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Model calculated global, regional and megacity premature mortality due to air pollution

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Abstract

Air pollution by fine particulate matter ($PM_{2.5}$) and ozone (O_3) has increased strongly with industrialization and urbanization. We estimated the premature mortality rates and the years of human life lost (YLL) caused by anthropogenic $PM_{2.5}$ and O_3 in 2005 for epidemiological regions defined by the World Health Organization. We carried out high-resolution global model calculations to resolve urban and industrial regions in greater detail compared to previous work. We applied a health impact function to estimate premature mortality for people of 30 yr and older, using parameters derived from epidemiological cohort studies. Our results suggest that especially in large countries with extensive suburban and rural populations, air pollution-induced mortality rates have previously been underestimated. We calculate a global respiratory mortality of about 773 thousand yr^{-1} (YLL $\approx$ 5.2 million yr^{-1}), 186 thousand yr^{-1} by lung cancer (YLL $\approx$ 1.7 million yr^{-1}) and 2.0 million yr^{-1} by cardiovascular disease (YLL $\approx$ 14.3 million yr^{-1}). The global mean per capita mortality caused by air pollution is about 0.1 % yr^{-1}. The highest premature mortality rates are found in the Southeast Asia and Western Pacific regions (about 25 % and 46 % of the global rate, respectively) where more than a dozen of the most highly polluted megacities are located.

1 Introduction

Air pollution has intensified strongly since the industrial revolution, i.e. during the period known as the Anthropocene (Crutzen, 2002). Estimates of pollution emissions, satellite observations, measurement data from monitoring networks and model calculations unambiguously show that air quality has decreased on regional and global scales (Akimoto, 2003; Anenberg et al., 2010; Lelieveld and Dentener, 2000; Van Donkelaar et al., 2011). In parallel, evidence that air pollutants adversely impact human health, based on epidemiological studies, is mounting (e.g. Dockery et al., 1993; Pope et al., 1995;

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Samet et al., 2000; Brunekreef and Holgate, 2002; Bell and Domenici, 2008; Franck et al., 2011; Solomon et al., 2011).

Ozone (O_3) and particulate matter with an aerodynamic diameter smaller than 2.5 µm ($PM_{2.5}$) are two major contributors to hazardous air pollution with serious health impacts by cardiovascular and respiratory disease and lung cancer, and their long-term exposure has been associated with an increased risk of mortality (e.g. Krewski et al., 2009; Jerrett et al., 2009; Pellucchi et al., 2009; Pope et al., 2009; Brook et al., 2010). Unfortunately, in many parts of the world epidemiological studies and $PM_{2.5}$ and O_3 concentration data are lacking. To nevertheless obtain an overview of the global health impacts by air pollution, it is important to consistently include all regions in a comparative air quality analysis. Such information on the numbers of lives lost due to $PM_{2.5}$ and O_3 pollution is indispensable for public health planners and policymakers.

Here we assess the premature deaths caused by anthropogenic $PM_{2.5}$ and O_3 pollution for the global population of 30 yr and older in 2005, using a global atmospheric chemistry – general circulation model and applying a health impact function. We follow the approach of West et al. (2006) and Anenberg et al. (2010) who applied an atmospheric chemistry-transport model to assess human health impacts, though at relatively course resolution ($\sim 3^\circ$ latitude and longitude), and estimated the global mortality due to anthropogenic air pollution for the year 2000.

In the present study, we compute the global burden of premature human mortality for 2005, using updated emission and population data and a higher resolution model ($\sim 1.1^\circ$ latitude and longitude), so that urban and industrial regions are better resolved. Furthermore, we address regional and national impacts of air quality degradation to compare and categorize the mortality rates as well as the associated years of life lost by air pollution in different countries and major urban centers (megacities). Next the methodology and the data sources are presented (Sect. 2), followed by the results and their explanation (Sect. 3), an uncertainty analysis (Sect. 4), comparison with previous work (Sect. 5) and the conclusions (Sect. 6).

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2 Methods

The global, annual premature mortality due to anthropogenic $PM_{2.5}$ and O_3 concentrations in 2005, relative to unpolluted, pre-industrial conditions, has been estimated by employing the human health impact function also used by Anenberg et al. (2010):

$$\Delta\text{Mort} = y_0[1 - \exp(-\beta\Delta X)]\text{Pop} \quad (1)$$

where ΔMort is the change in annual mortality due to a given pollutant; y_0 is the baseline mortality rate (BMR) for a given population; β is the concentration-response function (CRF); ΔX is the change in concentration of a given pollutant X (from the preindustrial level to the year 2005); and Pop is the total population with an age of ≥ 30 yr exposed to the given pollutant (abbreviations and acronyms are listed in the Table A.1).

BMR data, described as the number of deaths in a particular year for a given population, were obtained from the World Health Organization (WHO) Statistical Information System on the country-level (WHO, 2012) based on the International Classification of Diseases 10th Revision (ICD-10) classification system for five types of mortality. The range of ICD-10 codes used for cardiovascular, lung cancer, and respiratory mortality are the codes I00 – I99, C33 – C34 and J00 – J99, respectively. Here we present results for these three categories. Data were obtained at the country level where available, and if unavailable, the appropriate WHO regional level BMR data were used for each country. Country level data were used for 36 countries and regional data were assigned to 195 countries.

The CRF describes the increased risk of a population associated with a certain health response when exposed to a particular pollutant. In this study, the CRF has been derived from the log-linear relationship between the change in pollutant concentration and the relative risk (RR) of health impacts, as established in epidemiological cohort studies and given by the function:

$$\text{RR} = \exp(\beta\Delta X) \quad (2)$$

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The epidemiological studies by Krewski et al. (2009) and Jerrett et al. (2009) were used for the CRFs for $PM_{2.5}$ and O_3, respectively. Both studies use data from the American Cancer Society (ACS) Cancer Prevention Study II (CPS-II) cohort, which includes participants who were at least 30 yr in age at the time of enrollment.

The study by Krewski et al. (2009) is an extended analysis of the ACS CPS-II cohort, providing estimates of mortality risks in the United States from various causes with improved statistical methods, and for a longer follow-up period relative to previous analyses (Krewski et al., 2000a,b). Results of RR for $PM_{2.5}$ were taken from the analysis performed for the years 1999–2000, using a random effects standard Cox model adjusted for 44 individual-level and seven ecologic covariates. The resulting mortality risk for cardiopulmonary disease (CPD) is RR = 1.129, and CRF = 0.012133. Note that we apply this factor to cardiovascular disease (CVD) since the majority of premature mortality related to $PM_{2.5}$ is caused by CVD rather than effects on the lungs. The discrepancy introduced by this assumption appears to be minor, which will be discussed in Sect. 4. The lung cancer mortality risk RR = 1.137, with a CRF = 0.012839. The 95 % confidence intervals (CI) are also reported. For example, an increase of 10 $\mu g\,m^{-3}$ in the concentration of $PM_{2.5}$ is associated with an increase of 12.9 % (95 % CI: 9.5–16.4 %) in CVD and 13.7 % (95 % CI: 5.6–22.5 %) in lung cancer mortality.

Jerrett et al. (2009) analyzed the ACS CPS-II cohort, focusing on the relationship between long-term O_3 exposure and the RR of deaths by respiratory causes. The analysis spanned data on daily maximum O_3 concentrations between 1 April and 30 September for the years 1977 to 2000. In the two-pollutant model with a known constant concentration of $PM_{2.5}$, an increase of 10 ppbv of the seasonal (April–September) average daily one-hourly maximum O_3 mixing ratio is associated with an increase of 4.0 % (95 % CI: 1.3–6.7 %) respiratory mortality, based on a mean RR = 1.04, and CRF = 0.003922.

We used concentrations of $PM_{2.5}$ and O_3 for the year 2005 and the preindustrial conditions obtained by Pozzer et al. (2012a) applying the EMAC atmospheric chemistry – general circulation model (Jöckel et al., 2006; Pringle et al., 2010; de Meij et al., 2012). We focus on 2005 because for this year a new, relatively high-resolution dataset of

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anthropogenic emissions is available (Doering et al., 2009; Pozzer et al., 2012b), and also because the meteorology of 2005 may be considered representative, i.e. without major annual modes (e.g. El Niño, North Atlantic Oscillation). The model has a horizontal resolution of about 1.1° × 1.1° (~ 100 km latitude and longitude near the equator), and a vertical resolution of 31 levels up to the lower stratosphere, of which the surface concentrations were used for this study. The concentrations of $PM_{2.5}$ used here are the yearly averages obtained from the Pozzer et al. (2012a) model version, and those of O_3 are the averages of the six-month daily maximum concentrations. The anthropogenic concentrations of $PM_{2.5}$ and O_3 were calculated as the differences between the year 2005 and the preindustrial conditions.

Model evaluation based on in situ and remote sensing observations indicates that the simulations reproduce the main spatial atmospheric distribution of the sulfate and ammonium aerosols. For nitrate some differences were found. The analysis of black and organic carbon (BC and OC) over Europe shows that the model typically overestimates BC. Over the USA the aerosol components BC and OC are in general overestimated and over China underestimated by the model (de Meij et al., 2012; Pozzer et al., 2012b). The seasonal distribution of aerosol optical depth is well represented by the model, and the model results agree well with observed $PM_{2.5}$ and O_3, and particularly well with total oxidant, i.e. $O_x = NO_2 + O_3$ (Pozzer et al., 2012a).

Global population numbers for 2005 were obtained from the Columbia University Center for International Earth Science Information Network (CIESIN) database with a resolution of 2.5 × 2.5 arc – min (about 0.04° × 0.04°; CIESIN, 2005). Since our study is based on epidemiological research that focuses on the ACS CPS-II cohort, the global population of 30 yr and older was used as the population variable (Pop) in the health impact function. The annual United Nations Population Division (UNPD) publication "World Population Prospects" reports the total annual population numbers of each country for five-year age groups. The global population of ≥ 30 yr in age was computed as the fraction of people of ≥ 30 in each country from the UNDES (2011), and applying these fractions to the appropriate grid cells of CIESIN data to obtain the total

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and gridded global target population. The total global population of 30 yr and older in 2005 is calculated to amount to 2.93 billion individuals.

The disability-adjusted life years (DALYs) of a population encompass the years of healthy life unfulfilled by individuals in the population due to both poor health and mortality. The DALYs are quantified as the sum of the years lost due to disability (YLD) and the years of life lost by premature mortality (YLL; WHO, 2008). In this study, we only examine the premature deaths due to air pollution, and thus focus on YLL as the measure of DALYs. YLL is given by the function:

$$\Delta\mathrm{YLL} = \Delta\mathrm{Mort}(\mathrm{YLL}_0/y_0) \quad (3)$$

where ΔYLL is the YLL due to premature mortality caused by $PM_{2.5}$ and O_3 pollution; and YLL_0 is the baseline YLL. Baseline YLL data were obtained from the WHO Health Statistics and Health Information System for the year 2004 with 3 % discounting and age weights, where younger ages are given a higher value than the later years in an individual's life (WHO, 2008).

In the next section, we present the calculated global levels of anthropogenic air pollution, premature mortality rates and the associated YLL by categorizing the model results according to WHO regions, sub-regions and mortality strata (WHO, 2002). The WHO divides the world into six regions, 14 epidemiological sub-regions, while five strata are applied to these regions to further classify the mortality risk of the populations within a given region (Table A2).

3 Results

The model calculated increases in $PM_{2.5}$ concentrations ($\mu g\,m^{-3}$) and O_3 mixing ratios (ppbv) between preindustrial times (approximately mid-19th century) and 2005 are presented in Fig. 1. High levels of air pollution are typically found in the USA, Europe, the Middle East, South and East Asia. In the USA anthropogenic $PM_{2.5}$ is highest in the east; O_3 in the central-south and southwest. In Europe $PM_{2.5}$ increases are highest

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in the central-west and O_3 in the south. The highest anthropogenic $PM_{2.5}$ levels worldwide are found in eastern China, while in most of East and South Asia $PM_{2.5}$ is very high. In these extensive, economically emerging regions large populations are exposed to strongly growing levels of particulate air pollution.

In East India and Southeast China anthropogenic O_3 is also rather high. It is also high in the Mediterranean and Middle East, and highest around the Arabian Gulf. It should be emphasized that some regions are additionally affected by natural $PM_{2.5}$; mainly mineral dust particles in North Africa, the Middle East and East Asia. Though these are accounted for in our model, they are not part of the present analysis because mineral dust is assumed to be natural and its sources were the same in both the preindustrial and present-day simulations. For an overview of global air pollution and regional hot spots of poor air quality, see, e.g. Sokhi (2011). Model calculated regional distributions and budgets of aerosol pollution are discussed in Pozzer et al. (2012b).

The epidemiological calculations, based on our model output, indicate that the exposure of the global population to anthropogenic $PM_{2.5}$ demands a premature death toll of about 2.0 million by cardiovascular disease and 186 thousand by lung cancer, thus referring to the year 2005 relative to the pre-industrial atmosphere (Table 1). The global distribution is shown in the top panel of Fig. 2. This is equivalent to an annual mortality of 689 and 64 per million capita of ≥ 30 in age, respectively. Of the global premature cardiovascular deaths, when sub-divided according to the WHO strata, the Western Pacific (Wpr-B) and Southeast Asian (Sear-D) regions are most strongly impacted with mortalities of about 955 and 519 thousand per year, respectively. Further analysis on the country level reveals that almost all premature deaths in the Wpr-B stratum occur in China (about 900 thousand per year), which ranks highest on the country list for cardiovascular mortality (Table 2). India is second with a total estimated premature mortality of about 421 thousand people yr^{-1}. Additional nations in South and East Asia with high rankings in Table 2 are Bangladesh and Pakistan, with premature cardiovascular mortality rates of 60–65 thousand yr^{-1}. An estimated global total of about 14.3 million years

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of life lost (YLL) is due to premature cardiovascular mortality (Table 1). About 75 % is found to occur in the Wpr-B and Sear-D strata.

The calculated global premature mortality by lung cancer of about 186 thousand per year also has a large share (63 %) by the Wpr-B stratum in the Western Pacific region; here not only the Southeast Asian but also the Eur-A stratum, mostly in Western Europe, contribute significantly to the total mortality (both with ~ 18 thousand yr^{-1}, i.e. both strata about 10 % of the global premature mortality by lung cancer) (Table 1). The global distribution is shown in the middle panel of Fig. 2. The country level analysis reveals that, of the 117 thousand deaths yr^{-1} estimated in the Wpr-B stratum, about 108 thousand occur in China (Table 2). When normalized to the number of inhabitants, the country rankings can change drastically, except for China. While China is present at or near the top according to all rankings, the subsequent two countries with the highest premature mortality rates by lung cancer (India and USA) are substituted by the smaller nations Belgium and the Netherlands (Table 2). Note that the top ranking of Eastern European countries according to Table 2 is partly related to the high baseline mortality rates in the Eur-C stratum (1.45 % yr^{-1} for cardiovascular mortality, i.e. between 2 and 3 times the BMRs for other strata, see Table 1). The analysis of YLL due to premature lung cancer mortality reveals a worldwide total of around 1.7 million yr^{-1} of life lost per year (Table 1). Of this total, the majority (nearly 64 %) is again found in the Wpr-B stratum of the Western Pacific region.

Anthropogenic O_3 is estimated to result in about 0.77 million yr^{-1} respiratory deaths globally, equivalent to 264 per million capita yr^{-1} (≥30 in age) (Table 1). The global distribution is shown in the lower panel of Fig. 2. The largest share of all strata is by Wpr-B in the Western Pacific region with 39 %, and the second largest by Sear-D, mostly in Southern Asia with a share of 27 %. The country level analysis indicates that China (273 thousand premature deaths yr^{-1}) and India (170 thousand yr^{-1}) are the top two nations globally for respiratory mortality by O_3 pollution (Table 2). When normalizing according to population numbers, Algeria, Macao and Taiwan rather lead the ranking with highest per capita respiratory mortality. Worldwide, a total of more than 5.2

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million YLL is estimated due to respiratory disease (Table 1). The top three contributing strata to this YLL estimate are Sear-D (34 %), largely in South Asia, Wpr-B (32 %) in the Western Pacific region, and the Emr-D (between Morocco and Afghanistan) and Afr-D (mostly Central-West Africa) strata (5 %).

Figure 3 presents four regions with enhanced premature mortality by $PM_{2.5}$ and O_3 (sum of lung cancer, cardiovascular and respiratory disease). Urban and industrial areas with strong pollution emissions and high population density can be identified from Fig. 3, often in combination with meteorological conditions favorable for enhanced air pollution, e.g. low rainfall and sunny conditions (slow $PM_{2.5}$ removal, strong photochemical O_3 formation). In the USA several of the major cities, such as New York, Philadelphia, Baltimore and Washington, and also Chicago, Atlanta, Houston and Dallas are apparent. In Europe the Ruhr area and Cologne (West Germany), Paris, the Randstad conurbation in the Netherlands, Belgium, major cities in the UK (London) and the Po Valley near Milan stand out. Southern Poland (Krakow), Hungary (Budapest), the Western and Eastern Ukraine (Donetsk) and Istanbul are also prominent with high premature mortality rates.

Figure 3 shows that the highest mortality rates worldwide are found in Northern India, Bangladesh and Eastern China. In India the populous Indo-Gangetic Plain (especially the central area) and megacities such as Delhi, Kolkota and Mumbai are locations of major premature mortality by air pollution. In China the regions from Beijing and Tianjin down to Xi'an, Shanghai and Nanjing, the Pearl River area, Hong Kong, and Szechuan Province are strongly affected. Further east, major metropolitan areas in South Korea (Seoul) and Japan (Tokyo, Osaka) also have high rates of premature mortality.

In Table 3 we ranked the 20 megacities and major population centers in terms of globally highest mortality rates due to air pollution. These urban areas were selected through an algorithm that assigns a threshold population density greater than 2000 individuals km^{-2} and subsequently selects coherent areas with the largest populations. Several of the megacities in South and East Asia rank high on the list, including also Jakarta in Indonesia and Karachi in Pakistan. The number of YLL is highest in

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the Pearl River area (excluding Hong Kong) with >276 thousand yr^{-1} out of a population of nearly 40 million. The cities that rank second and third are in India, i.e. Delhi and Kolkota (population nearly 19 million), for which we calculate an YLL of about 153 thousand and 124 thousand yr^{-1}, respectively.

Alternatively, if we rank the megacities according to per capita premature mortality for individuals over 30 (for which the mortality rates are representative), the highest rates are calculated for Tianjin, Beijing and Delhi, followed by the Szechuan area, Shanghai and Kolkata. The per capita mortalities in these cities are estimated at 0.19–0.28 % yr^{-1}. Again, it should be kept in mind that we only account for anthropogenically enhanced mortality rates by non-mineral aerosols. Thus, road dust, dust from construction sites, agricultural activities and desert dust are not accounted for in the present study. This can be relevant for several cities, e.g. in South and East Asia and especially in North Africa and the Middle East, so that actual mortality rates by $PM_{2.5}$ in these locations are expected to be higher.

4 Discussion of uncertainties

The use of the concentration response function of Jerrett et al. (2009) to estimate respiratory mortality attributable to long-term ozone exposure by Anenberg et al. (2010) was criticized by Prueitt and Goodman (2011). They suggested that the study should not be extrapolated globally and that no causal association has been established. In their reply Anenberg et al. (2011) justify the use of RR estimates from Jerrett et al. (2009) considering that they are consistent with the RR estimates used for long-term $PM_{2.5}$ mortality (Krewski et al., 2009). They also argue that socioeconomic data are not strong confounders and that national data of the USA may be considered more representative than other available data, which are rather city specific. Anenberg et al. (2011) acknowledge, however, that by applying alternative RR estimates by Bell et al. (2004) the global premature respiratory deaths decreased by about 50 %. Since the RRs

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by Bell et al. (2004) only account for the short-term mortality by O_3, whereas Jerrett et al. (2009) address the long-term impacts, it is expected that the latter are higher.

We reiterate that the concentration response functions applied here have been based on epidemiological cohort studies by the American Cancer Society (ACS), and do not necessarily characterize conditions in other countries for which such information is lacking. However, for regions with comparable sources of air pollution and a similar range of living conditions as in the USA the ACS results appear to be representative. For example, COMEAP (2009) concludes that the "ACS cohort study of several hundred thousand people in metropolitan areas across the USA forms the best source of coefficients suitable for application in the UK". Furthermore, factors such as smoking (including passive smoking), occupational hazards and personal characteristics are not expected to be confounding factors, since they do not vary with air pollution on a daily basis in epidemiological studies (Ostro, 2004). On the other hand, in countries where the chemical composition of particulates and the exposure conditions are rather different, e.g. in Southeast Asia and the western Pacific region (with high premature mortality rates), the ACS results are likely to be less representative, and region specific epidemiological studies will be needed (Cohen et al., 2005).

For the comparison of RRs used here with others in the literature, we recall that Krewski et al. (2009) report cardiopulmonary disease (CPD) by $PM_{2.5}$, whereas ideally one should distinguish cardiovascular (CVD) and respiratory disease by $PM_{2.5}$. Lung cancer is reported separately. Also because we use CVD data for the baseline mortality rates, we expect the discrepancy to be small. Amann and Schöpp (2011) present a compilation of RRs based on several recent cohort studies associated with a $10\,\mu g\,m^{-3}$ change in $PM_{2.5}$ in the USA and Europe, based on data by R. T. Burnett (linear relative risk model for adult mortality associated with $PM_{2.5}$ exposure – methodology for the 2005 Global Burden of Disease Outdoor Air Pollution project, 2010). The reported ranges of RRs are:

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Cardiovascular disease	RR = 1.11–1.76(0.93–2.47);
Respiratory disease	RR = 1.06–1.08(0.79–1.52);
Lung cancer	RR = 1.06–1.14(0.82–1.69).

The overall 95 % confidence intervals from all studies are indicated in parentheses. Since CPD is dominated by CVD (for which we use RR = 1.129), it follows that the RRs by the ACS used here are well within the range of estimates of epidemiological cohort studies, i.e. rather toward the lower end (except lung cancer), and that the RRs derived in different studies are quite consistent.

We carried out an uncertainty analysis assuming random errors, by propagating the quantified errors of all terms in Eq. (1), estimated from the 95 % confidence intervals (CI95), based on the following assumptions. From the CI95 for CPD reported by Krewski et al. (2009) we estimate an uncertainty range of ±27 % (applied to CVD), and for lung cancer (LC) of ±100 %. From the CI95 for respiratory disease (RD) reported by Jerrett et al. (2009) we estimate an uncertainty range of ±67 %. These uncertainties are multiplied with the 2σ standard deviations of the concentration response function (β), being 0.0061, 0.0064 and 0.0019 for CVD, LC and RD, respectively.

For the population data we apply an uncertainty range of ±2 %, considering that the UN Department of Economic and Social Affairs Population Division estimates the population data of a country like China uncertain by approximately this fraction (http://esa.un.org/unpd/wpp/). The uncertainties in the concentration changes of pollutants (ΔX) are represented by the model simulated annual 2σ standard deviations for each location (i.e. all model grid cells at the surface). For the baseline mortalities (y_o) we also apply the 2σ standard deviations, area weighted over all relevant locations (populated grid cells). The error propagation calculations account for the covariance between the different terms in Eq. (1).

The results are listed in Table 4, indicating that the uncertainty ranges are typically a factor of two or more for relatively small strata. In the larger strata the uncertainties are smaller, about ±5–50 % for lung cancer and cardiovascular mortality and up

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to ±10 % for respiratory mortality. The global averages appear to be rather robust with a statistical uncertainty range up to about ±5 %. The relatively small uncertainty ranges apply to the highest level of aggregation (large strata and globally), while at the country and megacity level the uncertainties are much larger, typically exceeding a factor of two. Note that this analysis does not account for non-representativeness of the concentration response functions used, e.g. applying them to developing countries, which causes additional uncertainty. Further, we have assumed that the effects only apply to the part of the population older than 30 yr, so that these estimates represent lower limits. Yet, the influence of this assumption is expected to be small because the baseline mortality rates at ages under 30 yr are relatively low (COMEAP, 2010).

5 Comparison to previous work

Ostro (2004) estimated premature mortality worldwide by particulate matter in the urban environment based on PM_{10} concentrations from monitoring stations and model-based estimates (PM_{10} is PM with an aerodynamic diameter smaller than 10 µm). This implicitly includes road dust, dust from construction sites and natural sources, not accounted for in the present work. The $PM_{2.5}$ levels used by Ostro (2004) were extrapolated from PM_{10} through scaling factors. The applied RR for CPD is 1.155, being higher than ours, and 1.232 for lung cancer, being much higher than ours and outside the range of recent studies (Sect. 4). Yet the global premature mortality by CPD was estimated at 1.07 million yr^{-1}, about half of our estimate. Since the difference cannot be attributed to the concentration response functions, it is probably due to the applied $PM_{2.5}$ concentrations for which unfortunately no detailed documentation is available (Ostro, 2004). Part of the discrepancy is also related to the focus by Ostro (2004) on the urban population.

Prüss-Üstün et al. (2008) composed country profiles of the environmental burden and disease for 192 of the WHO member states for the year 2002. They included the burden by outdoor air pollution through mean metropolitan PM_{10} measurements

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based on publicly available statistics. The study thus also focused on the urban population. It included respiratory mortality among children under five years in age and cardiopulmonary and lung cancer mortality among adults older than 30 yr. Since the CRF of respiratory mortality is much lower than that of CPD and lung cancer mortality together, we assume that the results by Prüss-Üstün et al. are dominated by the latter two categories. By comparing our country-level estimates for CVD and lung cancer mortality, we find that for small countries, dominated by urban populations, our results and the Prüss-Üstün et al. study agree closely. On the other hand, for large countries with a wide range of population and pollution conditions, including a large rural population, our estimates tend to be higher by an order of magnitude. This underscores the importance of including regions that are data-sparse, often not included in public statistics, and the need to account for both rural and urban environments.

Ezzati et al. (2002) and Cohen et al. (2005) investigated cardiovascular and lung cancer mortality for adults over 30 yr old and acute respiratory infection mortality of children under five for the year 2000. The estimated mortality was classified according to the 14 WHO sub-regions also used here. Both studies focused on the urban population and air pollution by $PM_{2.5}$, reporting nearly 800 thousand excess deaths in the year 2000. The difference with our work is largely explained by the strongly growing urban population between 2000 and 2005, and the fact that we also account for the non-urban environment. Furthermore, Ezzati et al. (2002) and Cohen et al. (2005) assume a $PM_{2.5}$ minimum of 7.5 $\mu g\,m^{-3}$ below which the pollution would not affect human health (also assumed by Ostro, 2004). We did not adopt such a threshold because our calculations for the pre-industrial atmosphere include natural aerosol concentrations, which are typically not much lower than 7.5 $\mu g\,m^{-3}$.

A comparison with the global results of Anenberg et al. (2010), who applied the same methodology as we did but for the year 2000, indicates some significant differences (up to 50 %); especially our YLL calculations appear to be systematically lower. Our estimate of global respiratory mortalities (773 thousand yr^{-1}) is about 10 % higher (our YLL of 5.2 million yr^{-1} is ~ 15 % lower). One difference is that we applied updated baseline

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mortality rates on the country level (when available), whereas Anenberg et al. (2010) integrated over six large regions. Our estimated mortality by cardiovascular disease (2 million yr^{-1}) is about 40 % lower than that by CPD reported by Anenberg et al. (2010) while our estimate for lung cancer (186 million yr^{-1}) is about 15 % lower. The combined years of life lost by $PM_{2.5}$ (~ 16 million yr^{-1}) is nearly 50 % lower than reported by Anenberg et al. (2010). The discrepancies are possibly also related to the much finer resolution of our model, so that urban population and air pollution data are represented more consistently.

6 Conclusions

Our model results corroborate that in most areas premature mortality rates by air pollution are dominated by the adverse impacts of $PM_{2.5}$, primarily by cardiovascular disease, while in some regions, e.g. in Northeastern and Central America and rural areas in South and East Asia, respiratory disease by O_3 can be of similar magnitude. Future studies are recommended to also address the role of mineral dust particles, which can reach high concentrations in the region between North Africa, the Middle East, South and East Asia (the "dust belt"). The present results suggest that anthropogenic $PM_{2.5}$ and O_3 near the surface cause the highest mortality in densely populated parts of Asia, with China and India contributing foremost on the country level.

The premature mortality rates by $PM_{2.5}$ are highest in urban regions such as the Pearl River area, Delhi, Kolkota, Jakarta, Shanghai, Dhaka, Beijing, Mumbai, the Szechuan area, Tokyo, Seoul and others in South and East Asia. In addition, relatively high mortality rates are found in some European cities such as London, Paris, Berlin, Moscow, the Po Valley in Northern Italy, the central part of the Netherlands, Belgium and the Ruhr area in Germany, and cities in North America such as Chicago, New York and Los Angeles. By considering per capita mortality, however, a somewhat different picture emerges, with urban and industrial areas in several eastern European countries ranking high (e.g. in Hungary, the Ukraine and Bulgaria). The highest

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premature respiratory mortality related to O_3 is found in China, India, the USA, Japan and Bangladesh, whereas the per capita mortality is topmost in Algeria, Macao and Taiwan.

Our model results indicate that globally the premature mortality rate due to $PM_{2.5}$ is about 2.2 million yr^{-1} (~ 0.08 % per capita yr^{-1}) and by O_3 nearly 0.8 million yr^{-1} (~ 0.03 % per capita yr^{-1}), equivalent to approximately 16 and 5 million yr^{-1} of life lost per annum, respectively. This refers to the year 2005. The regions with the highest mortality due to air pollution worldwide are the Western Pacific (~ 46 %), Southeast Asia (~ 25 %) and Europe (~ 13 %). Uncertainty analysis shows that these global estimates are rather robust. At the level of small countries and urban centers, the uncertainties are much larger, typically higher than a factor of two. Furthermore, non-representativeness of concentration response factors from epidemiological studies in the USA and Europe, applied to populations in other parts of the world, has not been accounted for, and region specific epidemiological studies are recommended.

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Table 1. Baseline mortality rate, mortality (in thousands and per million capita), and YLL by cardiovascular and lung cancer deaths related to anthropogenic $PM_{2.5}$ and respiratory mortality related to O_3, for the population ≥ 30 yr, calculated for the WHO mortality strata in 2005.

			$PM_{2.5}$								O_3			
			Cardiovascular mortality				Lung cancer				Respiratory mortality			
	Stratum	People ≥ 30 yr (millions)	Baseline mortality (% yr^{-1})	Persons (thousands)	Per 10^6 capita ≥ 30 yr	YLL (thousands)	Baseline mortality (% yr^{-1})	Persons (thousands)	Per 10^6 capita ≥ 30 yr	YLL (thousands)	Baseline mortality (% yr^{-1})	Persons (thousands)	Per 10^6 capita ≥ 30 yr	YLL (thousands)
Africa	Afr-D	99	0.54	23	232	207	0.01	0.5	5	5	0.31	26	256	274
	Afr-E	109	0.51	17	156	155	0.01	0.4	4	5	0.27	14	129	151
Americas	Amr-A	199	0.42	49	249	245	0.09	10	50	73	0.12	42	210	194
	Amr-B	202	0.42	15	73	103	0.03	1	5	9	0.14	21	101	124
	Amr-D	28	0.34	2	59	12	0.02	0.1	3	1	0.17	3	99	18
Southeast Asia	Sear-B	140	0.45	60	428	497	0.04	5	37	48	0.22	26	187	219
	Sear-D	524	0.58	519	990	4452	0.02	18	34	170	0.29	207	394	1781
Europe	Eur-A	264	0.55	121	460	505	0.07	18	67	136	0.12	46	174	180
	Eur-B	109	0.94	62	573	409	0.06	5	49	52	0.09	12	107	82
	Eur-C	140	1.45	93	669	666	0.06	4	30	42	0.10	15	106	144
Eastern Mediteranean	Emr-B	57	0.53	6	112	51	0.01	0.2	3	2	0.08	7	121	54
	Emr-D	1267	0.67	71	563	610	0.02	2	17	23	0.17	25	198	234
Western Pacific	Wpr-A	103	0.37	24	229	105	0.07	5	46	29	0.16	28	271	91
	Wpr-B	829	0.45	955	1152	6239	0.05	117	141	1072	0.23	303	366	1664
World		2930	0.70	2019	689	14 260	0.05	186	64	1665	0.16	773	264	5215

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Table 2. Top ten countries with the highest annual premature cardiovascular, lung cancer and respiratory mortality (total and per capita) for the population ≥ 30 yr old in 2005.

	$PM_{2.5}$								O_3			
	Cardiovascular mortality				Lung cancer				Respiratory mortality			
	Total (thousands)		% per capita		Total (thousands)		% per capita		Total (thousands)		% per capita	
1	China	898	China	0.13	China	108	China	0.02	China	273	Algeria	0.04
2	India	421	Bangladesh	0.12	India	14	Belgium	0.01	India	170	Macao	0.04
3	Bangladesh	65	Pakistan	0.12	USA.	9.1	Netherlands	0.01	USA	38	Taiwan	0.04
4	Pakistan	60	Czech Republic	0.10	Germany	4.5	Macao	0.01	Japan	27	Bangladesh	0.04
5	Russia	46	Hungary	0.10	Japan	4.5	Hungary	0.01	Bangladesh	23	Honk Kong	0.04
6	USA	46	Korea (Dem. Rep.)	0.10	Indonesia	3.8	Honk Kong	0.01	Indonesia	16	Korea (Dem. Rep.)	0.04
7	Indonesia	44	Ukraine	0.10	Viet Nam	3.6	Czech Republic	0.01	Pakistan	12	India	0.04
8	Germany	39	India	0.10	Korea	2.8	Poland	0.01	Nigeria	11	China	0.04
9	Viet Nam	30	Macao	0.10	France	2.6	Viet Nam	0.01	Viet Nam	10	Nepal	0.04
10	Ukraine	29	Bulgaria	0.10	UK	2.5	Korea	0.01	UK	9.5	Bhutan	0.03

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Table 3. Megacities and major population centers with highest mortality due to air pollution worldwide. Ranking in absolute numbers (highest YLL), and in parentheses the relative ranking according to the per capita mortality for individuals older than 30 yr.

	Megacity, population center	Population (million)	Population ≥ 30 yr (million)	$PM_{2.5}$ cardiovascular mortality (per year)	$PM_{2.5}$ lung cancer mortality (per year)	O_3 respiratory mortality (per year)	$PM_{2.5} + O_3$ mortality (per year)	YLL by $PM_{2.5} + O_3$ (per year)	Sum mortality per capita (% yr^{-1})	Sum mortality per capita ≥ 30 yr (% yr^{-1})
1	Pearl River area (7)	39.6	21.8	29 182	3530	9713	42 425	376 253	0.11	0.19
2	Delhi (3)	18.5	7.3	14 121	485	3188	17 794	153 136	0.10	0.24
3	Kolkata (6)	18.6	7.4	10 503	362	3499	14 365	123 637	0.08	0.19
4	Jakarta (9)	20.7	8.6	10 316	882	2463	13 662	114 637	0.07	0.16
5	Shanghai (5)	13.3	7.0	9975	1206	3338	14 520	94 528	0.11	0.20
6	Dhaka (8)	17.4	6.2	7953	274	2689	10 917	93 966	0.06	0.17
7	Beijing (2)	9.7	5.2	10 204	1228	2395	13 828	91 048	0.14	0.26
8	Mumbai (10)	16.7	6.6	6740	232	2694	9667	83 211	0.06	0.15
9	Moscow (12)	13.6	8.2	6930	299	822	8051	60 347	0.06	0.10
10	Tokyo (24)	28.5	19.5	5690	1135	6848	13 674	54 476	0.05	0.07
11	Seoul (25)	19.2	11.3	4994	1289	1019	7303	50 028	0.04	0.06
12	Szechuan area (4)	5.1	2.8	4758	574	1269	6602	43 307	0.13	0.24
13	Tianjin (1)	3.9	2.1	4262	512	1036	5813	38 237	0.15	0.28
14	Honk Kong (11)	6.3	4.0	3727	452	1726	5906	37 970	0.09	0.15
15	New York (20)	12.2	7.1	3141	616	1807	5565	28 536	0.05	0.08
16	Karachi (13)	10.4	3.4	2385	76	776	3238	28 517	0.03	0.09
17	Bangkok (19)	7.5	4.0	1961	168	1045	3175	26 590	0.04	0.08
18	Los Angeles (18)	10.6	6.2	2171	426	2520	5118	25 650	0.05	0.08
19	Istanbul (14)	9.5	4.1	2977	215	558	3751	25 531	0.04	0.09
20	Mexico City (28)	18.0	7.5	2021	134	1516	3673	24 531	0.02	0.05

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Table 4. Mortality (in thousands) and the range (in percent) according to the 95 % confidence interval (CI95) by cardiovascular and lung cancer deaths due to anthropogenic $PM_{2.5}$ and respiratory mortality due to O_3, for the population ≥ 30 yr, calculated for the WHO mortality strata in 2005. The ranges have been computed at the highest grid resolution available and were subsequently propagated and aggregated to the level of strata, including the covariance between the terms of Eq. (1), which leads to a decrease of the CI95 range at increasing levels of aggregation.

		$PM_{2.5}$				O_3	
		Cardiovascular mortality		Lung cancer		Respiratory mortality	
	Stratum	Persons (thousands)	CI95 (±%)	Persons (thousands)	CI95 (±%)	Persons (thousands)	CI95 (±%)
Africa	Afr-D	23	109	0.5	118	25	4
	Afr-E	17	30	0.4	54	14	6
Americas	Amr-A	50	16	10	9	42	6
	Amr-B	15	39	1	41	20	9
	Amr-D	2	124	0.1	169	3	21
Southeast Asia	Sear-B	60	19	5	16	26	8
	Sear-D	519	5	18	9	207	2
Europe	Eur-A	121	11	18	9	46	5
	Eur-B	63	16	5	12	12	8
	Eur-C	93	18	4	19	15	12
Eastern Mediteranean	Emr-B	6	149	0.2	170	7	13
	Emr-D	71	129	2	67	25	8
Western Pacific	Wpr-A	24	45	5	28	28	11
	Wpr-B	955	4	116	3	303	2
World		2019	5	186	3	773	1

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Table A1. Abbreviations and acronyms.

ACS	American Cancer Society
BMR	Baseline Mortality Rate
CI	Confidence Interval
CIESIN	Columbia University Center for International Earth Science Information Network
CRF	Concentration Response Function
CPD	Cardiopulmonary Disease
CVD	Cardiovascular Disease
DALY	Disability-Adjusted Life Years
ECHAM	European Centre Model Hamburg
EMAC	ECHAM/MESSy Atmospheric Chemistry MESSy Modular Earth Submodel System
LC	Lung Cancer
Mort	Annual mortality
$PM_{2.5}$	Particulate Matter with an aerodynamic diameter smaller than 2.5 µm
PM_{10}	PM with an aerodynamic diameter smaller than 10 µm
Pop	Total population with an age of $\geq$ 30 yr
RD	Respiratory Disease
RR	Relative Risk
UNDES	United Nations Department of Economic and Social Affairs
UNPD	United Nations Population Division
WHO	World Health Organization
YLD	Years Lost due to Disability
YLL	Years of Life Lost

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Table A2. WHO regions, mortality strata, child and adult mortality characteristics, and the countries and territories included.

Region	Stratum	Child mortality	Adult mortality	Countries and territories within stratum
Africa	Afr-D	High	High	Algeria, Angola, Benin, Burkina Faso, Cameroon, Cape Verde, Chad, Comoros, Equatorial Guinea, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Madagascar, Mali, Mauritania, Mauritius, Mayotte, Niger, Nigeria, Reunion, Saint Helena, Sao Tome and Principe, Senegal, Seychelles, Sierra Leone, Togo
	Afr-E	High	Very high	Botswana, Burundi, Central African Republic, Congo, Côte d'Ivoire, Democratic Republic of the Congo, Eritrea, Ethiopia, Kenya, Lesotho, Malawi, Mozambique, Namibia, Rwanda, South Africa, Swaziland, Uganda, United Republic of Tanzania, Zambia, Zimbabwe
Americas	Amr-A	Very low	Very low	Canada, Cuba, Greenland, Saint Pierre and Miquelon, United States of America
	Amr-B	Low	Low	Anguilla, Antigua and Barbuda, Argentina, Aruba, Bahamas, Barbados, Belize, Bermuda, Brazil, British Virgin Islands, Cayman Islands, Chile, Colombia, Costa Rica, Dominica, Dominican Republic, El Salvador, Falkland Islands, French Guiana, Grenada, Guadeloupe, Guyana, Honduras, Jamaica, Martinique, Mexico, Montserrat, Netherland Antilles, Panama, Paraguay, Puerto Rico, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Suriname, Trinidad and Tobago, Turks and Caicos Islands, United States Virgin Islands, Uruguay, Bolivarian Republic of Venezuela
	Amr-D	High	High	Bolivia, Ecuador, Guatemala, Haiti, Nicaragua, Peru
Southeast Asia	Sear-B	Low	Low	Indonesia, Sri Lanka, Thailand
	Sear-D	High	High	Bangladesh, Bhutan, Democratic People's Republic of Korea, East Timor, India, Maldives, Myanmar, Nepal
Europe	Eur-A	Very low	Very low	Andorra, Austria, Belgium, Croatia, Cyprus, Czech Republic, Denmark, Faeroe Islands, Finland, France, Germany, Gibraltar, Greece, Guernsey, Iceland, Ireland, Isle of Man, Israel, Italy, Jersey, Liechtenstein, Luxembourg, Malta, Monaco, Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Svalbard, Sweden, Switzerland, UK
	Eur-B	Low	Low	Albania, Armenia, Azerbaijan, Bosnia and Herzegovina, Bulgaria, Georgia, Kyrgyzstan, Poland, Romania, Serbia and Montenegro, Slovakia, Tajikistan, The former Yugoslav Republic of Macedonia, Turkey, Turkmenistan, Uzbekistan
	Eur-C	Low	High	Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, Russia, Ukraine
Eastern Mediterranean	Emr-B	Low	Low	Bahrain, Iran, Jordan, Kuwait, Lebanon, Libyan Arab Jamahiriya, Oman, Qatar, Saudi Arabia, Syrian Arab Republic, Tunisia, United Arab Emirates
	Emr-D	High	High	Afghanistan, Djibouti, Egypt, Iraq, Morocco, Palestinian Territories, Pakistan, Somalia, Sudan, Yemen
Western Pacific	Wpr-A	Very low	Very low	Australia, Brunei Darussalam, Japan, New Zealand, Singapore
	Wpr-B	Low	Low	Cambodia, China, Cook Islands, Fiji, French Polynesia, Guam, Hong Kong, Kiribati, Lao People's Democratic Republic, Macao, Malaysia, Marshall Islands, Pitcairn, Fed. States of Micronesia, Mongolia, Nauru, New Caledonia, Niue, Norfolk Island, Northern Mariana Islands, Palau, Papua New Guinea, Philippines, Republic of Korea, Samoa, Solomon Islands, Taiwan, Tokelau, Tonga, Tuvalu, Vanuatu, Viet Nam, Wallis and Futuna

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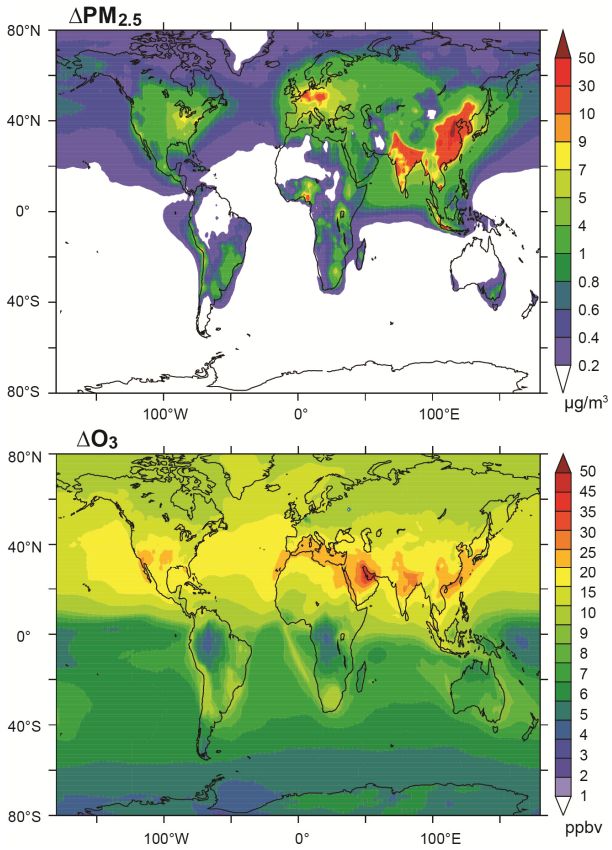

Fig. 1. Model calculated annual average increases of anthropogenic $PM_{2.5}$ and O_3 from the preindustrial era until 2005.

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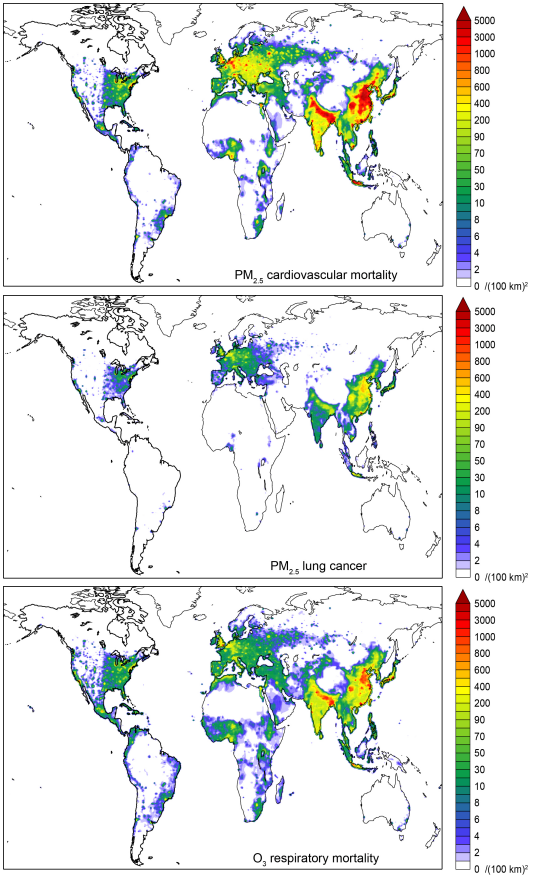

Fig. 2. Global premature mortality by cardiovascular, lung cancer and respiratory disease (individuals per 100 × 100 km^2) due to $PM_{2.5}$ and O_3 for the population ≥ 30 yr in 2005.

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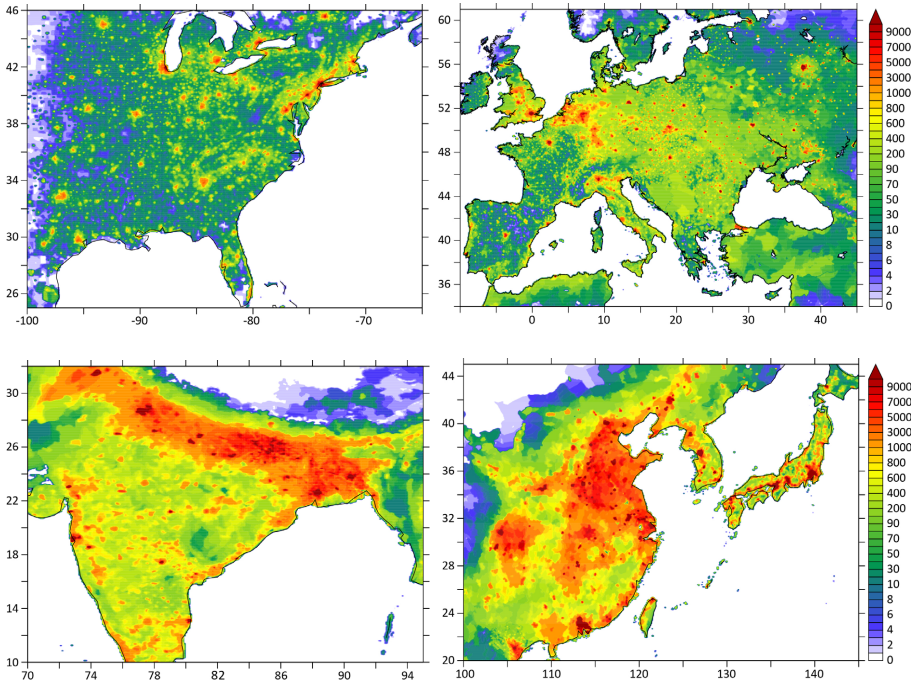

Fig. 3. Sum of cardiovascular, lung cancer and respiratory mortality related to $PM_{2.5}$ and O_3 in the Eastern USA, Europe, Southern and Eastern Asia in 2005 (individuals per 100 × 100 km^2). The level of detail in these figures represents a resolution of ~ 10 km based on the population data (~ 4 km) and atmospheric composition model output (~ 100 km).

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