{g}/\text{m}^3$

Region	Total	Comparison with other available studies
World	30.6	27 (van Donkelaar et al., 2010)
Europe (includes WEU, EEU & FSU)	21.8	16-17 (de Leeuw and Horálek, 2009); 15-17 (van Donkelaar et al., 2010)
North America (NAM)	15.6	11-13 (van Donkelaar et al., 2010); 13.8 (estimate for Eastern US (Liu et al., 2005))
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 (van Donkelaar et al., 2010))
Sub Saharan Africa (AFR)	15.6	
Middle East and North Africa (MEA)	18.4	26 (Estimate for North Africa, (van Donkelaar et al., 2010))

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 (WHO, 2006). As seen in **Error! Reference source not found.** more than 80% of the world's population is estimated to exceed the WHO AQG for PM2.5 of 10 $\mu\text{g}/\text{m}^3$ while more than 30% also exceed the WHO Interim Target-1 of 35 $\mu\text{g}/\text{m}^3$.

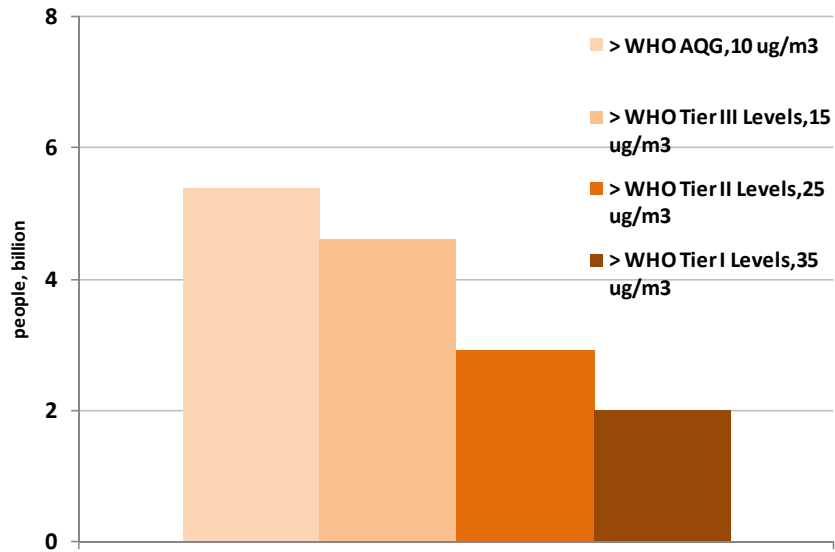


Figure 3: Global Population exposed to ambient concentrations of PM2.5 exceeding long-Term WHO AQG and three IT Levels in 2005.

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, UNDP and WHO (2009); IEA/UNDP/UNIDO (2010). 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 3 presents our estimates of the share of population using solid fuels in rural and urban areas.

Table 3: Fractions of population dependent on solid fuels, 2005, %

MESSAGE regions*	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 4 (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 Anenberg et al. (2010), 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, WHO (2002); Cohen et al. (2004)) 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 as discussed earlier that these results are indicative and that health impact estimations from ambient exposures are subject to a number of other uncertainties in for instance in the concentration thresholds. The upcoming GBD (2010) is expected to review a number of the underlying uncertainties based on latest epidemiological evidence.

Table 4: 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	3061	2.7 (0.72-4.23)	22.83 (6-35.5)

Our estimates in Tables 5a and 5b 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). Particularly relevant is that the DALY estimates are much higher than those estimated earlier from OAP which is due to the very high incidence of the morbidity burden among children less than five years of age which accounts for more than 68% of the total, with the largest fraction of these occurring in Sub Saharan Africa.

We can compare these estimates to that of Smith et al. (2004) 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.

Table 5a: 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).

Disease, sex and age	Annual HAP related Mortality (Million)				
	SAS	PAS	AFR	CPA	LAM
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

Table 5b: 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	Annual HAP related DALYs (Million)				
	SAS	PAS	AFR	CPA	LAM
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

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 PM_{2.5} concentration for the year 2005 was estimated at 31-35 $\mu\text{g}/\text{m}^3$. More than 80% of the world's population is seen to currently exceed the WHO AQG for PM_{2.5} of 10 $\mu\text{g}/\text{m}^3$ while more than 30% also exceed the WHO Interim Target-Tier 1 level of 35 $\mu\text{g}/\text{m}^3$. 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. Premature child deaths from household solid fuel use for instance exceed those estimated by WHO (2008) from HIV/AIDS and malaria. This indicates that effective air pollution related policies can be expected to have a significant impact on the improvements in health and wellbeing, especially in developing countries. This paper provides a methodological basis that can be used for assessing the air pollution related health impacts of energy policy scenarios.

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

1. Representing Urban/Rural Fractions of PM2.5 in TM5

TM5 model simulations were performed at a spatial resolution of 1°x1° 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 the aerosol from natural sources, and secondary aerosol. The subgrid increment parameterization attributes calculated primary aerosol concentrations 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. Population density is derived from the high (0.1°x0.1°) resolution CIESIN population dataset provided by Columbia University (<http://www.ciesin.org/>).

The urban increment of primary aerosol concentration at the 1°x1° grid cell is calculated according to population density and the area over which they are emitted.

Taking into account only vertical exchange, so without horizontal mixing (transport to/from neighboring cells), the concentration C in a 1°x1° grid cell of the model is given by:

$$C = \frac{E}{\lambda} \quad [1]$$

with E = in-cell emission intensity of BC+PPOM (primary emissions of black carbon and particulate organic matter).

λ =in-cell mixing rate, including vertical dilution

The actual concentration in the grid box, as resulting from the full TM5 horizontal transport dynamics, is obviously lower. This TM5 modelled grid box concentration (BC+PPOM) is represented by $CTM5$ (=grid-cell average), resulting from the full calculation.

If we distinguish rural from urban emissions, we can define the (non-horizontally-mixed) rural concentration as:

$$C_{RUR} = \frac{E_{RUR}}{\lambda} = \frac{1 - f_{up}}{1 - f_{ua}} \frac{E}{\lambda} \quad [2]$$

with f_{up} = urban population fraction in the 1°x1° grid cell derived from 0.1°x0.1° 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. This concentration has to be corrected

for the horizontal mixing (see below). To conserve the grid-average concentration, after the calculation of C_{RUR} , the urban concentration must fulfill the requirement that:

$$f_{ua}C_{URB} + (1 - f_{ua})C_{RUR} = CTM5 \quad [3]$$

Equation 3 does not account for horizontal transport. To correct this theoretical maximum for the horizontal transport we define for each grid cell as in Equation 4, the ratio between C as defined in [1] and $CTM5$ (the concentration obtained by the model) as the horizontal mixing correction factor, and apply this to correct C_{RUR} in equation 3.

$$F_h = \frac{CTM5}{C} \quad [4]$$

$$C_{RUR,corr} = \frac{1 - f_{up}}{1 - f_{ua}} \frac{E}{\lambda} \cdot F_h = \frac{1 - f_{up}}{1 - f_{ua}} \frac{E}{\lambda} \cdot \frac{\lambda}{E} \cdot CTM5 = \frac{1 - f_{up}}{1 - f_{ua}} CTM5 \quad [5]$$

Equation 5 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 $C_{RUR,corr}/C_{URB}$ and to $CTM5/C_{RUR,corr}$:

- 1) *Primary BC and POM ($C_{RUR,corr}$)* should not be lower than 0.5 times the TM5 grid average. This is based on observations in Europe (Putaud et al., 2004; Van Dingenen et al., 2004).
- 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 from $0.1^\circ \times 0.1^\circ$ sub-grid cells at the rural – urban border to avoid artificial gradients.

2. Methodology for estimation of health impacts from outdoor and household air pollution

We estimate health impacts from ambient air pollution using 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 (WHO, 2002). We apply an approach similar to that detailed in Smith et al. (2004), 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 WHO (2008) and (4) estimating the attributable number of deaths and diseases.

The population attributable fraction to exposure is calculated based on Murray et al. (2003) and is estimated as:

$$PAF = \frac{P * (RR - 1)}{[P * (RR - 1) + 1]} \quad [1]$$

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 (Smith et al., 2004).

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

$$AF_{sfu} = \left[\frac{p_e (r_r - 1)}{p_e (r_r - 1) + 1} \right] \quad [2]$$

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, AB_{sfu} use can be estimated as:

$$AB_{sfu} = AF_{sfu} CDL = \left[\frac{p_e (r_r - 1)}{p_e (r_r - 1) + 1} \right] CDL \quad [3]$$

3. Comparison of Preliminary and Scaled values of average PM2.5 concentrations

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 $\mu\text{g}/\text{m}^3$.

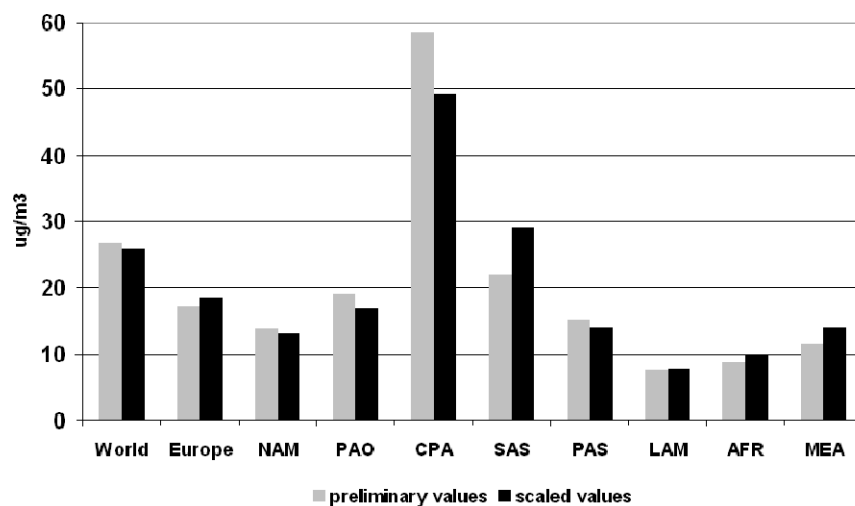


Figure 4: Comparison of Preliminary and Scaled values of average PM_{2.5} concentrations (neglecting the effects of dust, sea salt and SOA, without urban increment).

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