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Implementing the US air quality standard for PM_{2.5} worldwide can prevent millions of premature deaths per year

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Abstract

Background: Air pollution by fine aerosol particles is among the leading causes of poor health and premature mortality worldwide. The growing awareness of this issue has led several countries to implement air pollution legislation. However, populations in large parts of the world are still exposed to high levels of ambient particulate pollution. The main aim of this work is to evaluate the potential impact of implementing current air quality standards for fine particulate matter (PM_{2.5}) in the European Union (EU), United States (US) and other countries where PM_{2.5} levels are high.

Methods: We use a high-resolution global atmospheric chemistry model combined with epidemiological concentration response functions to investigate premature mortality attributable to $PM_{2.5}$ in adults ≥ 30 years and children < 5 years. We perform sensitivity studies to estimate the reductions in mortality that could be achieved if the $PM_{2.5}$ air quality standards of the EU and US and other national standards would be implemented worldwide.

Results: We estimate the global premature mortality by $PM_{2.5}$ at 3.15 million/year in 2010. China is the leading country with about 1.33 million, followed by India with 575 thousand and Pakistan with 105 thousand per year. For the 28 EU member states we estimate 173 thousand and for the United States 52 thousand premature deaths in 2010. Based on sensitivity analysis, applying worldwide the EU annual mean standard of 25 μ g/m³ for $PM_{2.5}$ could reduce global premature mortality due to $PM_{2.5}$ exposure by 17 %; while within the EU the effect is negligible. With the 2012 revised US standard of 12 μ g/m³ premature mortality by $PM_{2.5}$ could drop by 46 % worldwide; 4 % in the US and 20 % in the EU, 69 % in China, 49 % in India and 36 % in Pakistan. These estimates take into consideration that about 22 % of the global $PM_{2.5}$ related mortality cannot be avoided due to the contribution of natural $PM_{2.5}$ sources, mainly airborne desert dust and $PM_{2.5}$ from wild fires.

Conclusions: Our results reflect the need to adopt stricter limits for annual mean $PM_{2.5}$ levels globally, like the US standard of 12 μ g/m³ or an even lower limit to substantially reduce premature mortality in most of the world.

Keywords: Air quality, Outdoor air pollution, Fine particulate matter, PM_{2.5} standards, Premature mortality

Abbreviations: AF, Attributable fraction; ALRI, Acute lower respiratory infection; AQG, Air quality guidelines; CEV, Cerebrovascular disease; CI, Confidence interval; CIESIN, Columbia University Center for International Earth Science Information Network; COPD, Chronic obstructive pulmonary disease; ECHAM, European Centre Model Hamburg; EMAC, ECHAM/MESSy Atmospheric Chemistry, MESSy Modular Earth Submodel System; EPA, Environmental Protection Agency; GBD, Global Burden of Disease; GDP, Gross Domestic Product; IHD, Ischemic heart disease; LC, Lung cancer; PM_{2.5}, Particulate Matter with an aerodynamic diameter smaller than 2.5 μ m; Pop, Total population with an age of <5 years and ≥30 year; RR, Relative risk; WHO, World Health Organization; y₀, Baseline mortality rate; Δ Mort, Annual premature mortality

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Background

Outdoor air pollution by fine particles ranks among the top ten global health risk factors that can lead to premature mortality [1]. Most of these particles originate from combustion engines, power plants, industry, household energy use, agriculture, biomass burning and natural sources like desert dust.

Epidemiological cohort studies, mainly conducted in the United States and Europe, have shown that the longterm exposure to PM_{2.5} (particles with an aerodynamic diameter less than 2.5 µm) is associated with increased mortality from cardiovascular, respiratory diseases and lung cancer [1-7]. It has been estimated that 70-80 % of premature deaths attributable to outdoor air pollution are due to ischemic heart disease and strokes, 15-25 % to chronic obstructive pulmonary disease and acute lower respiratory infections and about 5-6 % to lung cancer [8–10]. Fine particulates can cause health impacts even at very low concentrations [11–14]. Previously, no concentration level has been defined below which health damage can be fully prevented while the Global Burden of Disease (GBD) applies a PM_{2.5} threshold of 7.3 ± $1.5 \, \mu g/m^3 \, [1].$

The World Health Organization (WHO) ambient air quality guidelines suggest an annual mean PM2.5 concentration limit of 10 μg/m³ and 25 μg/m³ for the 24hourly mean [11]. Populations in large parts of the world, especially in East and Southeast Asia and the Middle East, are exposed to levels of fine particulate pollution that far exceed the WHO guidelines. WHO reported that in 2012 outdoor air pollution was responsible for the deaths of 3.7 million people [9]. WHO also emphasizes that indoor and outdoor air pollution combined are among the largest health risk worldwide, both being of similar magnitude. Air pollution is considered the number one environmental cause of premature death in the European Union (EU) [15]. Air pollution additionally impacts the quality of life by causing nonlethal chronic respiratory problems including asthma. It causes loss of working days and high healthcare costs, affects climate and perhaps weather, harms ecosystems, limits visibility and damages monuments and buildings. The direct costs to the European Union society from air pollution, including damage to crops and buildings, are estimated at about €23 billion per year [15, 16].

In the United States (US), substantial reductions of particulate pollution have been achieved in the recent past. The Environmental Protection Agency (EPA) in December 2012 took further steps to reduce particle pollution by tightening the annual National Ambient Air Quality Standard for fine particles (PM $_{2.5}$) from 15 to 12 $\mu g/m^3$. Benefits of the US clean air act for 1970–1990 were estimated at a central value of \$22.2 trillion compared to the implementation costs of \$0.52 trillion

[17, 18]. Many other countries have not yet enforced regulations to control PM_{2.5}. Estimates of mortality and morbidity attributable to outdoor air pollution are useful to justify air quality control policies and help improve public health. The aim of this work is to evaluate the implementation of recent air quality standards for PM2.5 in the EU, US and other countries worldwide and to estimate the public health gains that could be expected if EU or US standards for long term exposure were adopted and enforced internationally. In Table 1 and section 4 we present information on the current regulations for annual mean PM25 concentrations that have been adopted in the EU, US and other countries. We also present proposed targets that have not been officially adopted, mainly in several Asian countries which contribute strongly to high PM_{2.5} levels and related mortality, and finally the World Health Organization Air Quality Guideline for annual mean PM_{2.5} levels.

Methods

Estimation of PM_{2.5} related mortality

To estimate premature mortality attributable to PM_{2.5} we used the following health impact function

$$\Delta Mort = y_o \cdot AF \cdot Pop \tag{1}$$

Where y_o is the baseline mortality rate [8, 19, 20] of the population (Pop) exposed to air pollution. We used mortality data from the World Health Organization [21] for ischemic heart disease (IHD), cerebrovascular disease (CEV), chronic obstructive pulmonary disease (COPD), and lung cancer (LC) for the population above 30 year (\geq 30 year), and for acute lower respiration infection (ALRI) for children below 5 years (<5 years). We focused on the above detailed health outcomes to be consistent with the Global Burden of Disease 2010 study [1].

The corresponding population data have been obtained from the Columbia University Center for International Earth Science Information Network [22], available at high resolution (about $5 \times 5 \text{ km}^2$).

AF is the fraction of the disease burden attributable to the risk factor (here $PM_{2.5}$). The attributed fraction is defined as

$$AF = (RR - 1)/RR \tag{2}$$

RR is the relative risk of certain health impacts of the population exposed to outdoor $PM_{2.5}$ air pollution. To estimate the global burden of disease attributable to $PM_{2.5}$ we follow the same methodology as Lelieveld et al. [8], and apply the integrated health risk function from Burnett et al. [23], also used by Lim et al. [1] for the GBD in 2010.

$$RR = 1 + a\{1 - exp[-b(X - X_o)^p]\}$$
(3)

Table 1 Summary of PM_{2.5} standards in selected countries (in μg/m³)

Countries/Unions	PM _{2.5} annual mean (µgm ⁻³)	Status	Source
European Union	25	Adopted	EU, Air Quality Directive, 2008/50/EC
United States	12	Adopted	EPA Regulatory Actions, 2014
Canada	10	Adopted	Canadian Ambient Air Quality Standards, 2014
Colombia	25	Adopted	Green, J. and Sánchez S., 2012
Chile	20	Adopted	Green, J. and Sánchez S., 2012
Equador	15	Adopted	Green, J. and Sánchez S., 2012
El Salvador	15	Adopted	Green, J. and Sánchez S., 2012
Mexico	15	Adopted	Green, J. and Sánchez S., 2012
Puerto Rico	15	Adopted	Green, J. and Sánchez S., 2012
Rep of Dominica	15	Adopted	Green, J. and Sánchez S., 2012
Argentina (Buenos Aires)	15	Adopted	Green, J. and Sánchez S., 2012
Bolivia (La Paz)	10	Adopted	Green, J. and Sánchez S., 2012
Australia	8	Adopted	Australian Gov., Dep. of the Environment and Heritage
China (Beijing)	35	Proposed	CAI-Asia, Particulate Matter Standards in Asia, 2010
India	40	Proposed	CAI-Asia, Particulate Matter Standards in Asia, 2010
Japan	15	Proposed	Environmental Quality Standards in Japan, 2014
Pakistan	15	Proposed	CAI-Asia, Particulate Matter Standards in Asia, 2010
Bangladesh	15	Proposed	CAI-Asia, Particulate Matter Standards in Asia, 2010
Saudi Arabia	15	Proposed	Kingdom of Saudi Arabia: National Env. Standard, 2014
WHO	10	Guideline	World Health Organization Air Quality Guidelines 2005

We refer to Burnett et al. [23] and Lelieveld et al. [8] for details on the exposure response models for the five disease categories. X is the annual mean PM_{2.5} concentration in 2010. We used the EMAC global atmospheric chemistry - general circulation model to simulate annual mean PM_{2.5} concentrations [24] (Fig. 1). EMAC comprises sub-models that represent tropospheric and lower stratospheric processes and their interaction with oceans, land and human influences [24-27]. We obtained results for the year 2010, applying monthly varying emissions from EDGAR - the Emission Database for Global Atmospheric Research [26]. We apply the same methodology as Lelieveld et al. [8] to estimate the premature mortality in 2010, combining all aerosol types that contribute to PM_{2.5}, and using the same lower limits as Burnett et al. (around 7.3 µg/m³ depending on the disease category) for the background concentration X_o below which no impact is assumed [23]. To have a measure of the uncertainty range for the mortality estimations, we mainly use the lower and upper bound of RR to calculate the minimum and maximum AF and mortality.

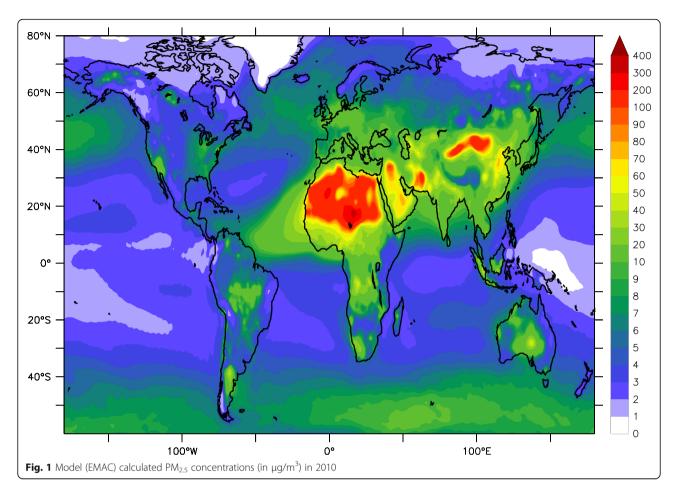
Details about the EMAC atmospheric chemistry model, comparison of the output to in situ and remote sensing observations, and output robustness is available in Jöckel et al. [25], Lelieveld et al. [8, 28], Pozzer et al. [24, 26, 27] and references therein.

To assess the impact of applying air quality standards by the EU, US and other countries for $PM_{2.5}$ pollution we performed sensitivity calculations where we set these standards as upper limit for the variable X in equation 3, thus assuming they are strictly implemented.

PM_{2.5} standards and guidelines

European Union: The directive on ambient air quality and cleaner air for Europe [29] defines "objectives for ambient air quality designed to avoid, prevent or reduce harmful effects on human health and the environment as a whole". Under this directive EU member states are required to reduce the exposure to PM_{2,5} in urban areas on average by 20 % in 2020 relative to 2010 levels. The states are obliged to bring exposure levels below 20 µg/m³ by 2015 in these areas. Throughout their territory member states will need to respect the annual mean PM_{2.5} limit value of 25 μg/m³. This value must have been achieved by 2015. In the air quality directive a PM_{2.5} reference level of $25 \mu g/m^3$ is set, initially as target value to be met by 2010 and as limit value to be met by 2015. In a second stage a lower limit of 20 μ g/m³ must be met by 2020. Information from PM_{2.5} monitoring stations is still limited and needs to be extended to verify full implementation of the directive.

United States: In December 2012, the US Environmental Protection Agency (EPA) tightened the air quality



standards for $PM_{2.5}$ to improve air quality and public health. The primary annual mean $PM_{2.5}$ concentration limit was lowered from 15 $\mu g/m^3$ to 12 $\mu g/m^3$. EPA has issued a number of regulations to meet the revised standard. EPA estimates that meeting the annual fine particle standard of 12 $\mu g/m^3$ will provide health benefits at an economic value estimated at \$4 to \$9.1 billion per year in 2020, which translates into a return of \$12 to \$171 for every dollar invested in pollution reduction. Estimated annual costs of implementing the standard are \$53 to \$350 million [30].

Canada: On May 2013, the Canadian Environmental Protection Act established for the first time a long-term annual target for $PM_{2.5}$ of 10 $\mu g/m^3$ to be met by the year 2015, and a more stringent value of 8.8 $\mu g/m^3$ to be met by 2020 [31].

Australia: On June 1998, the National Environment Protection Council (NEPC) in Australia set national standards for annual mean $PM_{2.5}$ to not exceed 8 $\mu g/m^3$, which is by far the strictest national limit worldwide. The standards should have been met by the year 2008 [32].

Other countries: We have conducted an internet search for information about regulations of PM_{2.5} in other countries with enhanced particulate pollution, and found that

for many countries in Asia, Africa and Latin America records and data are scarce. In Latin America only few countries have set national ambient air quality standards. Colombia adopted a limit of 25 μ g/m³ for annual mean PM_{2.5}. Chile set a level of 20 μ g/m³, while Ecuador, El Salvador, Mexico, Puerto Rico and the Dominican Republic have adopted a standard of 15 μ g/m³. Provinces in Argentina and Bolivia implement regulations based on their own standards. Buenos Aires set a value of 15 μ g/m³ annual mean PM_{2.5}, and La Paz 10 μ g/m³ [33].

The "Clean Air Initiative for Asia" [34] was established in 2001 as the premier air quality network for Asia by the Asian Development Bank, World Bank, and USAID. Its mission is to promote ways to improve air quality in Asian cities and provide information on air quality monitoring, status, and trends, and also on national air quality standards in Asian countries. While several Asian countries have adopted a standard for PM_{10} , more is needed in the development of a $PM_{2.5}$ standard. In China an upper annual mean $PM_{2.5}$ limit of 35 μ g/m³ is suggested for the Beijing municipality area and Hong Kong special administrative region (SAR). The reported annual mean $PM_{2.5}$ concentration in Beijing is 89.5 μ g/m³, far exceeding the national standard

(https://www.chinadialogue.net/blog/6686-Beijing-passes-law-to-curb-air-pollution/en). Zheng et al. (2014) [35] analyzed long-term measurement data in Central Beijing, indicating an annual mean concentration of about $100~\mu g/m^3$. In India an upper annual mean $PM_{2.5}$ limit of $40~\mu g/m^3$ has been proposed, which has not been formally adopted. Japan, Pakistan, Bangladesh and Saudi Arabia propose a limit of $15~\mu g/m^3$ [36–38]. For other countries with high $PM_{2.5}$ pollution and associated mortality, like Russia, Ukraine, Indonesia, Viet Nam, Japan, Thailand, Egypt, Turkey, Iran, Iraq, Nigeria, Sudan and Myanmar we could not find specific regulations.

World Health Organization Air Quality Guidelines (WHO AQG): The WHO guideline for long-term $PM_{2.5}$ exposure is an annual mean concentration of 10 $\mu g/m^3$. With this AQG WHO offers guidance in reducing the health impacts of air pollution, but they are neither standards nor legally binding criteria. Epidemiological studies have not identified thresholds below which adverse health effects do not occur, thus the guideline value cannot fully protect humans from health impacts [11, 39].

Results

We apply the exposure response model (Eq. 3) of Burnett et al. [23], to estimate the global and country level premature mortality due to CEV, IHC, COPD, and LC for the population \geq 30 year, and due to ALRI for children <5 years in 2010, related to the long-term exposure to PM_{2.5}. Consistent with Lelieveld et al. [8] for the year 2010 we estimate 3.15 million premature deaths

(95 % confidence interval (CI95): 1.52-4.60 million) by PM_{2.5} worldwide, due to CEV (1.31 million), IHD (1.08 million), COPD (374 thousand), LC (161 thousand) and ALRI (230 thousand). Figure 2 (top) highlights the hot spot locations in red with high rates of premature mortality due to PM_{2.5} in 2010. The countries with the highest estimated premature mortality are China (1.33 million; CI95: 0.64-1.94 million), India (575 thousand; CI95: 277-840 thousand) and Pakistan (105 thousand; CI95: 51-153 thousand). For the EU our estimate is about 173 thousand (CI95: 83-253 thousand) with Germany ranking first (34 thousand), followed by Italy (19 thousand), France (17 thousand), United Kingdom (15 thousand), Romania (15 thousand) and Poland (14 thousand). Other countries in Europe with high premature mortality are Russia (67 thousand) and Ukraine (51 thousand). The United States ranks 7th on the global list of premature mortality due to PM25 (Table 2) with about 52 thousand deaths in 2010 (CI95: 25-76 thousand). Table 2 shows the top 20 countries with highest PM_{2.5} related premature mortality in 2010, while Table 3 presents mortality data estimated for the 28 countries of the EU.

Our global estimate of premature mortality due to long term exposure to $PM_{2.5}$ (3.15M/year) agrees closely with the 3.22M/year estimate reported by the GBD study in 2010 [1] and the 3.24M/year estimate of Apte et al. [40]. Lelieveld et al. [28] estimated 2.2M/year for the global $PM_{2.5}$ related mortality for 2005, which is 30 % less than our current estimate. This difference can be

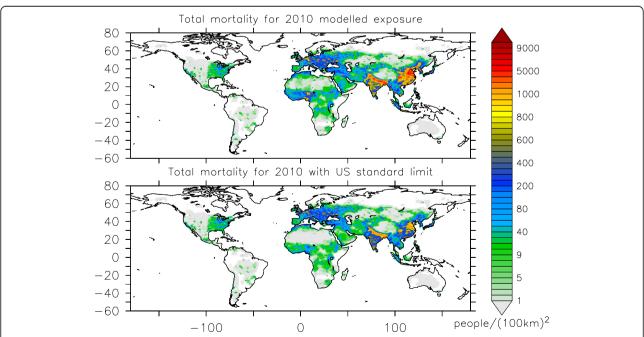


Fig. 2 PM_{2.5} related premature mortality for the population <5 and ≥30 years old (in deaths/area of $100 \times 100 \text{ km}^2$). *Top*: year 2010. *Bottom*: Implementing the US standard of 12 μ g/m³

Table 2 Top 20 countries with highest annual premature mortality attributed to PM_{2.5} in 2010 for the population <5 and ≥30 years old and the corresponding mortality after the implementation of the EU and US air quality standards

Country	Year 2010 deaths (×10 ³)	EU limit (25 µgm ⁻³) deaths (×10 ³)	US limit (12 µgm ⁻³) deaths (×10 ³)
China	1327	910 (31)	416 (69)
India	575	502 (13)	294 (49)
Pakistan ^a	105	84 (20)	67 (36)
Nigeria ^a	89	78 (12)	76 (15)
Bangladesh	85	76 (11)	38 (55)
Russia	67	67 (0)	66 (1)
USA	52	52 (0)	49 (6)
Indonesia	51	48 (6)	33 (35)
Ukraine	51	51 (0)	49 (4)
Viet Nam	43	36 (16)	18 (58)
Germany	34	34 (0)	26 (24)
Egypt ^a	34	33 (3)	33 (3)
Turkey	31	31 (0)	25 (19)
Iran ^a	25	24 (4)	22 (12)
Sudan ^a	24	24 (0)	24 (0)
Japan	24	24 (0)	21 (13)
Myanmar	21	21 (0)	14 (33)
Italy	19	19 (0)	15 (21)
Iraq ^a	19	19 (0)	19 (0)
Thailand	18	18 (0)	15 (17)
World	3155	2600 (17)	1712 (46)

In parenthesis the % reduction in premature mortality

explained mainly by the new integrated health risk function and concentration response factors that we apply here and in particular also that we account for both anthropogenic and natural sources for $PM_{2.5}$ in 2010, while Lelieveld et al. [28] accounted only for anthropogenic pollution in 2005. In addition, trends in $PM_{2.5}$ concentrations and populations caused a significant increase in air pollution related deaths in densely populated countries like China and India. Further, in previous work premature mortality due to respiratory disease was attributed to O_3 pollution, whereas more recently this has been subdivided into COPD by O_3 and $PM_{2.5}$. Hence the relative role of $PM_{2.5}$ has increased at the expense of O_3 in recent concentration exposure models.

In this work we also assess the contribution of natural sources of $PM_{2.5}$, like desert dust, biomass burning (i.e., wild fires) and sea salt to premature mortality. Our estimates indicate that natural sources cause about 692 thousand deaths in 2010 (22 % of the total global mortality attributed to $PM_{2.5}$). For the above estimations we assume that all $PM_{2.5}$ particles with different composition,

Table 3 Annual premature mortality attributed to $PM_{2.5}$ in 2010 for the population <5 and ≥30 years old in the EU member countries and the corresponding mortality after the implementation of the EU and US air quality standards

Country	Year 2010 deaths (×10 ³)	EU limit (25 µgm ⁻³) deaths (×10 ³)	US limit (12 µgm ⁻³) deaths (×10 ³)
Germany	34	34	26 (24)
Italy	19	19	15 (21)
France	17	17	15 (12)
United Kingdom	15	15	14 (7)
Romania	15	15	12 (20)
Poland	14	14	10 (29)
Hungary	7.1	7.1	5.4 (24)
Spain	6.5	6.5	6.4 (2)
Czech Republic	6.5	6.5	4.3 (34)
Netherlands	4.7	4.7	2.9 (38)
Bulgaria	4.7	4.7	3.4 (28)
Belgium	4.4	4.4	2.9 (34)
Greece	3.9	3.9	3.1 (21)
Slovakia	3.7	3.7	2.7 (27)
Austria	3.0	3.0	2.4 (20)
Croatia	2.2	2.2	1.8 (18)
Lithuania	2.1	2.1	2.1 (0)
Portugal	1.8	1.8	1.8 (0)
Denmark	1.6	1.6	1.5 (6)
Latvia	1.3	1.3	1.3 (0)
Sweden	0.928	0.928	0.897 (3)
Slovenia	0.685	0.685	0.517 (25)
Ireland	0.538	0.538	0.538 (0)
Estonia	0.498	0.498	0.498 (0)
Finland	0.445	0.445	0.445 (0)
Malta	0.164	0.164	0.118 (28)
Cyprus	0.142	0.142	0.132 (7)
Luxemburg	0.106	0.106	0.078 (26)
EU total	173	173	138 (20)

In parenthesis the % reduction in premature mortality

coming from different emission sources, are equally toxic. Based on a sensitivity study by Lelieveld et al. [8], who assumed that carbonaceous compounds are five times more toxic than inorganic and crustal compounds (e.g., dust) but maintaining the overall toxicity of total $PM_{2.5}$, the contribution of natural sources to total mortality significantly reduces to about 460 thousand deaths in 2010 (15 % of the total premature mortality). Table 4 shows the contribution of $PM_{2.5}$ from natural sources to the annual mortality for the countries that are mostly affected. In an earlier study we estimated premature mortality from cardiopulmonary diseases due to the long-term exposure to

^aIn these countries PM_{2.5} is dominated by airborne desert dust

Table 4 Top 20 countries with highest fraction of annual premature mortality attributed to natural sources of $PM_{2.5}$ over total $PM_{2.5}$ related mortality in 2010 for the population <5 and ≥30 years old

Country	$PM_{2.5}$ deaths (×10 ³)	Natural sources deaths (×10 ³)	Fraction (%
Sudan	24	24 (23)	100 (96)
Iraq	19	19 (18)	100 (95)
Saudi Arabia	14	14 (13)	100 (93)
Niger	13	13 (12)	100 (92)
Mali	9.4	9.3 (9.0)	99 (96)
Chad	7.4	7.3 (7.2)	99 (97)
Burkina Faso	9.3	9.1 (8.6)	98 (92)
Egypt	34	33 (31)	97 (91)
Cameroon	8.3	7.9 (7.2)	95 (87)
Ghana	9.3	8.7 (8.0)	93 (86)
D.R. Congo	15	13 (13)	87 (87)
Nigeria	89	76 (61)	85 (68)
Algeria	13	11 (11)	85 (85)
Morocco	13	11 (10)	85 (77)
Iran	25	21 (20)	84 (80)
Uzbekistan	11	7.8 (6.8)	71 (62)
Pakistan	105	65 (27)	62 (26)
India	575	94 (14)	16 (2)
Indonesia	51	8.2 (8.5)	16 (17)
China	1327	125 (46)	9 (3)
World	3155	692 (460)	22 (14)

In parentheses results of sensitivity calculations where carbonaceous aerosol compounds are assumed to be five times more toxic compared to inorganic and crustal compounds

desert dust to be about 402T/year in 2005 [19]. For this estimate we used a linear health response function, and instead of the annual mean dust concentration we applied median values due to the episodic nature of desert dust outbreaks. In the same study we estimated 622 thousand deaths when we account for annual mean dust concentration.

Sensitivity calculations

We present sensitivity calculations where we set different upper limits for the annual mean $PM_{2.5}$ concentration (X in equation 1) based on air quality standards and regulations. To estimate potential reductions in mortality rates we take into consideration the deaths that cannot be avoided after implementation of the $PM_{2.5}$ upper limits, due to the contribution of natural sources to the total $PM_{2.5}$ and therefore to mortality (mainly airborne desert dust and natural biomass burning).

First, based on Table 1, we assume that all current national regulations and proposed limits for annual mean $\rm PM_{2.5}$ are fully implemented. The estimated global premature mortality is reduced by 9 % from 3.15 million to 2.86 million per year [CI95: 1.38-4.17M]. The main contributors to this reduction are the standards implemented in China causing about 16 % less deaths, Pakistan with 34 % less deaths, Bangladesh with 41 % less deaths and the US with 4 % less deaths.

In a second sensitivity calculation we apply the annual mean PM_{2.5} concentration of 25 μg/m³ as an upper limit, following the EU standard. We estimate 2.60 million [CI95: 1.25-3.80M] premature deaths per year globally; 17 % less compared to our base estimate for 2010 (Table 2). The estimated total and country level mortality within the EU remains almost unchanged, indicating that this standard is mostly met already. Our model results suggest that in many EU countries the annual mean total and anthropogenic PM_{2.5} concentrations are well below this limit (e.g., Scandinavia, Western Europe), thus the annual mean $PM_{2.5}$ limit of 25 $\mu g/m^3$ is too high to make a difference, and a reduction of mortality attributable to $PM_{2.5}$ will require stricter limits. If the EU limit is applied in China, the main contributor to global PM_{2.5} related mortality, premature mortality could be reduced by 31 %, and about 417 thousand premature deaths would be avoided per year [CI95: 201-609T]. In India this limit could reduce premature mortality by about 13 % (73 T less deaths; CI95: 35-107T]. In a second stage the EU directive 2008/50/EC set a lower limit of 20 µg/m³ to be met by the year 2020. If we apply this limit in 2010 globally, mortality could be reduced by 26 % per year, still with a minor change within the EU. In China we estimate a reduction by 44 and 22 % in India (about 585 and 129 thousand less, respectively).

In a final sensitivity calculation we apply the limit of 12 μg/m³ based on the standard enacted in the US. According to our data, this limit could reduce the global premature mortality by 46 % compared to the 2010 estimates, from 3.15 [CI95: 1.52-4.60M] to 1.71 million deaths per year [CI95: 0.825-2.50M] (Table 2; Fig. 2, bottom), preventing about 1.44 million deaths/year. Our estimates indicate that in the United States the annual mortality could be reduced from 52 to 49 thousand per year [CI95: 24-72T], hence leading to a small improvement (by 4 %) in preventing mortality. If the EU would implement the 12 μ g/m³ limit, instead of the 25 μ g/m³, premature mortality could be reduced by 20 % to about 138 thousand per year [CI95: 66-201T], which is a considerable change; about 8.6 thousand deaths per year would be avoided in Germany, 4.1 thousand in Italy, 2.4 thousand in France, 1.2 thousand in the United Kingdom, 3.0 thousand in Romania, 4.3 thousand in Poland, 1.7 in Hungary, 2.2 in Czech Republic and 1.8 in Netherlands (Table 3). If the relatively strict US limit of 12 μg/m⁻³ would be applied in China, premature mortality could be

reduced by 69 %, and about 911 thousand premature deaths would be avoided per year [CI95: 0.440-1.33M]. In India the implementation of the US upper limit concentration could reduce premature mortality by about 49 % and about 281 thousand deaths would be avoided per year [CI95: 136-411T]. In Pakistan and Bangladesh, he 3rd and 5th countries in the global ranking of 2010 PM_{2.5} associated mortality, the stricter US limit could reduce premature mortality by 36 % (about 38 thousand less deaths per vear [CI95: 18-55T]) and 55 % (about 47 thousands less premature deaths per year [CI95: 23-69T]), respectively. Therefore, implementing the stricter US limit could make a significant difference (Table 2). In Nigeria, which is the 4th ranking country in 2010 with an estimated 89 thousand deaths per year, PM25 is overwhelmed by natural sources mainly from Saharan desert dust, which contributes about 85 % to the total PM25 related mortality causing about 76 thousand deaths. The implementation of the US limit could hence only reduce mortality by 15 % (about 12 thousand less deaths per year [CI95: 6.1-18T]). Similarly, natural sources contribute strongly to PM_{2.5} and therefore to mortality in other countries mainly around the dust belt, an area that extends from North Africa across the Middle East and South Asia to East Asia (Table 4). For these countries it is not possible to meet the strict US limit, not even the EU limit, as high desert dust concentrations are dominant in large areas where the annual mean concentrations typically range from 20 μg/m³ to 200 μ g/m³.

Based on the $PM_{2.5}$ regulations and proposed standards listed in Table 1, Fig. 3 summarizes the global premature mortality estimations when we apply the 8, 10, 12, 15, 20, 25, 30, 35 and 40 $\mu g/m^3$ annual mean $PM_{2.5}$ upper limit concentrations and the 2010 levels. This graphical representation illustrates that the reduction of

mortality rates is more sensitive to lower standards (e.g., <20 $\mu g/m^3$) compared to higher standards. The 12 $\mu g/m^3$ limit would reduce global mortality by 15 % compared to the 15 $\mu g/m^3$ limit, and by 27 % compared to the 20 $\mu g/m^3$ limit, while a limit tightening from 35 to 25 $\mu g/m^3$ would decrease global premature mortality by 10 %. We reiterate that to perform our sensitivity calculations we take into consideration that mortality caused from natural sources of $PM_{2.5}$ cannot be controlled by air quality regulations. Our analysis shows that the relatively strong global response to $PM_{2.5}$ reductions towards lower limits is mainly caused by the greater number of highly populated areas that would benefit from air quality control measures at these relatively low concentration levels.

Table 2 summarizes the results of our sensitivity calculations for the top 20 countries with highest PM_{2.5} mortality in 2010 and how mortality would change when applying the current EU and US air quality standards as upper limits. Table 3 presents the same information for the 28 EU member countries. Our results contribute to the body of evidence suggesting the need to adopt stricter limits for annual mean PM25 levels, like the US limit of 12 µg/m³ or even a lower limit to substantially reduce premature mortality in most of the world, while in strongly polluted regions like South and East Asia essentially any PM_{2.5} reduction can significantly reduce premature mortality. We reiterate that there is no strong evidence for a "safe" PM_{2,5} concentration threshold below which no health risk can be assumed (we have applied around 7.3 μ g/m³ depending on the disease category).

Discussion

In this work we used the integrated exposure response function (IER) of Burnett et al [23] to estimate the number of premature deaths due to PM_{2.5} air pollution

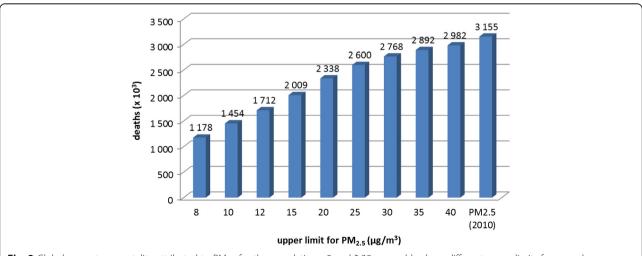


Fig. 3 Global premature mortality attributed to $PM_{2.5}$ for the population <5 and ≥30 years old, where different upper limits for annual mean $PM_{2.5}$ are applied. The right column indicates mortality in 2010

induced CEV, COPD, IHD, LC (for adults ≥30 year) and ALRI (for children <5 years). The IER model is a superior predictor of RR compared to others previously used in burden assessments, to more realistically accounts for health effects at very high PM_{2.5} concentrations [23]. This is particularly relevant for regions with very high pollution levels like East and South East Asia. As we follow the method of Lelieveld et al. [8], based on Burnet et al [23] and the Global Burden of Disease - GBD 2010 [1] we also apply their uncertainty calculations and adopt their 95 % confidence interval (CI95) for PM_{2.5} related mortality. The confidence interval represents statistical uncertainty of the parameters used in the concentration response function. In previous work we derived statistical uncertainties by propagating the quantified random errors of all terms in equation 1, estimated from the 95 % confidence intervals (CI95). The uncertainties in the PM_{2.5} calculations were represented by the model simulated annual 2σ standard deviations for all model grid cells at the surface [28]. The quantified errors showed that the global mortality estimates are quite robust with an uncertainty up to about ±5 % for annual PM_{2.5} induced mortality, while at the country level the uncertainties are much larger. For uncertainty analyses and sensitivity calculations that address the shape of the health impact functions and concentration thresholds (Xo) we refer to analyses by Lelieveld et al. [8, 28], Burnett et al. [23] and Giannadaki et al. [19]. These issues have been also discussed by expert panels [41-44]. The existence of "safe" PM_{2.5} concentration thresholds below which no health effects occur is considered ambiguous. Scientific uncertainty about the relative toxicity of particles emitted from different source categories is one of the major weaknesses in our ability to understand the relative contributions of each source to the PM_{2.5} related mortality [45]. Studies by the Health Effect Institute suggest that certain source classes (e.g., coal combustion and traffic) should be given priority in regulation and that there is less evidence that particles from other source classes (e.g., biomass burning and natural emissions of crustal materials) increase mortality risk [46]. However, a set of usable coefficients for PM_{2.5} compounds from different sources is not available in the published literature. Lelieveld et al. [8], motivated by the reports from expert judgment studies [42-44], performed sensitivity calculations assuming that the toxicity of carbonaceous particles is five times that of inorganic and crustal compounds, maintaining the average toxicity of PM_{2.5}. The expert studies indicate that aspects of the methodology and representativeness are likely to lead to several fold larger uncertainty than indicated by CI95, corroborated by the results of the sensitivity calculations on differential toxicity. While aerosol compounds such as heavy metals, soot and certain organic substances are

likely to be more toxic than mineral dust and inorganic salts, they form a mixture within $PM_{2.5}$ and cannot be treated separately based on epidemiological cohort studies. Therefore, the CI95 mentioned above for the health effects of the long-term exposure to $PM_{2.5}$ should be considered as a lower limit of the overall uncertainty.

Conclusions

We estimated the PM_{2.5} related premature mortality in 2010 at 3.15 million worldwide, with China ranking highest, followed by India, Pakistan, Nigeria and Bangladesh. For the EU our estimate for 2010 is 173 thousand premature deaths, and 52 thousand in the US. We performed sensitivity calculations to assess the impact of applying PM_{2.5} upper limits based on air quality standards in the EU and US, and other nationally adopted or proposed standards for annual mean PM_{2.5} pollution. Our results show that even small changes at the lower standards of annual mean PM_{2.5} concentrations could have a significant impact on mortality rates. This results from the fact that at low PM_{2.5} levels many relatively populous areas would profit from air quality improvements. Our findings underscore the large positive impact on human health by implementing the US air quality standard of 12 µg/m³ for annual mean PM_{2.5}. Finally, we estimated the impact on mortality due to PM_{2.5} from natural sources, mainly desert dust and wild fires, which to date represents a challenge to public health in the countries in and around the dust belt. For these countries it will not be possible to meet the US and EU standards.

Additional file

Additional file 1: Mortality calculations: Main cases and Sensitivity scenarios. (ZIP 56740 kb)

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Availability of data and materials

The datasets supporting the conclusions of this article are included within the article and its Additional file 1.

Authors' contributions

DG and JL planned the research, AP performed the model calculations, DG analysed the results, and DG, JL and AP wrote the paper. All authors contributed to the manuscript. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

Consent for publication

Not applicable

Ethics approval and consent to participate

Not applicable.

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