LETTER

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The contribution of outdoor air pollution sources to premature mortality on a global scale

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Assessment of the global burden of disease is based on epidemiological cohort studies that connect premature mortality to a wide range of causes[1–5], including the long-term health impacts of ozone and fine particulate matter with a diameter smaller than 2.5 micrometres ($PM_{2.5}$)[3–9]. It has proved difficult to quantify premature mortality related to air pollution, notably in regions where air quality is not monitored, and also because the toxicity of particles from various sources may vary[10]. Here we use a global atmospheric chemistry model to investigate the link between premature mortality and seven emission source categories in urban and rural environments. In accord with the global burden of disease for 2010 (ref. 5), we calculate that outdoor air pollution, mostly by $PM_{2.5}$, leads to 3.3 (95 per cent confidence interval 1.61–4.81) million premature deaths per year worldwide, predominantly in Asia. We primarily assume that all particles are equally toxic[5], but also include a sensitivity study that accounts for differential toxicity. We find that emissions from residential energy use such as heating and cooking, prevalent in India and China, have the largest impact on premature mortality globally, being even more dominant if carbonaceous particles are assumed to be most toxic. Whereas in much of the USA and in a few other countries emissions from traffic and power generation are important, in eastern USA, Europe, Russia and East Asia agricultural emissions make the largest relative contribution to $PM_{2.5}$, with the estimate of overall health impact depending on assumptions regarding particle toxicity. Model projections based on a business-as-usual emission scenario indicate that the contribution of outdoor air pollution to premature mortality could double by 2050.

Air pollution is associated with many health impacts, including chronic obstructive pulmonary disease (COPD) linked to enhanced ozone (O_3), and acute lower respiratory illness (ALRI), cerebrovascular disease (CEV), ischaemic heart disease (IHD), COPD and lung cancer (LC) linked to $PM_{2.5}$ (ref. 8). Many previous studies have been based on air quality measurements, largely focusing on urban pollution[3,4,11–14]. Atmospheric chemistry and transport models have been used to account for other environments, including those for which no measurement data are available[15–22].

Recently, enhanced resolution regional and global models and satellite data have been applied to improve estimates of $PM_{2.5}$ and O_3 concentrations and their impact on air quality[19–24]. Here we present results obtained with an atmospheric chemistry–general circulation model, applied at high resolution to compute global air quality changes, combined with population data, country-level health statistics and pollution exposure response functions (Methods). Our calculations of air pollution related mortality are based on the method of the global burden of disease (GBD) for 2010 (ref. 5), applying improved exposure response functions that more realistically account for health effects at very high $PM_{2.5}$ concentrations compared to former assessments[8]. This is particularly relevant for some parts of the world where air pollution has increased nearly unabated and for future scenarios that project the continued growth of emissions. Following the GBD[5] we also include desert dust (which is largely natural) with $PM_{2.5}$; hence strictly speaking we assess the effects of atmospheric composition.

The air quality guidelines of the World Health Organization (WHO) and national regulatory policies are based on exposure response functions that rely on $PM_{2.5}$ mass concentrations, implicitly treating all fine particles as equally toxic without regard to their source and chemical composition. However, expert elicitation suggests that carbonaceous particles are more toxic than crustal material, nitrates and sulfates[10]. A recent study[25] finds that $PM_{2.5}$ from coal combustion leads to increased mortality risk from cardiovascular disease and LC, but that the evidence is much weaker for other sources, whereas estimates using non-specific $PM_{2.5}$ mass alone may underestimate the total effect of $PM_{2.5}$ on mortality. Further, this study did not find support for mortality from biomass combustion and soil dust particles[25]. However, this and a subsequent report by the Health Effects Institute in the USA also note that there were only a limited number of cities in these investigations where these sources and components were likely to be measured consistently[26,27]. While the evidence for differential toxicity is far from conclusive, we conducted a secondary analysis assuming that carbonaceous $PM_{2.5}$ is five times more toxic than inorganic particles, though maintaining the same overall health impact of $PM_{2.5}$.

We have calculated premature mortality linked to CEV, COPD, IHD and LC for adults $\geq$30 years old, and ALRI for infants $<$5 years old (Table 1 and Extended Data Tables 1 and 2). Our estimate of the global $PM_{2.5}$ related mortality in 2010 is 3.15 million people with a 95% confidence interval (CI95) of 1.52–4.60 million. The main causes are CEV (1.31 million) and IHD (1.08 million), and secondary causes are COPD (374 thousand), ALRI (230 thousand) and LC (161 thousand). Our global estimate of O_3 related mortality by COPD is 142 (CI95: 90–208) thousand. Our total estimate of 3.30 (CI95: 1.61–4.81) million people in 2010 agrees closely with the GBD[5]. This is in addition to the estimated 3.54 million deaths per year caused by indoor air pollution due to use of solid fuels for cooking and heating[5]. Figure 1 shows the geographic distribution and demonstrates the locations of hotspots in China, India and many of the large urban centres.

Considering the global population of 6.8 billion in 2010, it follows that the mean per capita mortality attributable to air pollution is about 5 per 10,000 person-years. Of these 5 persons per 10,000 worldwide, about 2 die by CEV, 1.6 by IHD, 0.8 by COPD, 0.35 by ALRI and 0.25 by LC. The highest per capita mortality is found in the Western Pacific region, followed by the Eastern Mediterranean and Southeast Asia. The combination of high per capita mortality with high population density explains the (by far) highest number of deaths in the Western Pacific, China being the main contributor (1.36 million per year). Note that the mortality attributable to air pollution in China is approximately an order of magnitude higher than that attributable to Chinese road transport injuries and HIV/AIDS, and ranks among the top causes of death[28]. Southeast Asia has the second highest premature mortality, where India is the main contributor (0.65 million per year). The global

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Table 1 | Premature mortality related to $PM_{2.5}$ and O_3 for the population <5 and ≥30 years old

WHO region	Year	Population ($\times 10^6$)	Mortality attributable to air pollution (deaths $\times 10^3$)						
			$PM_{2.5}$					O_3	Total
			ALRI < 5 yr	IHD ≥ 30 yr	CEV ≥ 30 yr	COPD ≥ 30 yr	LC ≥ 30 yr	COPD ≥ 30 yr	
Africa	2010	809	90	55	77	11	2	2	237
	2050	1,807	158	185	262	38	5	12	660
Americas	2010	930	0	44	8	4	7	5	68
	2050	1,191	0	75	15	7	11	11	119
Eastern Mediterranean	2010	602	56	115	86	12	5	12	286
	2050	1,021	66	321	246	37	13	40	723
Europe	2010	867	1	239	95	13	27	6	381
	2050	886	1	307	156	18	37	11	530
Southeast Asia	2010	1,762	64	327	250	124	15	82	862
	2050	2,332	104	865	807	419	48	227	2,470
Western Pacific	2010	1,812	19	299	794	209	107	35	1,463
	2050	1,861	16	413	1,120	309	155	57	2,070
World	2010	6,783	230	1,079	1,311	374	161	142	3,297
	2050	9,098	346	2,166	2,604	828	270	358	6,572

Regions are defined by the World Health Organization, see Extended Data Table 1. Results for 2050 are based on a business-as-usual scenario.

mortality linked to air pollution is strongly influenced by these high numbers in Asia.

We determined the impacts of seven source categories by subtracting them one by one from the emissions in our model. These sensitivity calculations show the efficacy of individually controlling these sources. The 15 countries with highest premature mortality attributable to air pollution in 2010 are listed in Table 2 along with the contribution of each source category. Residential and commercial energy use (RCO) is the largest source category worldwide, contributing nearly one-third, and almost a factor of 2 more under the alternative assumption of differential toxicity. Note that this only refers to mortality by outdoor exposure to this source. Our estimate of 1.0 million deaths per year by RCO is in addition to the 3.54 million deaths per year due to indoor air pollution from essentially the same source[5].

The next largest anthropogenic source category is agriculture (AGR), contributing one-fifth; however, this reduces significantly under the assumption of differential particle toxicity. The successive principal anthropogenic categories are power generation (PG), industry (IND), biomass burning (BB) and land traffic (TRA), and taken together they cause nearly one-third of all air pollution mortality. If carbonaceous particles are five times more toxic than sulfates and nitrates, these sources together account for one-quarter of the mortality. Natural sources make up for the remaining one-sixth of the total. However, if crustal material is five times less toxic than carbonaceous $PM_{2.5}$ this reduces considerably. The most important source category in each region in 2010 is shown in Fig. 2.

RCO is foremost in the populous parts of Asia. It refers to small combustion sources, especially biofuel use (for heating and cooking), and also waste disposal and diesel generators. In China it contributes about 32%, in India, Bangladesh, Indonesia and Vietnam 50–60%, while in Nepal it is highest with nearly 70% (Extended Data Table 3). In western countries it is typically 5–10%, although in France and Poland it contributes about 15%. The contribution of this pollution source to mortality is sensitive to toxicity assumptions and large uncertainty related to IHD. Because of the comparatively large fraction of carbonaceous $PM_{2.5}$, under our alternative calculations where these aerosols are five times more toxic, RCO increases from 31% to 59% of global air pollution mortality. If, on the other hand, we assume that RCO does not contribute to IHD mortality, this fraction decreases from 31% to 26% (Methods).

Agriculture (AGR) has a remarkably large impact on $PM_{2.5}$, and is the leading source category in Europe, Russia, Turkey, Korea, Japan and the Eastern USA (Fig. 2). In many European countries, its contribution is 40% or higher. Agricultural releases of ammonia (NH_3) from

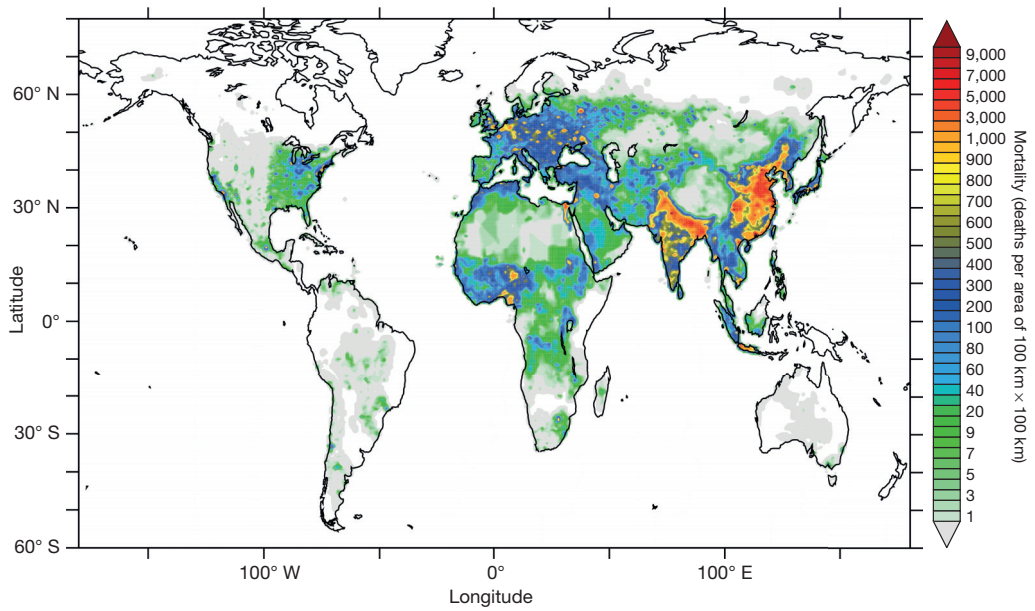

Figure 1 | Mortality linked to outdoor air pollution in 2010. Units of mortality, deaths per area of 100 km × 100 km (colour coded). In the white areas, annual mean $PM_{2.5}$ and O_3 are below the concentration–response thresholds where no excess mortality is expected.

Table 2 | Top 15 ranked countries of premature mortality linked to outdoor air pollution in 2010

Country	Deaths ($\times 10^3$)	Residential energy	Agriculture	Natural	Power generation	Industry	Biomass burning	Land traffic
China	1,357	**32** (**76**)	29 (7)	9 (3)	18 (7)	8 (3)	1 (2)	3 (2)
India	645	**50** (**77**)	6 (1)	11 (1)	14 (5)	7 (3)	7 (9)	5 (4)
Pakistan	111	31 (**67**)	2 (1)	**57** (23)	2 (1)	2 (2)	2 (3)	3 (3)
Bangladesh	92	**55** (**78**)	10 (2)	0 (0)	15 (6)	7 (2)	7 (8)	6 (4)
Nigeria	89	14 (31)	1 (0)	**77** (**52**)	0 (0)	0 (0)	8 (16)	0 (0)
Russia	67	7 (18)	**43** (**26**)	1 (0)	22 (17)	8 (5)	8 (21)	11 (13)
USA	55	6 (12)	29 (17)	2 (2)	**31** (19)	6 (5)	5 (9)	21 (**36**)
Indonesia	52	**60** (**64**)	2 (0)	0 (0)	5 (3)	4 (2)	27 (29)	2 (2)
Ukraine	51	6 (13)	**52** (**32**)	0 (0)	18 (17)	9 (7)	5 (18)	10 (13)
Vietnam	44	**51** (**74**)	12 (2)	0 (0)	13 (4)	8 (3)	12 (14)	4 (3)
Egypt	35	1 (2)	3 (3)	**92** (**88**)	2 (2)	1 (1)	0 (1)	1 (3)
Germany	34	8 (17)	**45** (26)	0 (0)	13 (10)	13 (8)	1 (3)	20 (**36**)
Turkey	32	9 (**20**)	**29** (19)	15 (6)	19 (14)	11 (8)	6 (19)	11 (14)
Iran	26	1 (3)	6 (6)	**81** (**75**)	4 (4)	3 (3)	1 (2)	4 (7)
Japan	25	12 (**29**)	**38** (22)	0 (0)	17 (15)	18 (14)	5 (8)	10 (12)
World	3,297	**31** (**59**)	20 (7)	18 (11)	14 (7)	7 (3)	5 (8)	5 (5)

Columns 3–9 show contributions (%) of the seven main source categories, the leading one in bold. For details and additional countries, see Extended Data Table 3. In parentheses are shown sensitivity calculations with carbonaceous particles having a five times larger impact than inorganic aerosol compounds.

fertilizer use and domesticated animals affect air quality through several multiphase chemical pathways, forming ammonium sulphate and nitrate. Since NH_3 abundance is often limiting in $PM_{2.5}$ formation, reduction of its emissions can make an important contribution to air quality control[29]. As agricultural emissions mostly form inorganic $PM_{2.5}$, the impact on mortality diminishes under the assumption that carbonaceous $PM_{2.5}$ is five times more toxic.

Natural sources (NAT) contribute strongly to mortality, being dominant in northern Africa and the Middle East, and also a leading category in Central Asia (Table 2 and Fig. 2). Although we categorize airborne desert dust as natural, a fraction is anthropogenic due to the role of humans in desertification and agricultural practices[30]. The chronic health and mortality impacts associated with exposure to dust are more uncertain than those due to typical air pollution in industrialized countries where most of the epidemiological cohort studies have been carried out. If all fine particles are equally toxic, then natural sources are responsible for about one-sixth of air pollution mortality. If fine carbonaceous particles are five times more toxic than crustal material, then natural sources account for only about one-tenth of air pollution induced mortality.

Power generation (PG) by fossil fuel fired power plants is the third largest anthropogenic source category, being an important source of SO_2 and NO_x, which are converted to sulfate and nitrate in the atmosphere. It accounts for about one-seventh of population exposure to $PM_{2.5}$ and O_3. Power plant emissions are quite important in the USA (>30%) and in Russia, Korea and Turkey (roughly 20%). Emissions from power generation also have particularly large impacts on fine particle concentrations in the Middle East, but frequently these go unnoticed as they are masked by desert dust. The role of this source is sensitive to the assumed $PM_{2.5}$ toxicity, reducing by a factor of 2 if sulfate and nitrate are five times less toxic than carbonaceous $PM_{2.5}$.

Industry (IND) is among the smaller source categories, with a global fraction of about 7% (Table 2); nevertheless, it contributes about twice this percentage in most of the western world. It includes iron and steel, chemical, pulp and paper, food, solvent and other manufacturing sectors, oil refineries and fuel production. This source of air pollution is generally significant in industrialized countries and emerging economies, but rarely the leading cause of premature mortality. Under the differential toxicity assumption, its contribution to mortality would reduce by more than a factor of 2.

Our calculations suggest that land traffic (TRA) emissions are responsible for about one-fifth of mortality by ambient $PM_{2.5}$ and O_3 in Germany, the UK and the USA, while globally they account

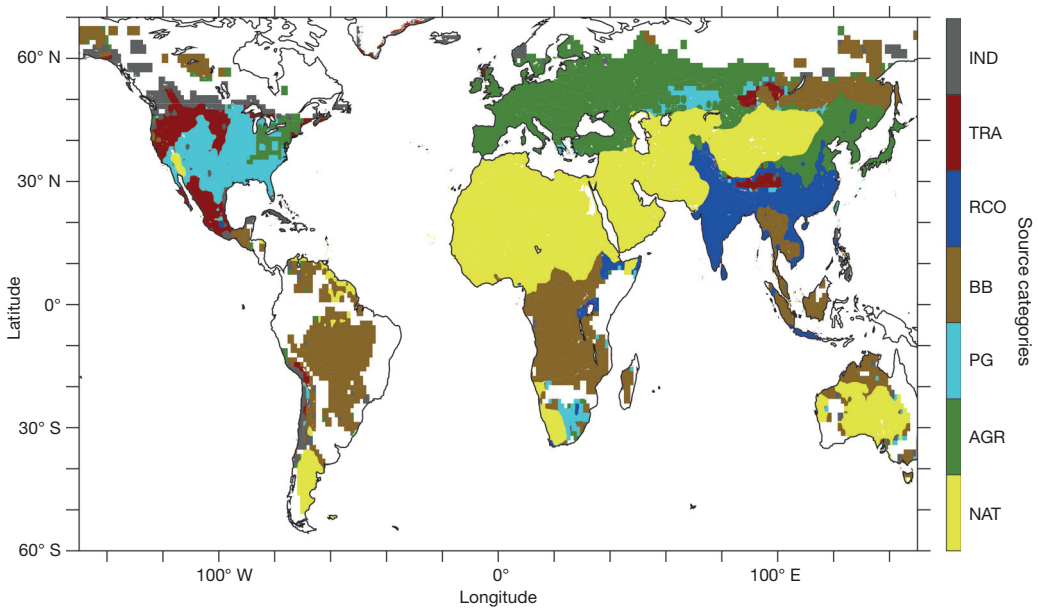

Figure 2 | Source categories responsible for the largest impact on mortality linked to outdoor air pollution in 2010. Source categories (colour coded): IND, industry; TRA, land traffic; RCO, residential and commercial energy use (for example, heating, cooking); BB, biomass burning; PG, power generation; AGR, agriculture; and NAT, natural. In the white areas, annual mean $PM_{2.5}$ is below the concentration–response threshold.

for about 5%. Because emissions of NO_x are the dominant source of traffic-related $PM_{2.5}$ in the form of nitrate, together with carbonaceous $PM_{2.5}$, the results from our alternative calculations—assuming carbonaceous particles are five times more toxic than nitrates and other inorganics—also indicate a 5% contribution, globally. Note that this contribution is likely to be a lower limit as traffic also emits other pollutants that are not included or influential on $PM_{2.5}$ (ref. 31) (Methods).

Biomass burning (BB) is also a relatively small source category with a global contribution of about 5%. Nevertheless, its areal range is large, for example in South America and Africa. It is the main source of air pollution in large parts of Canada, Siberia, Africa, South America and Australia. Because in many parts of these countries annual mean $PM_{2.5}$ is below the concentration–response threshold (Methods), these areas are shown white in Fig. 2. Biomass burning is also widespread in southeastern Asia, although in populous parts of Vietnam and Indonesia (for example, Java) residential energy use is larger and therefore the leading category (Table 2).

In the Southern Hemisphere biomass burning is generally the leading contributor to $PM_{2.5}$, with some exceptions. In Brazil it contributes about 70%, and in many African countries its impact can also be high, up to >90% in Angola. Note that the health impacts of $PM_{2.5}$ from biomass burning are quite uncertain, especially the attribution of IHD related mortality, due to a dearth of epidemiological cohort studies in regions where this pollution source predominates (Methods). Our calculations suggest that it is responsible for between 5% (equal toxicity) and 8% (differential toxicity) of air pollution induced mortality.

To understand how the premature mortality attributable to air pollution may develop in the coming decades, we applied a business-as-usual (BaU) emission scenario for the years 2025 and 2050, assuming that only currently agreed legislation is implemented that will affect future emissions[32]. Thus air quality and emission standards are fixed. Results for 2050 are presented here, and for 2025 in Extended Data Fig. 2 and Extended Data Tables 4, 5. Under the BaU scenario, moderate though significant increases of premature mortality will occur in Europe and the Americas, to a large degree in urban areas. Large increases are projected in Southeast Asia and the Western Pacific, leading to a global growth of premature mortality to 6.6 (CI95: 3.4–9.3) million (+100%) in 2050 (Table 1). This compares to a negligible population increase of infants (<5 years old), and a substantial increase (+68%) among people ≥30 years old in 2050 (implying an ageing population). Globally, the per capita mortality is projected to increase from 5 per 10,000 person-year in 2010 to about 7 per 10,000 person-year in 2050. The mortality attributable to air pollution will continue to be dominated by Asia with an unchanged fraction of about 75%.

The urban population is expected to grow relatively rapidly from 3.6 billion in 2010 to 5.2 billion in 2050, and combined with increasing air pollution concentrations the health impacts will escalate. Our estimate of urban premature mortality by outdoor air pollution in 2010 is 2.0 million, increasing to 4.3 million in 2050, representing 60% of the global total in 2010 and 65% in 2050. Urban population growth is responsible for part of this change, but the levels of air pollution in urban areas are also projected to grow rapidly. This is evident from our finding that the per capita mortality attributable to air pollution in 2010 is about 50% higher in urban than in rural environments. Under the BaU scenario this difference is expected to increase to nearly 90% in 2050.

Recently, much emphasis has been placed on rapidly emerging megacities (Methods). We calculate that 17 megacities and conurbations in Asia rank among the top 30 in terms of premature mortality worldwide, the leading one being the Pearl River Delta. When viewed instead from the perspective of individual risk, Tianjin and Beijing rank highest (Extended Data Table 6). While the per capita mortality attributable to air pollution is already extraordinary in Chinese megacities, according to the BaU scenario it will become even higher in Chinese and also Indian megacities by 2050. The combined premature mortality in the 30 largest conurbations accounts for about 7% of the worldwide burden of air pollution, indicating the relevance of all urban areas.

Our results suggest that if the projected increase in mortality attributable to air pollution is to be avoided, intensive air quality control measures will be needed, particularly in South and East Asia. The poorly characterized uncertainty about the relative toxicity of various classes of particles such as sulfates, nitrates, organics, crustal materials, black carbon, and especially smoke from biomass combustion, limits unambiguous attribution of sources. Nevertheless, our study suggests that emissions from residential energy use should be considered in air pollution control strategies and, if all fine particles are equally toxic, the reduction of agricultural emissions would improve air quality. An improvement in the efficacy of air pollution controls requires a better understanding of the relative toxicity of particles from various emissions sources.

Online Content Methods, along with any additional Extended Data display items and Source Data, are available in the online version of the paper; references unique to these sections appear only in the online paper.

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Author Contributions J.L., A.P. and M.F. planned the research, A.P. performed the model calculations, J.L., A.P., D.G. and J.S.E. analysed the results, and J.L. and J.S.E. wrote the paper. All authors contributed to the manuscript.

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METHODS

Model and emissions. We used the global ECHAM5/MESSy atmospheric chemistry (EMAC)–general circulation model at a spatial resolution of T106L31, that is, with a spherical spectral truncation of T106, which corresponds to a quadratic Gaussian grid of approximately 1.1° × 1.1° latitude × longitude (~110 km at the Equator), with 31 vertical hybrid terrain-following and pressure levels up to 10 hPa in the lower stratosphere. The core atmospheric model is the 5th generation European Centre Hamburg (ECHAM5, version 5.3.01) general circulation model[33]. EMAC includes sub-models that represent tropospheric and stratospheric processes and their interaction with oceans, land and human influences[34–36]. It uses the Modular Earth Submodel System (MESSy, v.1.09) to link submodels that describe emissions, atmospheric chemistry, aerosol and deposition processes; the results have been tested against *in situ* and remote sensing observations[37–49].

Following up on Lelieveld *et al.*[21], who focused on the year 2005, we present results for the years 2010, 2025 and 2050, applying monthly varying emission data from Doering *et al.*[50], also used by Pozzer *et al.*[32]. The data are from the Emission Database for Global Atmospheric Research (EDGAR), prepared by the Joint Research Centre of the European Commission in Ispra (Italy) at a resolution of 0.1° latitude and longitude[50,51]. For the year 2010 we performed sensitivity calculations in which seven main emission categories have been removed one by one to compute the impact of these sources and to estimate their contributions to air quality control and related mortality. We first calculated the apportionment of source categories to the total $PM_{2.5}$ and O_3 concentrations and then applied the computed fractions to the total mortalities attributable to air pollution.

The categories are: (1) 'Natural' (NAT), mostly desert dust but locally also sea salt and dimethyl sulphide derived sulphate, some nitrate and ammonium from natural sources, volcanic sulphur emissions and organics released by the vegetation; (2) 'Industry' (IND), including iron and steel, chemical, pulp and paper, food, solvent and other manufacturing sectors, oil refineries and fuel production; (3) 'Land transport' (TRA), that is, road and non-road transport on land; (4) 'Residential and commercial energy use' (RCO), referring to local and commercial energy use from small combustion sources for space heating and cooking, including diesel generators and biofuel use; (5) 'Power generation' (PG), that is, public energy production by fossil fuel fired power plants; 6) 'Biomass burning' (BB), that is, tropical forest fires and deforestation, savanna and shrub fires, middle and high latitude forest and grassland fires, and agricultural waste burning; and (7) 'Agriculture' (AGR), dominated by ammonia emissions associated with the use of fertilizers and domesticated animals. Not included in these categories are air traffic and shipping. We find that the removal of individual source categories leads to a near-linear response in the modelled contributions to mortality, indicated by the small scaling corrections needed (about 10%) to add up to 100% in the country level contributions, that is, in Table 2 and Extended Data Table 3.

The BaU scenarios for 2025 and 2050 assume that energy and food consumption are largely determined by population growth and economic development, which in turn drive air pollution sources based on current legislation and technology[32,50,51]. This represents a pessimistic, but plausible future prospect. Comparable to Shindell *et al.*[52], and different from the Representative Concentration Pathways of the Intergovernmental Panel on Climate Change[53], the BaU scenario differentiates between air pollution and climate change mitigation measures, as the latter typically require relatively long-term and structural societal changes. The scenarios used here are based on projections for energy and fuel computed by the Prospective Outlook for the Long-term Energy System (POLES) model[51,54] and for agriculture, land-use and waste projections by the Integrated Model to Assess the Global Environment (IMAGE)[55].

The population development in the BaU scenario is consistent with our mortality calculations, as described below, projecting 9 billion people in 2050. For additional details we refer to Pozzer *et al.*[32] and references therein. While BaU projections should not be conceived as 'predictions', especially for 2050, they represent the current trajectory into the future and may be considered a worst-case scenario, to explore what can be expected if air quality policies and health care remain as they are today. Note that these results are not sensitive to differential toxicity assumptions as the total mortality induced by $PM_{2.5}$ is not affected, only the attribution to source categories. For the future scenarios we used the baseline mortalities for 2010. Hence the implicit assumption is that smoking habits, diets and health care remain unchanged.

The model meteorology has been forced by pre-calculated sea surface temperatures and ice coverage based on a 10-year climatology (2000–2009) adopted from the AMIP-II database[56,57]. The model was applied in atmospheric chemistry-transport mode by switching the coupling between radiation and atmospheric chemistry off, so that atmospheric composition changes do not influence the model dynamics[32]. This is justified considering that air quality projections are primarily driven by emissions rather than climate change[58,59], even though natural sources, biomass burning and deposition processes can be influenced by climatic conditions[20,59–62]. For example, Fang *et al.*[62] project a 4% climate change effect for $PM_{2.5}$ related mortality and less than 1% for O_3 related mortality by the end of the 21st century.

Although our model resolution does not resolve small-scale heterogeneities in the urban environment, a comparison with satellite and ground-based remote sensing observations indicates that this is not critical. The exposure response functions used to calculate mortalities are based on annual mean concentrations for which these heterogeneities largely average out. This is illustrated by Extended Data Fig. 3, which compares a simulation for the year 2010 with ground-based AERONET remote sensing data of aerosol optical depth (AOD) (http://aeronet.gsfc.nasa.gov). Since our model approximates though not replicates meteorological conditions for the year 2010, and local flows near the AERONET stations cannot be captured, substantial scatter around the ideal 1:1 comparison is expected. The comparison shows that the model mean error and bias are small (the latter absent for the annual mean), and the correlation good. We have also performed a comparison between MODIS (satellite) and AERONET data of AOD, leading to similar spread and correlations, the latter also increasing through averaging (not shown).

The primary differences in the relationships between emissions and exposures for ground level sources, such as traffic, in comparison with elevated sources, such as power plants, have been accounted for in our model[43]. The relative impacts of secondary particles (such as sulfates and nitrates) from these sources are expected to be realistically simulated. On the other hand, models such as ours cannot capture the fine structure of near-source gradients in ultrafine PM along transportation corridors. Because of this our estimates of the relative impacts of urban traffic and urban sources of primary fine particles may be biased downward, though only to the extent that ultrafine PM is in fact responsible for the mortality seen in cohort studies. As discussed above, the relative toxicity of various constituents of ambient $PM_{2.5}$ has not been well established. Our sense is that the sensitivity study, allowing for carbonaceous particles to be five times as toxic as sulfates, nitrates and crustal material, is adequate to cover any potential differences in the relationships between emissions, exposure and differential toxicity of traffic related $PM_{2.5}$.

To investigate if our model reproduces urban concentration increments of $PM_{2.5}$ and O_3, that is, comparing the urban background with the rural environment, we compare our results with recent case studies[63–67]. For Paris and London our model computes urban $PM_{2.5}$ increments of 18% and 2%, respectively, consistent with the measurements and highly resolved model calculations. Our model calculations suggest that the leading sources of $PM_{2.5}$ in Paris are residential energy use, agriculture and traffic. Agricultural emissions (NH_3/NH_4^+) are transported from the rural environment and contribute to $PM_{2.5}$ in the city. For London we calculate that $PM_{2.5}$ is most strongly influenced by agriculture, traffic and power generation. The limited contribution by land traffic and the importance of atmospheric transport for air quality in London have been corroborated by observational analysis[63]. For Beijing we calculate an urban $PM_{2.5}$ increment of 5%, consistent with the conclusion by Zhang *et al.*[67] that regional sources are crucial contributors to $PM_{2.5}$. They estimate the contribution by traffic and waste incineration at 4%; our results suggest that traffic alone contributes 3% in this city and residential energy use 47%, which we find to be representative of China (Table 2).

Our model calculations indicate that these relatively small urban increments for $PM_{2.5}$ are typical for many, though not all, cities. For example, for Johannesburg (including Pretoria) we find +41% and for the Pearl River area +62%, and in both conurbations residential energy use is the leading source of $PM_{2.5}$. For O_3 we find generally small and negative urban increments due to titration of O_3 by local traffic emissions (in Paris −7% and in London −5%). Negative urban increments due to NO by traffic of a few per cent (comparing weekend with weekdays) have also been documented for American cities[68]. For Chicago, New York, Los Angeles and Atlanta we find negative O_3 increments of 1–5% due to traffic and power generation.

Sample size. No statistical methods were used to predetermine sample size.

Exposure response functions. The premature mortality attributable to $PM_{2.5}$ and O_3 has been calculated by applying the EMAC model for the present (2010) and projected future (2025, 2050) concentrations. We combined the results with epidemiological exposure response functions by employing the following relationship to estimate the excess (that is, premature) mortality:

$$\Delta\mathrm{Mort} = y_o[(\mathrm{RR}-1)/\mathrm{RR}]\mathrm{Pop} \quad (1)$$

ΔMort is a function of the baseline mortality rate due to a particular disease category y_o for countries and/or regions estimated by the World Health Organization[69] (the regions and strata are listed in the Extended Data Table 1). The term (RR − 1)/RR is the attributable fraction and RR is the relative risk. The disease specific baseline mortality rates have been obtained from the WHO Health Statistics and Health Information System. The value of RR is calculated for the different disease categories attributed to $PM_{2.5}$ and O_3 for the population below 5 years of age (ALRI) and 30 years and older (IHD, CEV, COPD, LC) using

exposure response functions from the 2010 GBD analysis of the WHO (and described below).

The population (Pop) data for regions, countries and urban areas have been obtained from the NASA Socioeconomic Data and Applications Center (SEDAC), hosted by the Columbia University Center for International Earth Science Information Network (CIESIN), available at a resolution of 2.5′ × 2.5′ (about 5 km × 5 km) (http://sedac.ciesin.columbia.edu/), and projections by the United Nations Department of Economic and Social Affairs/Population Division[70] (http://esa.un.org/unpd/wpp). Urban areas are defined by applying a population density threshold of 400 individuals per km^2, while for megacities and major conurbations the threshold is 2,000 individuals per km^2. We note that the resolution of our atmospheric model, about 1° latitude/longitude, is coarser than that of the population data, and our model does not resolve details of the urban environment. However, our anthropogenic emission data are aggregated from a resolution of 10 km to that of the model grid, accounting for relevant details such as altitude dependence (for example, stack emissions and hot plume rise effects)[43].

Lelieveld *et al.*[21] (henceforth L2013) derived the relative risk RR from the following exposure response function:

$$\mathrm{RR} = \exp[b(X - X_o)] \quad (2)$$

The term X represents the model calculated annual mean concentration of $PM_{2.5}$ or O_3. The value of X_o is the threshold concentration below which no additional risk is assumed (concentration–response threshold). The parameter b is the concentration response coefficient. However, it has been argued that this expression is based on epidemiological cohort studies in the USA and Europe where annual mean $PM_{2.5}$ concentrations are typically below 30 μg m^{-3}, which may not be representative for countries where air pollution levels can be much higher, for example in South and East Asia. This is particularly relevant for our BaU scenario. Therefore, here we have used the revised exposure response function of Burnett *et al.*[8] who also included epidemiological data from the exposure to second-hand smoke, indoor air pollution and active smoking to account for high $PM_{2.5}$ concentrations, and tested eight different expressions. The best fit to the data was found for the following relationship, which was also used by Lim *et al.*[5] for the GBD for the year 2010:

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

The RR functions were derived by Burnett *et al.*[8]. We applied this model for the different categories, represented by their figures 1 and 2, shown to be superior to other forms previously used in burden assessments. We also adopted the upper and lower bounds, likewise shown in these figures, representing the 95% confidence intervals (CI95). The latter were derived based on Monte Carlo simulations, leading to 1,000 sets of coefficients and exposure response functions from which the upper and lower bounds were calculated.

Following Burnett *et al.*[8] and Lim *et al.*[5] we combine all aerosol types, hence including natural particulates such as desert dust. Note that by using $PM_{2.5}$ mass, we do not distinguish the possibly different toxicity of various kinds of particles. This information is not available from epidemiological cohort studies, but could potentially substantially affect both our overall estimates of mortality and the geographical patterns. This is addressed by sensitivity calculations presented in the main text, Table 2 and Extended Data Fig. 1. For COPD related to O_3 we applied the exposure response function by Ostro *et al.*[3]:

$$\mathrm{RR} = [(X + 1)/(X_o + 1)]^b \quad (4)$$

where b is 0.1521 and X_o the average of the range 33.3–41.9 p.p.b.v. O_3 indicated by Lim *et al.*[5], that is, 37.6 p.p.b.v. Previously we used model calculated pre-industrial O_3 concentrations to estimate $X - X_o$ (ref. 21), leading to about 20% higher estimates for mortality by 'respiratory disease' related solely to O_3 compared to the current estimate for COPD due to both $PM_{2.5}$ and O_3.

For detailed discussion of uncertainties and sensitivity calculations that address the shape of exposure response functions, we refer to earlier work[5,8,21,22] and references therein. L2013 estimated statistical uncertainties by propagating the quantified (random) errors of all parameters in the exposure response functions. They found that the CI95 of estimated mortality attributable to air pollution in Europe, North and South America, South and East Asia are within 40%, whereas they are 100–170% in Africa and the Middle East. Our results are very close to the GBD, which substantiates the estimates by Lim *et al.*[5] and provides consistency with the most recent estimates for 2010, serving as a basis for our investigations.

We emphasize that the confidence intervals described here, and those reported by Lim *et al.*[5], reflect only the statistical uncertainty of the parameters used in the concentration–response functions. It is known that the uncertainty in interpretation of epidemiological results can be dominated by other model or epistemic uncertainties, such as those having to do with the control of confounders. Sources of uncertainty have been summarized by Kinney *et al.*[71], who underscore the need to determine the differential toxicity of specific component species within the complex mixture of particulate matter. Our sensitivity calculations (Table 2 and Extended Data Fig. 1) corroborate that this can have significant influence, especially in areas where carbonaceous compounds contribute strongly to $PM_{2.5}$.

We emphasize the dearth of studies that link $PM_{2.5}$ from biomass combustion emissions—rich in carbonaceous particles—to IHD. Expert judgment studies on the toxicity of particulate matter have reported uncertainties much larger than those suggested by analysis of parameter uncertainty alone[10,72]. Although the CI95 intervals provided above include a larger range of parameters and uncertainties than these earlier studies, they should be viewed as lower bounds on the true uncertainty in estimates of the health effects of $PM_{2.5}$ exposure, especially $PM_{2.5}$ from biomass burning and biofuel use. If we consider the possibility that biomass burning (BB, including agricultural waste burning) and residential energy use (RCO, dominated by biofuel use) do not contribute to mortality by IHD, the total mortality attributable to air pollution would decrease from 3.3 to 3.0 million per year (Extended Data Table 7). The largest effect is found in Southeast Asia where biomass combustion (RCO and BB) is a main source of air pollution. While the global contribution by residential energy use, as presented in Table 2, would decrease from 31% to 26%, and of biomass burning from 5% to 4% (the other categories increase proportionally), the ranking of the different sources and hence our conclusions remain unchanged, as RCO and BB would still be the largest and smallest source category, respectively.

Issues such as the shape of the concentration–response functions and the existence and specific levels of concentration–response thresholds have been discussed by the experts[10,71,72]. These have been accounted for by Burnett *et al.*[8], however, uncertainty related to the differences in central estimates given by various cohort studies is not reflected in the estimates of parameter uncertainty by Lim *et al.*[5]. This problem has grown more substantial recently as the results from new cohort studies have become available[73]. Furthermore, uncertainty about the relative toxicity of different constituents of $PM_{2.5}$ remains. Since the current study underscores that the sources of mortality attributable to $PM_{2.5}$ can differ strongly between different regions (Fig. 2), this aspect merits greater attention in future.

Comparison to previous work. We estimate the combined ($PM_{2.5}$ and O_3 related) global mortality attributable to air pollution in 2010 at 3.3 million. Our global estimate for $PM_{2.5}$ related mortality of 3.15 million per year is close to that of 3.22 million per year in the GBD study for 2010 (ref. 4). However, it is substantially higher than the recent multi-model study of Silva *et al.*[20] for the year 2000, being 2.1 million per year. The difference can be explained by the focus of Silva *et al.*[20] on anthropogenic pollution in 2000, whereas our study and the GBD account for emission increases between 2000 and 2010 and also include natural sources.

Our global estimate of O_3 related mortality by COPD in 2010 is 142,000, substantially lower than the estimates of Anenberg *et al.*[18], 700,000 deaths in 2000; L2013, 773,000 in 2005; and Silva *et al.*[20], 470,000 deaths in 2000; but quite close to the GBD estimate of 152,000 deaths in 2010. Much of the difference between our results (and those from the 2010 GBD) and previous work is explained by the fact that we attribute COPD to both O_3 and $PM_{2.5}$. When our results for COPD from both O_3 and $PM_{2.5}$ are combined, our overall estimate of COPD mortality from air pollution agrees with the above-mentioned studies within about 25–30%. The remaining differences are largely due to the use of a concentration response threshold, X_o, in our new work, which substantially reduces mortality estimates. Anenberg *et al.*[18] and L2013 did not apply a threshold but computed the natural background based on preindustrial emissions. In these analyses the calculated ambient concentrations are typically lower than X_o. For example, the global average O_3 ambient concentration at the surface in our preindustrial simulation is 19 p.p.b.v. The global mortality estimate for 2010 presented here is 10% higher than that of L2013 for 2005. This is primarily due to the fact that we also account for natural sources in the present work. If we subtract the natural fraction, our estimate of mortality attributable to anthropogenic air pollution for 2010 is 9% lower than that of L2013, mostly related to the new exposure response functions applied here.

Our calculations suggest that natural sources contribute relatively strongly to mortality attributable to air pollution (18%), about 600,000 per year, which is to a large degree caused by airborne desert dust. Recently we reported a global dust-related mortality rate of about 400,000 per year, substantially lower than the present estimate[22]. While here we follow the GBD methodology[5], it is likely to yield an upper limit. Instead of the annual mean dust concentrations Giannadaki *et al.*[22] used the median concentrations, motivated by the intermittent nature of dust events. Their sensitivity calculations indicate that had they used the mean concentration instead, their estimate of global dust-related mortality would have increased from 402,000 per year to 622,000 per year. Finally, if we assume that carbonaceous aerosols are five times more toxic than other compounds, including dust particles, the contribution by natural sources would decrease from about 600,000 per year (18%) to 360,000 per year (11%).

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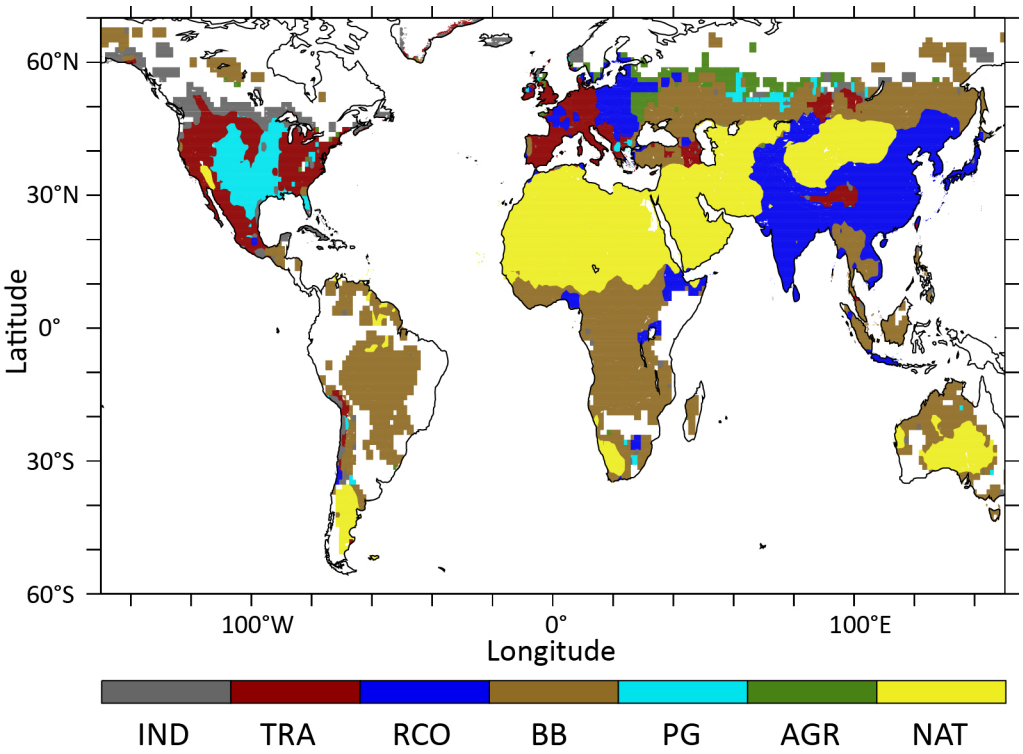
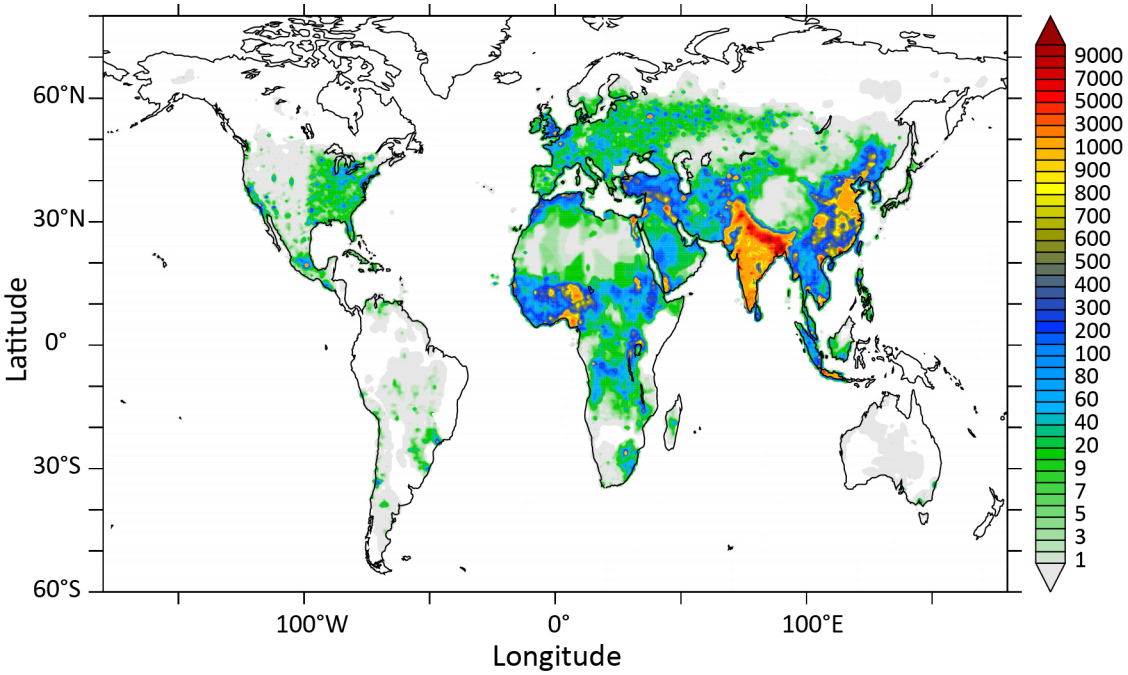
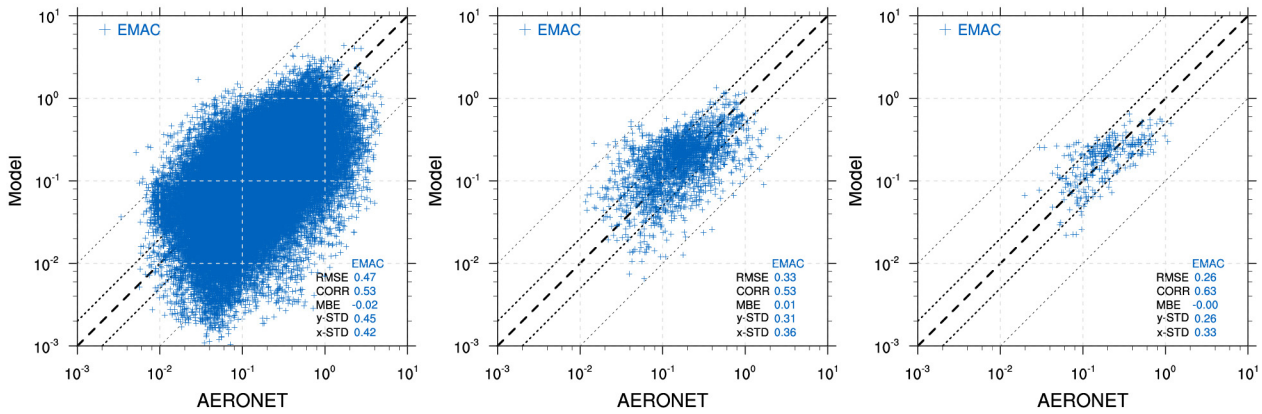


Extended Data Figure 1 | Source categories responsible for the largest impact on mortality linked to outdoor air pollution in 2010 from a sensitivity calculation with carbonaceous aerosol having a five times larger impact than inorganic and crustal compounds. IND, industry; TRA, land traffic; RCO, residential energy use (for example, heating, cooking); BB, biomass burning; PG, power generation; AGR, agriculture; and NAT, natural.



Extended Data Figure 2 | Increase in mortality linked to outdoor air pollution from 2010 to 2050 (business-as-usual scenario). Units (colour coded), deaths per area of 100 km × 100 km. In the white areas, no additional mortality is projected.



Extended Data Figure 3 | Comparison of EMAC model calculated aerosol optical depth (AOD) with AERONET observations, using all available measurements worldwide in the year 2010. Although the comparison with individual data points shows a large scatter (left panel), the bias is small (MBE), and time averaging improves the agreement. The middle panel shows a comparison of the monthly means, and the right panel the annual means (that is, showing individual stations) for which the mean error (root mean square error, RMSE) is smallest, the correlation highest and the bias absent. The long-dashed line indicates absolute agreement, the bold short-dashed lines agreement within a factor of two and the short-dashed lines agreement within a factor of ten.

Extended Data Table 1 | 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, Netherlands 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, United Kingdom
	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, Vietnam, Wallis and Futuna

Extended Data Table 2 | Premature mortality related to $PM_{2.5}$ and O_3 in 2010

			Mortality attributable to air pollution (deaths ×10^3)						
					$PM_{2.5}$			O_3	Total
	Strata	Population (×10^6)	ALRI <5 yr	IHD ≥30 yr	CEV ≥30 yr	COPD ≥30 yr	LC ≥30 yr	COPD ≥30 yr	
Africa	Afr-D	379	77	37	62	9	1	2	188
	Afr-E	430	13	17	15	3	0	1	49
Americas	Amr-A	352	0	38	6	4	6	3	57
	Amr-B	493	0	5	1	0	1	1	8
	Amr-D	85	0	0	0	0	0	0	1
Eastern Mediteranean	Emr-B	165	2	34	20	2	1	3	62
	Emr-D	437	54	80	67	11	3	9	224
Europe	Eur-A	410	0	73	33	7	16	3	132
	Eur-B	229	1	57	34	3	7	2	104
	Eur-C	228	0	110	28	2	4	0	144
Southeast Asia	Sear-B	324	3	37	23	7	3	2	75
	Sear-D	1,438	61	290	227	117	12	80	787
Western Pacific	Wpr-A	156	0	11	9	0	5	2	27
	Wpr-B	1,656	19	288	784	209	102	33	1,435
World		6,783	230	1,079	1,311	374	161	142	3,297

Extended Data Table 3 | Premature mortality by $PM_{2.5}$ and O_3 related diseases in 2010 in countries where it exceeds 9,000 individuals per year (<5 and ≥30 years old)

Country	Deaths in 2010	Natural	Industry	Land traffic	Residential energy	Power generation	Biomass burning	Agriculture
China	1,357,353	118,954	106,754	44,751	435,763	237,324	18,414	395,390
India	644,993	74,145	42,336	30,070	325,604	89,130	42,163	41,541
Pakistan	110,571	63,147	2,478	3,389	34,707	2,761	2,108	1,977
Bangladesh	91,923	0	6,117	5,656	50,382	13,697	6,418	9,652
Nigeria	89,022	68,479	176	85	12,006	258	7,554	462
Russia	67,152	630	5,193	7,731	4,885	14,606	5,477	28,628
USA	54,905	1,290	3,297	11,435	3,192	16,929	2,537	16,221
Indonesia	52,417	71	1,814	1,244	31,498	2,379	14,338	1,070
Ukraine	51,238	55	4,632	5,188	3,011	9,459	2,326	26,563
Vietnam	44,097	0	3,627	1,686	22,575	5,486	5,378	5,343
Egypt	35,322	32,651	210	450	190	816	61	941
Germany	34,422	0	4,452	6,928	2,684	4,402	279	15,675
Turkey	31,943	4,912	3,414	3,487	2,812	6,194	1,851	9,269
Iran	26,108	21,175	662	969	311	1,101	230	1,656
Japan	25,516	0	4,567	2,526	3,046	4,458	1,154	9,763
Sudan	24,255	22,249	59	47	200	133	1,488	77
Myanmar	22,537	10	1,082	842	8,287	2,662	8,707	944
Italy	20,809	1,251	2,930	3,519	1,454	3,192	376	8,085
Iraq	20,335	18,513	209	390	109	510	91	510
Thailand	19,843	0	2,211	1,469	5,207	2,944	6,529	1,481
France	17,800	0	2,515	3,152	2,468	2,113	211	7,339
Dem. Rep. Korea (N)	16,783	0	1,996	770	3,445	3,467	715	6,386
United Kingdom	15,488	0	1,627	3,091	854	2,412	63	7,438
Algeria	14,954	11,262	1,113	656	194	773	107	847
Dem. Republic Congo	14,880	901	193	45	1,405	121	12,119	92
Romania	14,633	0	1,336	1,825	1,403	3,225	573	6,270
Saudi Arabia	14,600	13,708	165	165	46	308	38	167
Poland	14,561	0	1,451	1,886	2,273	2,265	372	6,310
Korea (S)	14,352	0	2,045	1,108	2,049	2,803	321	6,024
Morocco (+W. Sahara)	14,217	10,929	966	346	224	856	99	795
Niger	13,061	12,893	9	0	89	16	32	19
Uzbekistan	11,598	7,341	671	590	578	623	265	1,526
Nepal	10,926	56	641	510	7,481	1,090	681	465
Mali	9,444	9,060	1	0	101	0	273	6
Ghana	9,317	5,552	105	68	525	68	2,922	75
Burkina Faso	9,295	8,851	3	0	127	20	256	35
World	3,297,370	596,895	226,137	163,852	1,002,370	464,748	179,268	664,100

Extended Data Table 4 | Premature mortality related to $PM_{2.5}$ and O_3 in 2025

			Mortality attributable to air pollution (deaths ×10³)						
					$PM_{2.5}$			O_3	Total
	Strata	Population (×10⁶)	ALRI <5 yr	IHD ≥30 yr	CEV ≥30 yr	COPD ≥30 yr	LC ≥30 yr	COPD ≥30 yr	
Africa	Afr-D	538	102	60	99	14	2	5	282
	Afr-E	597	17	30	27	4	1	2	81
Americas	Amr-A	395	0	48	8	5	7	6	74
	Amr-B	561	0	9	3	1	1	3	17
	Amr-D	105	0	1	0	0	0	0	1
Eastern Mediteranean	Emr-B	197	2	55	33	3	2	5	100
	Emr-D	579	61	133	109	17	6	19	345
Europe	Eur-A	423	0	79	37	8	17	5	146
	Eur-B	246	1	74	46	4	9	4	138
	Eur-C	221	0	123	34	3	5	0	165
Southeast Asia	Sear-B	362	3	54	38	11	5	7	118
	Sear-D	1,697	76	461	398	201	21	155	1,312
Western Pacific	Wpr-A	158	0	11	11	1	5	4	32
	Wpr-B	1,760	18	377	1,038	284	138	51	1,906
World		7,838	280	1,515	1,881	556	219	266	4,717

Extended Data Table 5 | Premature mortality related to $PM_{2.5}$ and O_3 in 2050

			Mortality attributable to air pollution (deaths ×10^3)						
					$PM_{2.5}$			O_3	Total
	Strata	Population (×10^6)	ALRI <5 yr	IHD ≥30 yr	CEV ≥30 yr	COPD ≥30 yr	LC ≥30 yr	COPD ≥30 yr	
Africa	Afr-D	874	137	121	203	28	4	8	501
	Afr-E	933	21	64	58	10	1	4	158
Americas	Amr-A	451	0	59	10	6	10	7	92
	Amr-B	609	0	15	5	1	1	4	26
	Amr-D	131	0	1	0	0	0	0	1
Eastern Mediteranean	Emr-B	222	2	80	50	5	3	7	147
	Emr-D	799	64	241	196	32	10	33	576
Europe	Eur-A	431	0	85	43	9	20	5	162
	Eur-B	252	1	95	68	6	12	5	187
	Eur-C	203	0	127	45	3	5	1	181
Southeast Asia	Sear-B	382	3	69	51	14	7	8	152
	Sear-D	1,950	101	796	755	405	41	219	2,317
Western Pacific	Wpr-A	149	0	10	9	0	4	4	27
	Wpr-B	1,712	16	403	1,110	309	151	53	2,042
World		9,098	346	2,166	2,604	828	270	358	6,572

Extended Data Table 6 | Population and premature mortality (deaths per year) related to $PM_{2.5}$ and O_3 in the most polluted megacities and conurbations in 2010, 2025 and 2050

	2010		2025		2050	
Megacity	Population ($\times 10^6$)	Deaths ($\times 10^3$)	Population ($\times 10^6$)	Deaths ($\times 10^3$)	Population ($\times 10^6$)	Deaths ($\times 10^3$)
London	8.1	2.8	9.1	3.4	10.2	4.2
Paris	8.4	3.1	9.2	3.8	10.2	4.6
Moscow	14.9	8.6	14.4	10.8	13.1	11.7
Po Valley	3.4	1.3	3.4	1.4	3.2	1.4
Istanbul	11.1	5.6	13.2	8.5	14.5	13.2
Teheran	9.7	2.9	11.1	4.8	11.4	6.9
Cairo	12.5	6.0	15.9	8.2	19.8	11.4
Lagos	8.3	3.7	12.7	6.0	22.0	11.2
Johannesburg[1]	6.9	1.5	7.7	2.3	8.6	3.8
Karachi	11.9	7.3	15.4	11.4	19.4	17.9
Mumbai[2]	18.0	10.2	22.1	17.4	26.8	33.1
Delhi	22.5	19.7	27.8	31.1	33.3	52.0
Kolkata	20.3	13.5	28.4	26.6	38.8	54.8
Dhaka	22.8	13.1	31.2	26.4	38.2	49.9
Jakarta	22.5	10.4	26.1	16.4	29.0	22.1
Chengdu	6.2	7.4	6.4	9.5	5.9	9.7
Beijing	10.8	13.7	11.3	17.3	10.4	17.7
Tianjin	3.7	4.9	3.9	6.2	3.6	6.3
Shanghai	14.1	14.9	14.3	18.9	13.2	19.4
Seoul	20.8	6.6	21.7	8.5	20.3	8.7
Tokyo	29.2	6.0	28.1	6.4	24.2	5.4
Osaka	13.5	2.8	12.8	3.1	10.9	2.6
Hong Kong	6.9	2.6	7.6	3.7	8.8	4.4
Pearl River area	53.1	49.2	56.0	65.2	52.9	67.4
Manila	19.8	0.6	26.5	2.3	37.3	4.5
Bangkok	8.8	3.1	9.5	4.9	9.2	5.7
New York	12.5	3.2	14.5	4.2	17.5	5.2
Los Angeles	12.2	4.1	14.6	5.2	17.7	7.0
Mexico City	10.7	1.6	12.3	3.3	13.9	5.3

[1] Includes Pretoria
[2] Includes east suburb

The names of the megacities cities have been colour coded according to the WHO regions: Europe, black font; Eastern Mediterranean, blue; Africa, red; Southeast Asia, green; Western Pacific, brown; Americas, purple.

Extended Data Table 7 | Premature mortality related to $PM_{2.5}$ and O_3 for the population aged <5 years and ≥30 years

WHO region	Year	Population ($\times 10^6$)	Mortality attributable to air pollution (deaths $\times 10^3$)						
			$PM_{2.5}$					O_3	Total
			ALRI <5 yr	IHD ≥30 yr	CEV ≥30 yr	COPD ≥30 yr	LC ≥30 yr	COPD ≥30 yr	
Africa	2010	809	90	55	77	11	2	2	237
	2010*			37					219
Americas	2010	930	0	44	8	4	7	5	68
	2010*			35					59
Eastern Mediterranean	2010	602	56	115	86	12	5	12	286
	2010*			104					275
Europe	2010	867	1	239	95	13	27	6	381
	2010*			213					355
Southeast Asia	2010	1,762	64	327	250	124	15	82	862
	2010*			169					704
Western Pacific	2010	1,812	19	299	794	209	107	35	1,463
	2010*			250					1,414
World	2010	6,783	230	1,079	1,311	374	161	142	3,297
	2010*			808					3,026

*In these rows, IHD mortality related to residential energy use (RCO) and biomass burning has been excluded.