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Global health burden of ambient $PM_{2.5}$ and the contribution of anthropogenic black carbon and organic aerosols

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ABSTRACT

Chronic exposure to fine particulate matter ($PM_{2.5}$) poses a major global health risk, commonly assessed by assuming equivalent toxicity for different $PM_{2.5}$ constituents. We used a data-informed global atmospheric model and recent exposure–response functions to calculate the health burden of ambient $PM_{2.5}$ from ten source categories. We estimate 4.23 (95% confidence interval 3.0–6.14) million excess deaths annually from the exposure to ambient $PM_{2.5}$. We distinguished contributions and major sources of black carbon (BC), primary organic aerosols (POA) and anthropogenic secondary organic aerosols (aSOA). These components make up to ~20% of the total $PM_{2.5}$ in South and East Asia and East Africa. We find that domestic energy use by the burning of solid biofuels is the largest contributor to ambient BC, POA and aSOA globally. Epidemiological and toxicological studies indicate that these compounds may be relatively more hazardous than other $PM_{2.5}$ compounds such as soluble salts, related to their high potential to inflict oxidative stress. We performed sensitivity analyses by considering these species to be more harmful compared to other compounds in $PM_{2.5}$, as suggested by their oxidative potential using a range of potential relative risks. These analyses show that domestic energy use emerges as the leading cause of excess mortality attributable to ambient $PM_{2.5}$, notably in Asia and Africa. We acknowledge the uncertainties inherent in our assumed enhanced toxicity of the anthropogenic organic and BC aerosol components, which suggest the need to better understand the mechanisms and magnitude of the associated health risks and the consequences for regulatory policies. However our assessment of the importance of emissions from domestic energy use as a cause of premature mortality is robust to a range of assumptions about the magnitude of the excess risk.

1. Introduction

The long-term exposure to ambient $PM_{2.5}$ has been associated with a multitude of detrimental effects on human health, including excess deaths from cardiovascular diseases, cerebrovascular diseases, acute lower respiratory-tract infection, diabetes, lung cancer, adverse birth outcomes and neonatal diseases (Cohen et al., 2017; Lelieveld et al., 2015; Murray et al., 2020; Schraufnagel et al., 2019a, 2019b). The disease risk following exposure to $PM_{2.5}$ is influenced by multiple factors including size, mass and composition of $PM_{2.5}$ (Schraufnagel et al., 2019a, 2019b). Although the biological mechanisms are not yet fully elucidated, evidence suggests that inhalation of pro-oxidant chemical components of $PM_{2.5}$, eg. anthropogenic secondary aerosols (aSOA), black carbon (BC) and primary organic aerosols (POA) (the latter two involving primary ultrafine combustion particles that can carry noxious species like polycyclic aromatic hydrocarbons), induce oxidative stress

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and inflammation, leading to respiratory and cardiovascular diseases (Bates et al., 2019; Daellenbach et al., 2020; Huang et al., 2012; Liu et al., 2014; Weichenthal et al., 2016). Recent studies have found aSOA to be significantly more toxic compared to other components in $PM_{2.5}$ (Bates et al., 2019; Liu et al., 2014; Puthussery et al., 2020). Daellenbach and colleagues also found that the oxidative potential of aSOA, mainly from ageing of residential biomass burning emissions, to be at least three times higher than the oxidative potential of biogenic SOA and secondary inorganic aerosols. Other studies showed similarly enhanced health effects for BC and POA, associated with combustion processes (Bates et al., 2019; Niranjan and Thakur, 2017). Transition metals like soluble copper and iron (e.g. co-emitted by coal burning), although generally present at much lower concentrations, may have relatively high oxidative potential compared to the organic species (Kajino et al., 2021; Park et al., 2018). While clinical studies on mechanisms and impacts of chronic exposure to components of $PM_{2.5}$ are still sparse, toxicological and epidemiological findings suggest that associations between adverse health effects and exposure to $PM_{2.5}$ are significantly stronger for anthropogenic carbonaceous components than total $PM_{2.5}$ mass (Grahame et al., 2014; Janssen et al., 2011; Lippmann et al., 2013; Magalhaes et al., 2018; Yang et al., 2021).

In spite of new insights into the major sources of $PM_{2.5}$, which can vary greatly by region (Health Effects Institute, 2019; Lelieveld et al., 2015; Silva et al., 2016), little is known about the global distribution of the potentially more harmful components (anthropogenic BC and organics) of $PM_{2.5}$ and their sources. Policies aiming at reducing the total $PM_{2.5}$ mass by targeting source sectors, not accounting for the specific emissions and toxicity of its components, may not optimally reduce the health burden caused by air pollution. We used a global atmospheric chemistry - general circulation model (Joeckel et al., 2010, 2005) to simulate near-surface concentrations of $PM_{2.5}$ and exposure–response functions for defined health outcomes (Murray et al., 2020) to estimate excess mortality among adults (>25 years), children (<5 years) and neonates (0–27 days). We expand upon our earlier work (Lelieveld et al., 2015), where we determined the impact of seven source sectors on excess deaths associated with exposure to $PM_{2.5}$, by using an updated emission inventory (Hoesly et al., 2018; Venkataraman et al., 2020), an improved version of the atmospheric chemistry model to identify the health impacts of ten source categories, and for the first time explicitly compute the exposure to BC, aSOA and POA and their major sources. Considering the uncertainties about the magnitude of the increased effect size of these anthropogenic organic aerosols compared with other ambient $PM_{2.5}$ pollution it is important to assess the policy implications of different plausible assumptions about relative toxicity. We performed sensitivity studies using a range of relative risk estimates based on the evidence of increased toxicity, including the high oxidative potential of the carbonaceous components of $PM_{2.5}$, that reflect the uncertainties in the magnitude of the effect.

2. Methods

2.1. Model setup and emission inventory

We used the ECHAM/MESSy atmospheric chemistry (EMAC) general circulation model at T106 horizontal spectral resolution (about 1.1°× 1.1° latitude × longitude) with 31 vertical hybrid terrain-following pressure levels up to 10 hPa in the lower stratosphere (Joeckel et al., 2016, 2005; Kerkweg et al., 2006; Lelieveld et al., 2015; Pozzer et al., 2012; Sander et al., 2005, p. 2). The core atmospheric model employed is the upgraded 5*th* generation European Centre Hamburg (ECHAM5) general circulation model. EMAC includes sub-models that represent tropospheric and stratospheric processes and their interaction with oceans, land and human influences. We used the Modular Earth Submodel System (MESSy, v.2.54 (Beer et al., 2020; Joeckel et al., 2010)) to link submodels that describe emissions, atmospheric chemistry, aerosol and deposition processes. The gas phase and heterogeneous chemistry was simulated through the MECCA submodel (Sander et al., 2019, 2011), which accounts for the photochemical oxidation of natural and anthropogenic emissions, as well as for volatile organic compounds. The GMXe submodel (Pringle et al., 2010) was used to simulate the gas/aerosol portioning and the microphysical processes in aerosols. GMXe also computes the aerosol size distribution into four hydrophilic and three hydrophobic modes.

The hydrophilic mode encompasses the entire aerosol size spectrum (i.e. nucleation, Aitken, accumulation and coarse) whereas the hydrophobic mode does not consider the nucleation mode. For this study, we updated the GMXe submodel by accounting for black and organic carbon in the accumulation and Aitken modes according to source sectors following a recent study (Paasonen et al., 2016). The fractions of black and organic carbon emissions in these modes by anthropogenic source sectors are presented in Table S1. The ORACLE submodel (Tsimpidi et al., 2018, 2014) was used to simulate the atmospheric evolution and composition of the organic aerosols. It computes the contributions of POA, aSOA and biogenic SOA to the total organic aerosols (OA) by accounting for emissions from combustion products from biofuel, fossil fuel, biomass burning and other biogenic sources. The EMAC global simulations were nudged towards ERA-Interim meteorological reanalysis data, and performed for two years: 2014 and 2015, with the first year dismissed as spin-up for the model.

We used the monthly varying Community Emissions Data System (CEDS) anthropogenic emission inventory (Hoesly et al., 2018) at 0.5°× 0.5° resolution for the primary emitted species like SO_2 (sulfur dioxide), NOx (oxides of nitrogen), CO (carbon monoxide), BC (black carbon), OC (organic carbon), NH_3 (ammonia) and speciated NMVOCs (non-methane volatile organic compounds). For India, we augmented the CEDS anthropogenic emissions data with a regional emission inventory (Venkataraman et al., 2020). Biomass burning emissions were obtained from the Global Fire Assimilation System (GFAS) inventory (Kaiser et al., 2012). The emission data were then pre-processed by distributing them over six emission heights as described elsewhere (Pozzer et al., 2009). Concentrations of ambient $PM_{2.5}$, BC, POA and aSOA were first estimated by simulating EMAC with all emission sources, and then the ten source sectors were removed one at a time, and the results linearized, to determine their contribution to ambient $PM_{2.5}$, BC, POA and aSOA, with the caveat that non-linearities in atmospheric chemistry can cause deviations from an additive response. Comparison of aerosol optical depth (AOD) and concentrations of $PM_{2.5}$, BC and OA simulated by EMAC with those from satellite retrievals, *in-situ* measurements and reanalyses data are discussed in SI Text.

The sectors considered are (a) land transportation (TRA) which includes emissions from road transportation, railways and other non-road transport, (b) industries (IND) which includes emissions from iron and steel, paper and pulp, chemical, food, solvent and other manufacturing, oil refineries and fuel production, (c) domestic energy use (DOM), i.e. the burning of solid fuels (biofuels) in households for cooking, lighting, heating and the use of liquid fuels in distributed generators, (d) energy generation (ENE) by power plants, (e) agricultural soils (AGR) that cause ammonia emissions from manure and fertilizer application and cultivation practices, (f) agricultural waste and residue burning (AWB), (g) emissions from ships and other water navigation (SHP), (h) biomass burning (BMB), which includes tropical, savanna, middle and high latitude forest fires, deforestation, peat fires, savanna and shrub fires and (i) biogenic and natural emissions from soils and plants, natural dust and sea salt (NAT). We note that BMB, which is a major emitter of black and organic carbon (2.1 teragram/year of BC and 19 teragram/year of organic carbon, which is ~ 25% of the total emission of black and organic carbon globally) are not necessarily located adjacent to major conurbations, while DOM, TRA, IND, ENE and AWB are mostly collocated with the population centers. The modelling of transition metals and their health impacts is beyond the scope of the current analysis, but we suggest that their role in the oxidative potential of $PM_{2.5}$ is addressed in future work.

2.2. Estimation of excess mortality

Calculating the burden of disease due to exposure to ambient $PM_{2.5}$ (Chowdhury et al., 2020; Lelieveld et al., 2015; Lelieveld et al., 2019; Lelieveld et al., 2020; Pandey et al., 2020) is achieved using exposure (dose) response relationships (Burnett et al., 2018, 2014; Murray et al., 2020) which assume that the harmfulness of $PM_{2.5}$ is determined solely by the inhaled mass of $PM_{2.5}$ and not by its composition. We use the recently formulated MR-BRT (meta-regression—Bayesian, regularized, trimmed) exposure–response function for the most recent Global Burden of Disease (Murray et al., 2020) to estimate the age-dependent relative risk (RR) from exposure to ambient air pollution. MR-BRT includes cause-specific risk expressions for ischemic heart disease, stroke (ischemic and hemorrhagic), chronic obstructive pulmonary disease, lung cancer and Type II diabetes among adults (population with age > 25 years), acute lower respiratory tract infection among children (population under the age of 5).

In addition to the risk expressions for adults and children, the MR-BRT also includes risk functions for excess mortality from low-birth weight and short gestation among neonates (population with age 0–27 days). The outcomes of low birth weight and short gestation include increased risk of mortality due to diarrheal diseases, upper respiratory tract infections, otitis media, meningitis, encephalitis, neonatal preterm birth, neonatal encephalopathy due to birth asphyxia and trauma, neonatal sepsis and other neonatal infections, neonatal jaundice, and other neonatal disorders.

The MR-BRT splines were fitted to relative risk and $PM_{2.5}$ exposure estimates from studies of ambient $PM_{2.5}$, household solid fuel use and secondhand smoking. Unlike the Integrated Exposure-Response functions (IER, (Burnett et al., 2014; Stanaway et al., 2018)), the MR-BRT functions were built by including multiple recent studies in high exposure settings (Hystad et al., 2019; Li et al., 2018; Yang et al., 2018; Yin et al., 2017; Yusuf et al., 2020) and excluding the active smoking-based studies. The excess mortality burden ($M(\gamma_t, age, disease)$) over a 5x5 km global grid, due to exposure to total concentration of ambient $PM_{2.5}$ (γ_t, where γ is the concentration and 't' denotes 'total $PM_{2.5}$'), was estimated by age and disease category, as in our earlier studies (Chowdhury et al., 2020; Lelieveld et al., 2019; Lelieveld et al., 2020) by

$$M(\gamma_t,age,disease) = P(age) \times BM(age,country,disease) \times \frac{RR(\gamma_t,age,disease)-1}{RR(\gamma_t,age,disease)} \quad (1)$$

where, *RR (γ_t,age,diseases)* was obtained using MR-BRT functions for all diseases by age. For IHD and stroke, age specific RRs are obtained using MR-BRT, for LC, T2-DM and COPD uniform *RRs (γ_t,diseases)* were used across all age groups among adults. *BM (age,country,diseases)* is the baseline mortality rate per 100,000 population, obtained from the GBD (http://ghdx.healthdata.org/gbd-results-tool) for all countries and kept uniform within a country at 5x5km resolution by age and disease. *P (age)* is the exposed population in a grid by age; the age distributions at 5-year intervals (adults > 25 years), 0–27 days for neonates and < 5 years for children were obtained from the Global Burden of Disease (GBD) datasets (http://ghdx.healthdata.org/record/ihme-data/gbd-2019-population-estimates-1950–2019), merged with the SEDAC population data at about 5x5 km horizontal resolution to obtain the age-specific population (*P(age)*) at the 5x5 km grid. Excess mortality $M(\gamma_t, age, disease)$ for the total mass (t) of PM2.5 was estimated separately by age for adults (at 5 year intervals for population > 25 years), neonates and children (under 5 years of age). $PM_{2.5}$, BC, POA and aSOA simulated by EMAC were statistically down-scaled, and all further analyses were performed at ~5 km spatial resolution. Subsequently, the results were aggregated and presented at the country level. For comparison, we also used the Global Exposure Mortality Model (GEMM (Burnett et al., 2018)) to estimate age-dependent hazard ratio (relative risk) functions and excess mortality for exposure to $PM_{2.5}$ for all non-communicable diseases (NCD) and lower respiratory infection (NCD + LRI) among adults (>25 years) and children (<5 years) to test the sensitivity of results to different exposure–response functions. The GEMM exposure–response function and associated results are described in the SI Text.

The exposure to ambient $PM_{2.5}$ can result in a chronic oxidant/antioxidant imbalance in the respiratory system, causing oxidative stress and inflammatory responses, with implications for the etiology of several common respiratory and cardiovascular diseases in humans. Oxidative stress might occur directly by inhalation of reactive oxygen species in $PM_{2.5}$, or indirectly from their catalytic generation within the body upon inhalation of toxic $PM_{2.5}$ compounds, eg. co-emitted by combustion sources. Recent studies have assessed the short-term exposure to aSOA, BC and POA to be significantly more hazardous than unpolluted desert dust, biogenic SOA and many inorganic species (Bates et al., 2019; Baumgartner et al., 2014; Cho et al., 2005; Chung et al., 2006; Daellenbach et al., 2020; Fang et al., 2017; Lin and Yu, 2011; Park et al., 2018; Puthussery et al., 2020; Verma et al., 2015; Wang et al., 2018), resulting from their enhanced oxidative potential. While aSOA is formed photochemically within the atmosphere, the primary BC and POA particles may carry harmful combustion products, including PAHs and transition metals (Bates et al., 2019; Charrier and Anastasio, 2011; Cho et al., 2005; Chung et al., 2006), to a large degree emitted in the ultrafine size mode (<100 nm), with the potential to directly translocate into the bloodstream upon inhalation (Miller et al., 2017).

The difference between excess mortality associated with exposure to the total $PM_{2.5}$ (γ_t) and (γ_t - γ_x), where, γ is the concentration and $\times$ =(*BC,POA,aSOA*) was attributed based on the exposure to species *x*. We note that, similar to the Integrated Exposure-Response function (IER)[5] and GEMM[4], the MR-BRT was also formulated by assuming equal toxicity among the different constituents of $PM_{2.5}$. With this assumption, we hypothesize that the toxicity increases with the inhaled dose (we assume *inhaled dose* $\propto$ *exposure*). Hence to assume a species *x* to be *n* times more toxic, the concentration of the species was multiplied by *n* and the concentrations of the remaining species in $PM_{2.5}$ were decreased accordingly. The excess mortality ($M_x(\gamma_x, \gamma_t, age, disease)$) that can be attributed to species *x* being considered *n* times more toxic is written as in Eq. (2)

$$M_x(\gamma_x,\gamma_t,age,disease) = M(\gamma_t,age,disease) - M(\gamma_t - (n\gamma_x),age,disease) \quad (2)$$

The total excess mortality burden ($M(\gamma_t, age, disease)$) was prescribed to remain identical, while the sector attribution to $PM_{2.5}$ related mortality burden changes, i.e. the sectors which contribute to excess mortality from species *x* amplifies by proportion of their contribution to $M_x(\gamma_x, \gamma_t, age, disease)$, compared to the other sectors. As there are limited cohort studies of the long-term health burden from BC, POA and aSOA, based on recent evidence about their higher oxidative potential from toxicological studies and epidemiological findings of short-term health impacts, we assess the implications of assuming them to be more harmful compared to other $PM_{2.5}$ species. Therefore we include five sensitivity tests: (a) we have assumed these anthropogenic carbonaceous species to be about twice as hazardous per $\mu g/m^3$ as other components of $PM_{2.5}$ (2BSP) (b) 1p5BS BC and aSOA were assumed to be 1.5 times (50%) more hazardous than the other species including POA, (c) 2BS - BC and aSOA were assumed to be twice (100%) as hazardous as the other species including POA, (d) 3BS - BC and aSOA were assumed to be three times (200%) as harmful as the other species including POA, and (e) 1p5BSP - BC, aSOA and POA were assumed to be 1.5 times (50%) more hazardous than the other species. Though we use the 2BSP assumption to discuss the relative health impacts based on recent toxicological and short-term epidemiological health impact studies, we emphasize that this should be further investigated in extended epidemiological cohort studies that link the exposure of different $PM_{2.5}$ components to chronic health outcomes. The present work does not provide evidence for the level of harmfulness of individual $PM_{2.5}$ components, but illustrates the important role of differential toxicities in the

assessment of health effects as a basis for policy making.

3. Results

3.1. Excess mortality from ambient $PM_{2.5}$

We used the EMAC global atmospheric chemistry model (Joeckel et al., 2010, 2005; Lelieveld et al., 2015), evaluated against multiple measurement datasets, to simulate surface concentrations of $PM_{2.5}$, organic aerosols and BC (SI Text, Fig. S1-S5). We estimate 4.23(95% confidence intervals 3.0–6.14) million excess deaths per year from exposure to ambient $PM_{2.5}$ globally using the the MR-BRT (meta-regression—Bayesian, regularized, trimmed) splines which were also used in the most recent Global Burden of Disease (GBD) study (Murray et al., 2020), of which 92%, 5%, and 3% occur among adults, neonates and children, respectively (Fig. S6). Fig. 1 depicts the excess mortality burden from exposure to ambient $PM_{2.5}$ by country. China (1.44 (1.1–1.89 CI:95%) million) is estimated to have the highest annual excess mortality followed by India (0.85(0.63–1.19) million) and Pakistan (0.15(0.1–0.25) million). Table 1 lists the health burden among adults, neonates and children for the top ten countries ranked by total excess mortality. Results for all countries are presented in SI DataS1.

We estimate that globally 91(66–126) adults per 100,000 and 49 (23–111) neonates and children per 100,000 die prematurely each year from the exposure to ambient $PM_{2.5}$, respectively (See Fig. S7 for results by country). Globally, ischemic heart disease (IHD(38%)) and stroke (32%) are the leading causes of death in adults followed by chronic obstructive pulmonary disease (COPD(15%)), lung cancer (LC(8%)) and type-2 diabetes mellitus (T2-DM(7%)). The distribution of excess mortality by adults, neonates and children can vary tremendously by country with a high proportion of neonatal and child mortality in emerging and middle income countries in Africa and South Asia (Fig. 1). For example, in the African countries Niger, Chad and Nigeria (SI DataS1), 59%, 55% and 54% of the total excess mortality occurs among children and neonates, and in India and Pakistan the corresponding percentages are 10% and 26%, respectively. India has the largest absolute excess neonatal and child mortality burden, accounting for 30% and 25% of the global total neonatal and child mortality from ambient air pollution, respectively. For China and high-income countries in North America, Western Europe, <1% of the total excess mortality from ambient $PM_{2.5}$ occurs among children and neonates (SI DataS1,S2). Our global estimates for disease-specific causes agree with the recent GBD study (Murray et al., 2020), but are considerably lower than those by studies using the Global Exposure Mortality Model (GEMM) (Burnett et al., 2018; Chowdhury et al., 2020; Lelieveld et al., 2019; Lelieveld et al., 2020) to estimate excess mortality resulting from all NCDs. The estimates using GEMM is presented as sensitivity analysis (discussed in the SI Text).

3.2. Source attribution

We determined the relative contributions of ten source categories, using the updated EMAC model and applying the improved global emission inventory. The sectoral contributions indicate the potential health benefits from their phase-out (in terms of avoidable excess deaths). The ten countries with highest numbers of excess deaths attributable to ambient air pollution are listed in Table 1 along with the source sectors. DOM is the leading anthropogenic sector worldwide, accounting for about 20% of the total excess mortality burden from ambient air pollution (Fig. 2, Fig. S7). DOM is a large contributor to

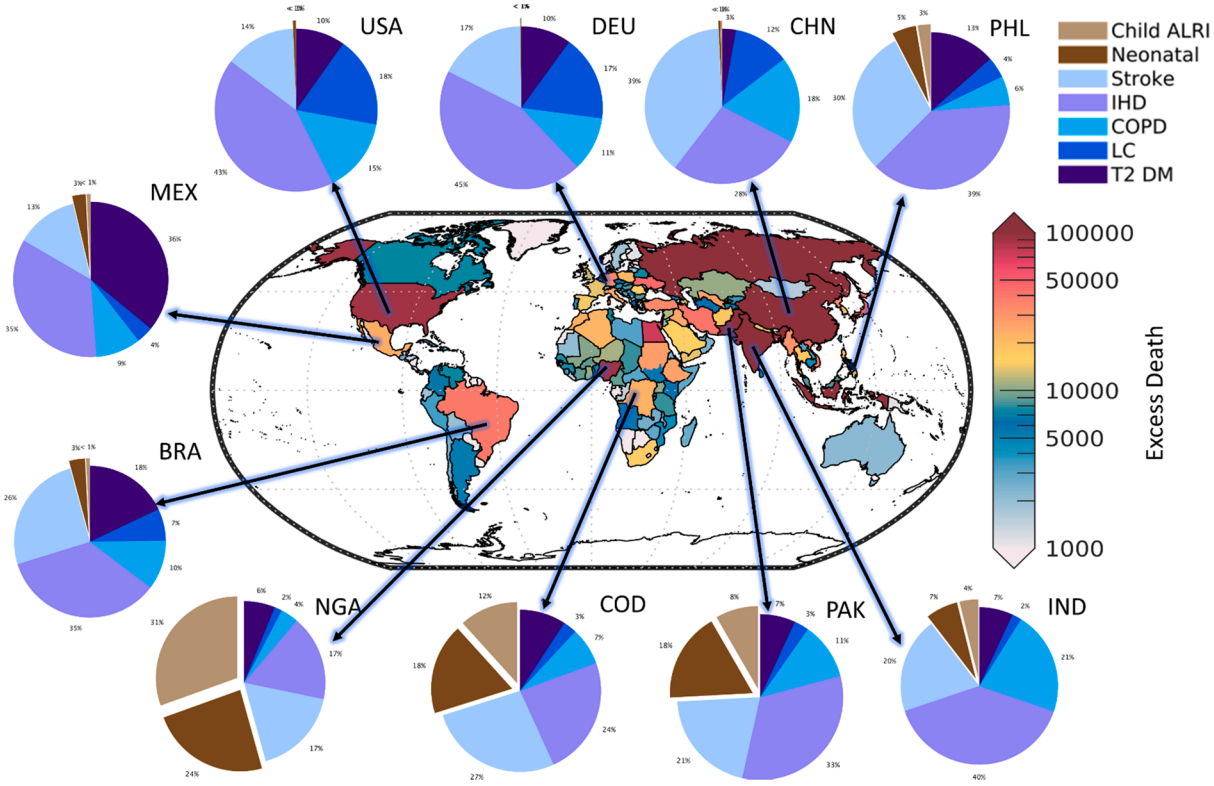

Fig. 1. Excess mortality due to the long-term exposure to ambient $PM_{2.5}$. Pie charts for ten countries identified by their ISO codes are depicted: USA (United States of America), DEU (Germany), CHN (China), PHL (Philippines), IND (India), PAK (Pakistan), COD (Democratic Republic of Congo), NGA (Nigeria), BRA (Brazil), and MEX (Mexico). Pie charts depicting percentage total excess mortality among adults, neonates, and children are presented in Supplementary Data 1. Pie charts show the global distribution of excess mortality burden from stroke, ischemic heart disease (IHD), chronic obstructive pulmonary disease (COPD), lung cancer (LC), and type-2 diabetes mellitus (T2 DM) among adults (>25 years), acute lower respiratory tract infections (ALRI) in children (<5 years), and diseases related to preterm birth and low birth weight among neonates (0–27 days). Pie charts for all the countries are presented in SI Data 2.

Table 1
Excess annual adult, neonatal and child mortality for the top ten countries, ranked by total mortality burden, and sector contributions from BMB(biomass burning), AWB(agricultural waste burning), IND(industries), ENE(energy), DOM(domestic), SHI(ships), WST(waste incineration), TRA(transportation), AGR(agricultural soils) and NAT(natural and biogenic) to total excess mortality associated with exposure to ambient $PM_{2.5}$, the second row for each country shows results for the 2BSP scenario.

Country	Excess Mortality Burden (95 %CI) *			Sector contributions (%)									
	Adults	Neonates	Children	BMB	AWB	IND	ENE	DOM	SHI	WST	TRA	AGR	NAT
China	14.31(10.95–18.74)	6.8(3.3–14)	4.5(3.1–6.7)	3	<1	20	17	22	<1	<1	4	20	13
				4	1	22	21	35	1	<1	5	7	5
India	7.66(5.82–10.1)	56.5(24.1–135.8)	33.1(21.2–51.8)	1	3	10	9	27	<1	<1	5	7	38
				1	5	12	10	46	<1	<1	7	3	16
Pakistan	1.15(0.83–1.61)	27.4(11.1–67.6)	13.1(8.2–20.4)	<1	2	3	1	16	<1	<1	3	3	70
				<1	4	4	2	25	<1	<1	4	3	58
Indonesia	1.13(0.78–1.67)	3.2(1.1–9.9)	1.1(0.6–1.9)	33	<1	5	7	25	1	<1	10	3	17
				38	<1	5	7	36	1	<1	12	<1	<1
Russian Federation	1.15(0.8–1.64)	0.2(0.09–0.7)	0.12(0.07–0.18)	8	3	8	17	14	1	<1	5	11	33
				12	4	8	18	19	1	<1	7	8	24
Nigeria	0.42(0.28–0.62)	21.9(9.3–52.3)	28.1(17.3–45.5)	7	<1	2	<1	35	<1	<1	2	11	53
				11	1	2	1	62	<1	<1	3	<1	21
Bangladesh	0.81(0.54–1.24)	5.1(1.7–15.6)	3.1(1.8–5.2)	2	<1	9	16	30	<1	<1	8	13	20
				4	1	10	17	53	<1	<1	10	2	3
USA	0.85(0.58–1.24)	0.5(0.2–1.4)	0(0–0.05)	12	<1	10	15	13	3	<1	11	12	24
				18	1	11	16	20	3	<1	13	6	12
Egypt	0.73(0.53–1.01)	0.8(0.3–2.9)	2.4(1.4–4)	<1	<1	2	5	3	<1	<1	4	3	83
				<1	<1	2	5	4	1	<1	6	2	79
Japan	0.48(0.34–0.70)	0.03(0–0.08)	0(0–0)	5	<1	20	20	6	2	<1	8	18	19
				6	1	21	24	9	3	<1	9	13	14
EU-27[#]	1.8(1.2–2.5)	0.4(0.1–1.1)	0.07(0.04–0.1)	1	<1	6	12	19	4	<1	10	18	27
				2	<1	7	14	27	4	<1	12	12	20
Rest of the World	8.5(5.7–12.7)	72(25–219)	51(29–90)	10	<1	6	7	13	1	<1	4	5	53
				14	1	7	7	19	1	<1	6	3	41
World	39(28.3–54.1)	195.5(77–521)	136.5(83–226)	5	1	12	11	20	1	<1	5	11	33
				7	2	13	13	32	1	<1	6	5	20

*Age-adjusted adult excess mortality x100,000. Neonatal and child excess mortality x 1,000.
[#]EU-27 includes Austria, Belgium, Bulgaria, Croatia, Cyprus, Czechia, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, Lithuania, Luxemburg, Malta, Netherlands, Poland, Portugal, Romania, Slovakia, Slovenia, Spain, and Sweden.

ambient air pollution in populous countries of South Asia where solid fuel use in households is prevalent. In Nepal, Bangladesh and India DOM accounts for 34%, 30% and 27% of the mortality burden. It is also predominant in the African countries Rwanda (44%), Burundi (44%) and Nigeria (35%), for example. In countries where domestic solid fuel burning is important, the child and neonatal mortality from ambient air pollution is highest. In Europe and North America the contribution of DOM is typically around 15%, while in Germany and Poland contributions are 19% and 26%, respectively. Fig. 2 depicts the sectoral contributions to ambient $PM_{2.5}$ by country, and data for all countries are listed in SI DataS3. IND is the second largest contributor (nearly 12% globally) to ambient $PM_{2.5}$ related excess mortality, with large contributions in East Asian countries such as China (20%), South Korea (20%) and Taiwan (21%). IND is also a large contributor in Europe and North America where it contributes > 8% to excess mortality from ambient air pollution.

Fine particulate constituents formed through ammonia emissions from AGR account for>11% of the global mortality burden (Fig. S8), being a major source in Korea and most of Europe (where AGR is the leading contributor). In China and India, AGR accounts for 20% and 7% of the total mortality burden, respectively. ENE is also a main contributor to the ambient $PM_{2.5}$ related mortality burden (>11% globally). In the East Asian countries Taiwan, Japan, Korea and China its contribution is > 16%. ENE is the largest anthropogenic source in the USA (15%). NAT is the largest contributor worldwide, accounting for 33% of the total deaths from ambient $PM_{2.5}$ exposure. NAT is the leading source in North African countries where desert dust levels are high. Our calculations suggest that TRA is an intermediate contributor to excess mortality burden (5% globally); however, in Europe and North America it accounts for>10%. On the other hand, nitrogen oxides from traffic are a main cause of pediatric asthma (Chowdhury et al., 2021) and strongly contribute to tropospheric ozone formation, not considered here. BMB is a relatively small contributor globally (5%), but with major local health impacts. It is the leading source in Central and East African and South-East Asian countries where it contributes > 40% to the mortality burden. BMB is also a large contributor in South American countries. SHP, AWB and WST together account for ~2% of the global excess mortality from ambient $PM_{2.5}$ exposure. In Scandinavian countries SHP accounts for > 12% of the excess mortality.

3.3. Black carbon

Global population-weighted mean BC exposure was estimated at 1.4 $\mu g/m^3$, with the highest level in China (2.8 $\mu g/m^3$), South Korea (2.7 $\mu g/m^3$), Nigeria (2.3 $\mu g/m^3$) and India (2.0 $\mu g/m^3$). In high-income countries in North America and Europe, the population-weighted BC exposure is relatively lower (<0.5 $\mu g/m^3$). The spatial distribution of global BC concentrations from the EMAC model is depicted in Fig. 3A. Globally, out of 1,000 excess deaths from exposure to $PM_{2.5}$, 35(24–49) are attributed to exposure to BC alone. We estimate about 150,000 (106,000–214,000) excess deaths per year from exposure to BC under assumption of no increase in toxicity compared with undifferentiated $PM_{2.5}$, 94% of which occurs among adults and 3% each among neonates and children. About 38% and 22% of the global excess deaths from BC exposure occur in China and India, respectively (see also Table S2 and Fig. 4A, and data for all countries in SI DataS4). The rate was estimated to be considerably higher in Argentina and South Africa, being 94 (60–151) and 83(55–132), respectively per 1,000 deaths from $PM_{2.5}$ exposure. For China and India the corresponding rates are 40(30–52) and 38(28–53), respectively. DOM and TRA were estimated to be the largest sources of BC (Fig. 4B, S9, Table S2, sector contributions by country are listed in SI Data S4) which agrees with previous findings

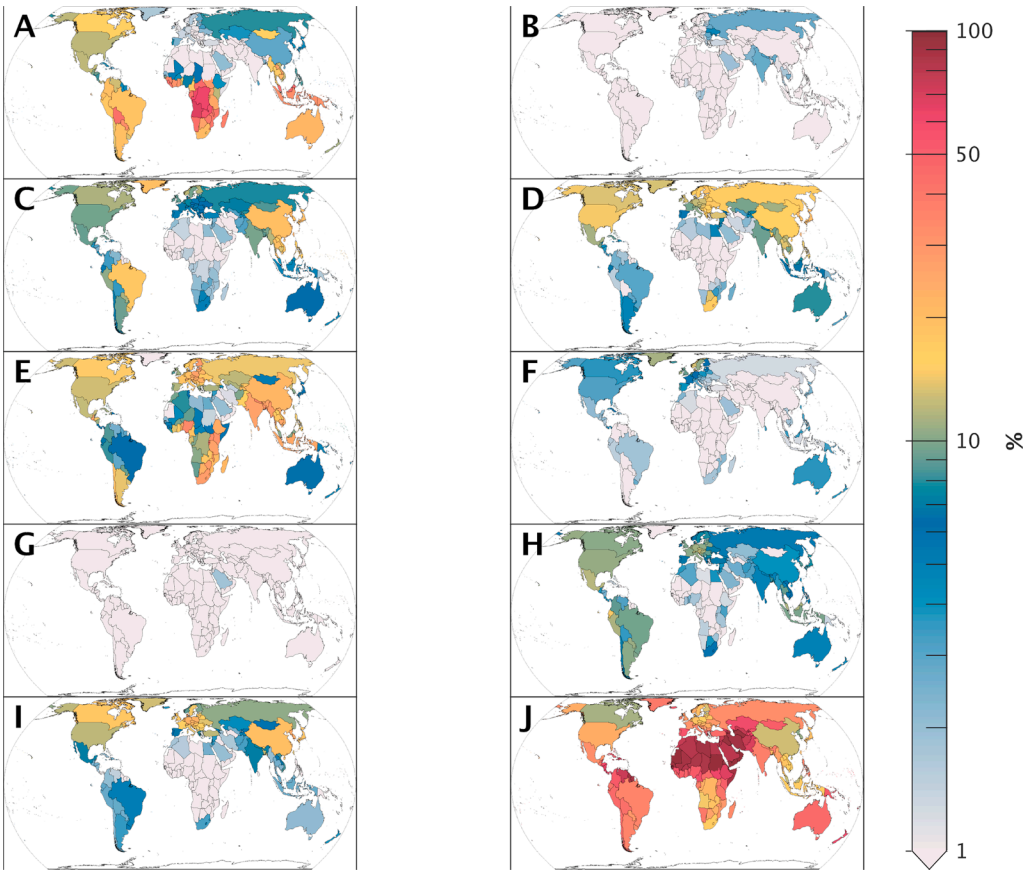

Fig. 2. Percentage contribution by source sectors (A) BMB, (B) AWB, (C) IND, (D) ENE, (E) DOM, (F) SHP, (G) WST, (H) TRA, (I) AGR, and (J) NAT to excess mortality from ambient $PM_{2.5}$ exposure. Data for all countries are listed in SI Data 3.

(Anenberg et al., 2011; Janssen et al., 2011; Li et al., 2016), accounting for 62% and 18%, respectively, of the global excess mortality (ie. out of 35 (24–49). BC related excess deaths per 1,000 due to $PM_{2.5}$ exposure, 28 (19–39) can be attributed to these two sectors globally. DOM was found to account for 63% and 76% of the total BC related excess deaths in China and India, respectively, while the contribution of TRA is 13% in both these countries.

In the UK and the USA, TRA is the largest source sector, accounting for 45% and 41% of the total number of excess deaths from BC. TRA is also the largest sector for BC related excess deaths in West Asian and North African countries, where dust levels are relatively high (>70%). ENE accounts for 8% of the total excess deaths from BC exposure. It is a large source of BC related deaths in the East Asian countries Taiwan (30%), Japan (24%), South Korea (20%) and China (17%). In the West European countries Germany, Belgium and France the contribution is ~10%. IND accounts for a comparably small proportion of BC related deaths globally (6%); however, it is a significant source in Brazil (14%), Japan (13%), the Philippines (12%) and the United Kingdom (12%). BMB is the largest source of BC-related excess deaths in West and Central African countries where it accounts for > 50% of the total BC-related health burden. AWB, SHP and WST were identified as relatively small sources, although AWB is a significant source in Ukraine (20%), the Russian Federation (11%), and Pakistan (7%). The contribution of SHP to BC-related excess deaths is estimated to be relatively high (>30%) in island nations and Scandinavian countries.

3.4. Primary organic aerosols

Organic aerosol (OA) particles in the atmosphere were partitioned into POA, aSOA and biogenic SOA using the ORACLE submodel in EMAC (Tsimpidi et al., 2018, 2014). The spatial distribution of total OA is depicted in Fig. S5a. Population-weighted mean global exposure to POA, which is directly emitted into the atmosphere from fossil and solid fuel combustion and biomass burning was estimated at 1.9 μg/m^3 (the spatial distribution of POA is depicted in Fig. 3B). The estimated POA concentrations estimated were lower than those in earlier studies (David et al., 2018) possibly arising from significant re-partitioning of POA compounds to the gas phase computed by the ORACLE submodel (Tsimpidi et al., 2018), with dilution during the transport of air masses (Robinson et al., 2007; Tsimpidi et al., 2018). The largest population-weighted POA exposure was estimated to occur in the West and Central African countries of Sierra Leone (7 μg/m^3), Nigeria (5 μg/m^3) and Democratic Republic of Congo (5 μg/m^3) where biomass burning activity is high. China (4 μg/m^3), Pakistan (3 μg/m^3), Indonesia (3 μg/m^3) and India (2 μg/m^3) were also estimated to have high population weighted POA exposure. We calculated 206,000(146,000–305,000) excess deaths per year from POA exposure globally, of which 38% and 15% occur in China and India (Fig. 5A), respectively. Globally, DOM and BMB were identified as the leading sources of POA-related deaths (Fig. 5B, S10, Table S3 and sector contributions by country are listed in SI DataS5). Out of the 48(34–72) per 1,000 excess deaths from exposure to $PM_{2.5}$, DOM and BMB were found to be responsible for 29(20–44) and 11(7–16) deaths. The other sectors together account for 8(5–11) deaths.

In India and China, 80% and 68%, respectively, of the total excess deaths from POA exposure are attributed to DOM. It is also a major source sector in (>40%) in North America and Europe. In Central, West and East African countries, BMB is the largest source (>80%). BMB is the largest contributor to POA-related deaths in the USA (47%). ENE contributes 7% to the total POA related number of excess deaths globally. It is a major source of POA-related deaths in the East Asian countries Taiwan (32%), South Korea (25%), and China (16%). IND and TRA contribute 4% and 3% to global POA related deaths, respectively. TRA is

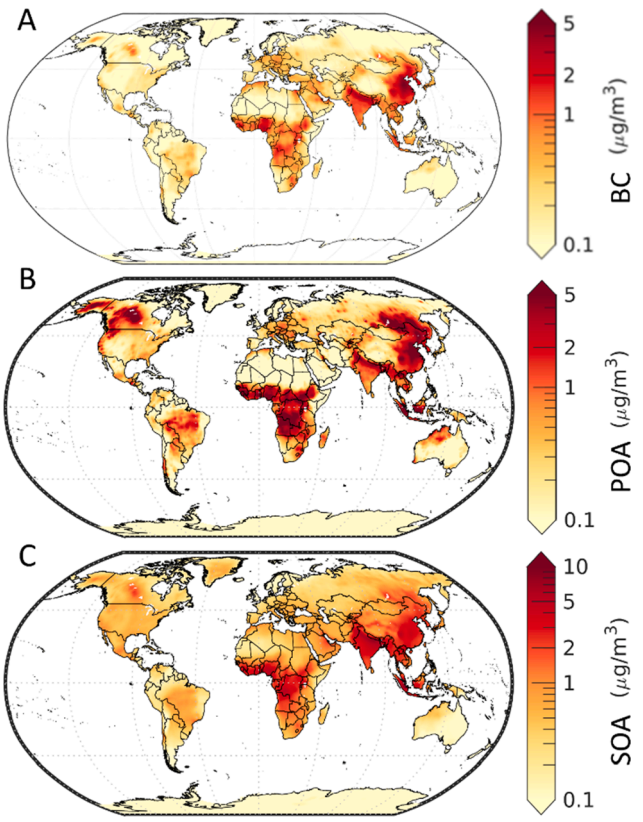

Fig. 3. Spatial distribution of (A) black carbon, (B) primary organic aerosols and (C) anthropogenic secondary organic aerosols.

estimated to be the largest source of POA-related excess death in the dust dominated West Asian and North African countries (>40%). In the United Kingdom and Germany, TRA was found to account for 30% and 12% of the total POA related excess deaths, respectively. AWB, SHP, and WST together account for ~2% of the total POA related deaths. AWB is a major source in Ukraine (20%), Pakistan (12%) and India (8%). SHP is only a very small contributor to POA-related mortality, even in island countries.

3.5. Anthropogenic secondary organic aerosols

SOA is formed in the atmosphere from the oxidation of gas-phase precursors, ie. volatile organic compounds (VOC). The ORACLE submodel (Tsimpidi et al., 2018, 2014) in EMAC differentiates between SOA formed from VOCs, semivolatile organic compounds and intermediate-volatility organic compounds; the submodel also tracks SOA concentrations from anthropogenic and biogenic emissions, which facilitates the distinction of aSOA (from fossil and solid fuel burning and biomass burning) from biogenic SOA. The ORACLE submodel also tracks how SOA concentrations are affected by emissions, and can be used to evaluate the effects of photochemical aging and long-range transport on the organic aerosol budget. As the POA upon emission is highly sensitive to ambient conditions, including dilution and temperature, ORACLE also accounts for the volatility of POA emissions and evaporation of POA to form SOA (Robinson et al., 2007; Tsimpidi et al., 2014). Population-weighted global mean aSOA was estimated at 2.2 μg/m^3 (the spatial distribution is depicted in Fig. 3C). Nepal (5 μg/m^3), India (4.5 μg/m^3), Bangladesh (4.5 μg/m^3) and Pakistan (4 μg/m^3) were estimated to have the largest population-weighted aSOA exposure. Globally, we estimated 260,000(183,000–384,000) excess deaths per year due to exposure to aSOA. India and China have the largest mortality burden due to aSOA (Fig. 6A, 30% and 24% of the global total, respectively). Table S4 lists the excess mortality from aSOA exposure for the countries with the highest mortality burdens from $PM_{2.5}$ (see Fig. 6A for the distribution by country, and data for all countries are provided in SI DataS6). Globally, out of 1,000 excess deaths from $PM_{2.5}$ exposure, 61(43–90) are attributed to aSOA exposure.

In Malaysia, Indonesia and Thailand the corresponding rates are 205 (129–327), 193(130–292) and 173(110–273), respectively. In the USA (73(50–107)), China (43(33–57)) and Germany (28(19–42)), these rates are relatively lower. DOM is the largest contributor to SOA related excess mortality in South Asia (>60%), China (>40%) as well as West and East European countries (Fig. 6B, Fig. S11). ENE is the second largest source, accounting for 13% of the total aSOA-related deaths. ENE is the largest contributor in the East Asian countries Korea (44%), Japan (41%) and Taiwan(41%). It is also a significant contributor in China (31%), Germany (27%), the UK (27%) and the USA (18%). BMB is the next largest contributor, accounting for 12% of the total aSOA-related deaths. It is the leading contributor in Central and West Africa, South America, and Southeast Asia. IND accounts for ~10% of the total aSOA related excess deaths with large contributions in the South American countries Brazil (41%) and Uruguay (33%). It is also a significant contributor in China and India (>10%). In West Asia and North Africa, TRA (global contribution 9%) is the largest source. AWB is a relatively small source (~6%), however, with a sizable share in Ukraine (23%), Pakistan (14%), the Russian Federation (13%) and India (11%). SHP and WST together account for < 1% of the total aSOA related deaths.

3.6. Sensitivity study on relatively high toxicity of BC, POA, aSOA

Based on recent evidence about their higher oxidative potential from toxicological studies, and epidemiological findings of short-term health impacts, we assess the implications by assuming BC, POA and aSOA to be about twice as harmful compared to other $PM_{2.5}$ species (2BSP). We

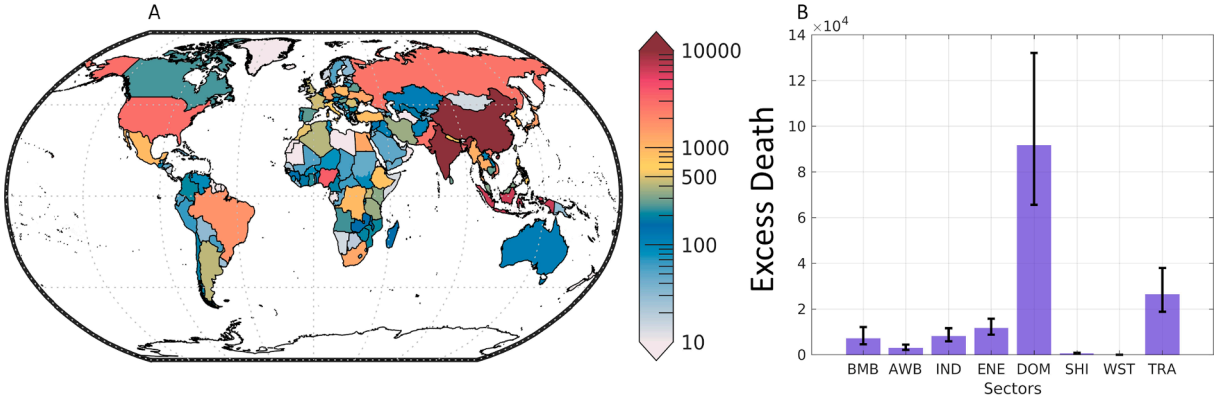

Fig. 4. (A) Spatial distribution of annual excess mortality from ambient BC exposure. (B) Global source contribution to excess mortality from ambient BC exposure.

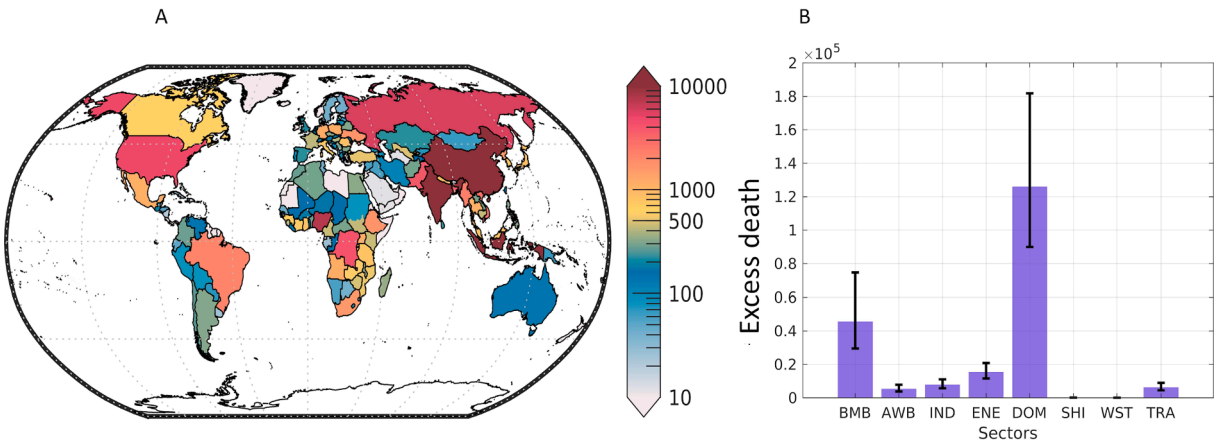

Fig. 5. (A) Spatial distribution of annual excess mortality from ambient POA exposure. (B) Global source contribution to excess mortality from ambient POA exposure.

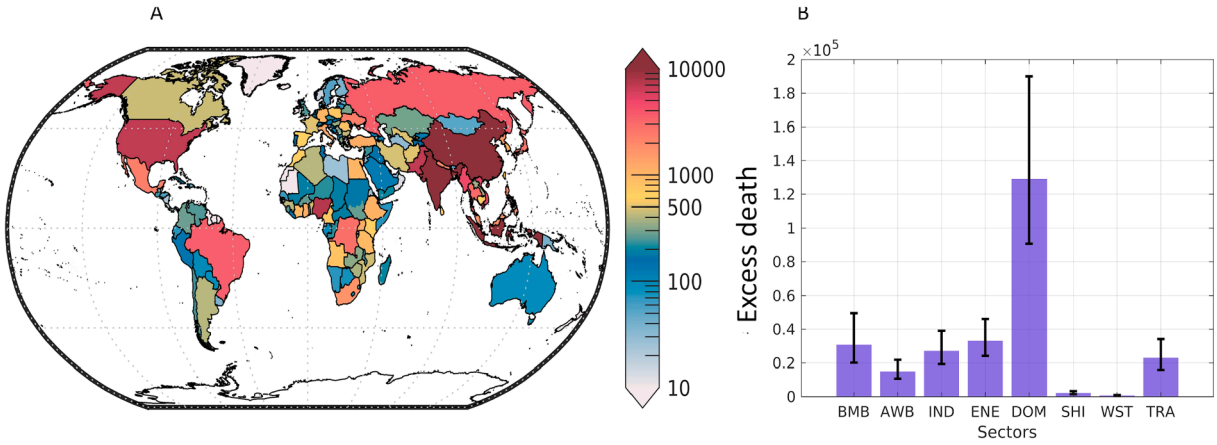

Fig. 6. (A) Spatial distribution of annual excess mortality from ambient aSOA exposure. (B) Global source contributions to excess mortality from ambient aSOA exposure.

also present calculations with alternative assumptions, showing that the policy implications of our results are not sensitive to the exact magnitude of harmfulness of carbonaceous species. If we assume that all species in $PM_{2.5}$ are equally health hazardous (EqT, Fig. S8, Fig. 7), we attribute 144(101–211) out of 1,000 excess deaths from exposure to $PM_{2.5}$ to BC, POA, and aSOA globally, which increases to 364(257–533) and further to 380(274–552) with the 2BSP and 3BS assumptions respectively. Globally, DOM contributes 58% to the total excess mortality from BC, POA, and aSOA followed by BMB (13%), ENE (9.5%), TRA (9%), IND (7%), and AWB (3.5%), and SHP and WST together contribute $< 1\%$. Therefore, under all the five sensitivity assumptions the contribution of these sectors to $PM_{2.5}$ mortality increases substantially, while the contributions of NAT and AGR (with limited impact on the atmospheric concentrations of BC/POA/aSOA) decrease steeply.

Under the 2BSP and the 3BS assumptions (Fig. 7), DOM emerges as the globally largest cause of excess mortality from ambient air pollution; completely mitigating DOM would avert 32% and 34.5% of the excess mortality from ambient $PM_{2.5}$ under the 2BSP and 3BS assumptions, respectively, compared to 20% under EqT, besides controlling for 2.3 (1.6–3.1) million excess mortality attributed to household air pollution exposure. The contributions of NAT and AGR decrease strongly, to 20% and 5% under the 2BSP, and 17.5% and 4% with the 3BS assumptions, respectively, more consistent with empirical data of the lower health risk by these sources (Table 1). With the 1p5BS, 2BS and 1p5BSP, NAT remains the largest source sector globally. The excess mortality from BMB and TRA, also being important sources of BC, POA and aSOA, increase significantly by about one third with the 2BSP assumption, while the contributions from IND and ENE (relatively smaller sources of BC, POA and aSOA) increase by 10% and 15%, respectively (Fig. 7). Note that these sector contributions vary with the major sources of BC, POA and aSOA, which can differ considerably by country (SIDataS1). For example, in the USA, ENE (14%) is the largest anthropogenic contributor to $PM_{2.5}$-related excess mortality under the EqT assumption, whereas the strong emissions of BC, POA and aSOA from DOM (20%) make it the largest contributor under the 2BSP assumption. This corresponds with the increasing role of non-fossil fuel related emissions of organic compounds in the USA (McDonald et al., 2018). Consequently, the contributions of NAT and AGR decrease by a factor of two. Similarly, in West Europe, phasing out AGR emissions (which contribute 21% to $PM_{2.5}$) would reduce excess mortality by 13%, while the phasing out of DOM and TRA emissions could avoid 41% of the excess mortality burden under the 2BSP assumption.

Under the 2BSP assumption DOM becomes the largest contributor in India (46%), where the role of NAT declines by more than a factor of two relative to EqT. DOM also emerges as a leading contributor ($>25\%$) in Germany and France, followed by ENE ($>13\%$) and TRA ($>12\%$). Under the 2BSP assumption, AGR remains important for excess mortality ($\sim 10\%$) in Europe, but its contribution decreases by nearly one third compared to EqT. In South Korea and Japan, ENE and IND remain the largest contributors (20% increase compared to EqT). In China, the contributions from NAT and AGR decrease to $\sim 7\%$, while the contribution of DOM increases to 34%. In West Asia and North Africa, with

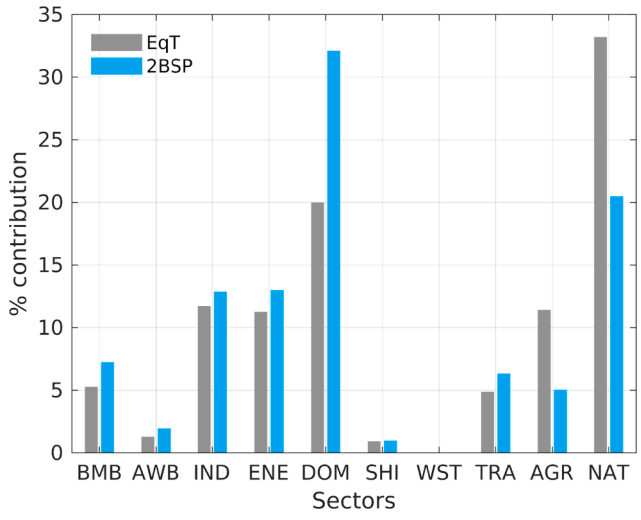

Fig. 7. Contributions of major source sectors to excess mortality from $PM_{2.5}$ under the EqT (all components of $PM_{2.5}$ are considered to be equally toxic) and 2BSP (black carbon, anthropogenic secondary secondary organic aerosols and primary organic aerosols considered to be twice as harmful as other components of $PM_{2.5}$ assumptions, showing the largest differences for DOM, AGR and NAT. The sectors ats x-axis are BMB- Biomass burning; AWB- Agricultural waste burning; IND- Industries; ENE- Power generation; DOM- Domestic solid fuel burning and other commercial activities; SHI- Ships; WST- Waste Incineration; TRA- Transportation; AGR- Agricultural soils; NAT- Natural and biogenic emission.

high levels of desert dust, NAT remains the largest source. In West, Central and East Africa BMB is the predominant source of excess mortality. Table 1 lists the sector contributions under the 2BSP assumption for the top ten countries with the largest excess mortality burdens (see Fig. S12, SI DataS3,S7 for all countries and major regions). The sensitivity calculations for the other four different assumptions show that these results are robust (SI Text, Fig. S13). Our results suggest that accounting for the toxicity of $PM_{2.5}$ components has major policy implications.

3.7. Limitations and uncertainties

The 95% CI's presented in this study are estimated by combining the uncertainties in baseline mortality rates obtained from the GBD and those for the MR-BRT splines used to obtain relative risk as in a previous study (Chowdhury et al., 2020). We acknowledge that the additional uncertainties associated with the modeling of BC, POA and aSOA are not included in the confidence intervals, but note that the comparison with measurements is generally favorable (SI Data). Here (Fig. S3) and in previous work (Lelieveld et al., 2019; Lelieveld et al., 2015) we have shown that our model captures $PM_{2.5}$ concentrations well, and that especially the correlations with observed annual averages (which smooth variability related to the weather conditions) is very high, which is most relevant in the current application as we focus on chronic impacts of $PM_{2.5}$, and this metric is applied in the the MR-BRT functions. A model comparison with ground-based remote sensing data from the global AERONET network indicated an R^2 close to 0.8 and negligible bias (Fig. S1). The need to capture small-scale spatial and temporal variabilities from emissions in $PM_{2.5}$ is limited, as the lifetime of the particles is about a week and secondary constituents, which often dominate the aerosol composition, are formed during atmospheric transport. This is evident from the small urban increments observed between $PM_{2.5}$ at measurement sites in cities and their environment (Lelieveld et al., 2015). For this reason, we found in previous work that the resolution of $PM_{2.5}$ calculations, varied between 10 km and 100 km (the latter comparable to our model results at $1.1^{\circ} \times 1.1^{\circ}$) does not significantly affect the $PM_{2.5}$ exposure calculations, and that the uncertainties of excess mortality estimates are dominated by the parameters of the response functions (Kushta et al., 2018). Nevertheless, these studies have generally been performed for high- and middle-income countries that have access to air quality data, while emission inventories and the lack of measurement data in low-income countries are associated with much larger uncertainty (Crippa et al., 2019, 2018; Kushta et al., 2018), which is difficult to quantify. Recent comparisons with other emission data have shown that the CEDS emission inventory has slightly higher BC and organic carbon emissions, particularly for China and Africa (Crippa et al., 2019, 2018; McDuffie et al., 2020). While it should be noted that the emission estimates in general have similarly large uncertainties for organic carbon, BC and DOM emissions, we acknowledge that the use of different emission inventories will influence the calculated BC, aSOA and POA exposures, which has not been accounted for in this work.

Multiple recent studies have indicated that exposure to primary and secondary organic particles is more hazardous to human health than the total mass of inorganic $PM_{2.5}$ (Bates et al., 2019; Baumgartner et al., 2014; Daellenbach et al., 2020; Fang et al., 2017; Lin and Yu, 2011; Park et al., 2018; Puthussery et al., 2020; Verma et al., 2015). To account for this in our sensitivity calculations, we adopted a range of relative health impacts. Daellenbach and colleagues (Daellenbach et al., 2020) established that exposure to anthropogenic SOA (aSOA) causes three times higher oxidative stress than exposure to secondary inorganic aerosols and biogenic SOA. Some studies (Bates et al., 2019; Charrier and Anastasio, 2011; Cho et al., 2005; Chung et al., 2006) indicated that oxidative stress might occur by the inhalation of reactive oxygen species deposited on primary emitted particles (POA or BC), or the catalytic generation of reactive oxygen species within the body upon inhalation of particles that contain substances such as quinones and transition metals. However, these studies focussed on the impacts of short-term exposure, mostly in traffic/curbside locations. Moreover, the majority of the acute impact studies do not consider the temporal variation of the exposure covariates as well as their large scale geographical variation. Though, few recent studies have reported on morbidity and mortality risk associated with long-term exposure to BC (Beelen et al., 2008; Chung et al., 2015; Hvidtfeldt et al., 2019; Ljungman et al., 2019; Yang et al., 2021), there are inconsistencies on the reported increased risk compared to all component $PM_{2.5}$. Such inconsistecies may be associated with differences in study designs and methodology and because limited evidences on the chronic impacts of aSOA and POA are available, we have assumed BC, aSOA and POA to be about twice as hazardous as other components of $PM_{2.5}$. We believe this is a reasonable assumption to illustrate the potential influence on policy decisions. We also performed calculations that consider these species to be only 1.5 times more toxic compared to the other components. Further, Daellenbach et al. (2020) suggested that upon acute exposure, aSOA is substantially more harmful than other SOA and POA. To model the implications of these findings, we formulated the 3BS scenario (exposure to BC and aSOA being three times more hazardous compared to other components of $PM_{2.5}$ including POA). To simulate similar health responses of long-term exposure to these species, we formulated the 2BS and 1p5BS scenarios (long-term exposure to BC and aSOA were considered 2 and 1.5 times more toxic compared to other components of $PM_{2.5}$). See Fig. S13 for sector contributions obtained with these scenarios.

Our finding that exposure to $PM_{2.5}$ from domestic burning should be regarded as a leading health risk factor, and that the significance of exposure to natural and agricultural $PM_{2.5}$ decreases accordingly, does not change under the different assumptions. We note that for countries where the ratio of BC, POA and aSOA to other components is very low (e.g. dust dominated countries in North Africa and Middle-East), our results must be interpreted with caution. The excess mortality we report in this study depends on the shape of the exposure–response function used and the diseases considered. We note that heavy metals e.g. cadmium and lead emitted from industries and power plants are also detrimental

to human health, but toxicity assumptions that include such compounds are beyond the scope of the current work. Mineral dust (considered in NAT) may also be a potential but likely minor source (compared to power generation and industry) of metals (Jin et al., 2019; Schaap et al., 2018). The results from our sensitivity analyses illustrate the implications of considering anthropogenic organic aerosols to be more hazardous on human health compared to other fine particulates, rather than providing evidence for such differences for which further studies on chronic impact of BC, aSOA and POA are suggested.

We estimated the contributions of BC, aSOA and POA by removing their mass and then calculating the attributable deaths as those from the total $PM_{2.5}$ mass minus the deaths from the removal of individual species. This approach has been used in previous studies (Chowdhury et al., 2021; Lelieveld, 2017; Lelieveld et al., 2015) for source attribution. As an alternative to the MR-BRT functions, which account for selected disease categories, in previous work we used the Global Exposure Mortality Model (GEMM) to calculate excess mortality, which applies hazard ratio functions that encompass all NCDs together with lower respiratory tract infections (Chowdhury et al., 2020; Lelieveld et al., 2019; Lelieveld et al., 2020). The number of excess deaths attributed to $PM_{2.5}$ were about two times higher compared to our current estimates using the MR-BRT. Here we follow the recommendation by Burnett and Cohen (2020) to primarily apply the integrated exposure–response functions for specific disease categories, and use the GEMM for sensitivity calculations. The results are presented in the SI Text. By employing the outcomes of the GEMM calculations in those of the sectoral contributions, we find that the sectoral attribution results and policy implications are in good agreement (Fig. 8). It follows that our conclusions about the relative health impacts of $PM_{2.5}$ and its components are not sensitive to the use of different exposure–response relationships (see also Methods, SI Text, Figs. 8, S14).

4. Conclusions

Using the recent MR-BRT exposure–response functions of the GBD (Murray et al., 2020), we estimated 4.23(3.0–6.14) million excess deaths from the exposure to ambient $PM_{2.5}$ globally for 2015 of which 92%, 5%, and 3% occur among adults, neonates and children, respectively. Our estimates agree with the recent GBD study (Murray et al., 2020), but are considerably lower than our previous studies (Chowdhury et al., 2020; Lelieveld et al., 2019; Lelieveld et al., 2020) using the Global Exposure Mortality Model (GEMM) (Burnett et al., 2018) that considers excess mortality resulting from all NCDs (see SI Text, Fig. S14). We reiterate that the MR-BRT functions of the GBD (Murray et al., 2020), address selected disease categories (Figs. 1, S6; SI Data8), whereas the GEMM addresses air pollution impacts by all NCDs plus lower respiratory tract infections. In Europe, for example, the other NCDs (accounted for by GEMM but not by MR-BRT) may contribute about one third to the mortality burden from air pollution (Lelieveld et al., 2019). Previously, we also added excess mortality from the long-term exposure to tropospheric ozone, not included here. We estimated a global mortality burden of about 1.3 million per year from ozone in 2015 (Chowdhury et al., 2020), but this does not affect the current discussion on $PM_{2.5}$ toxicity.

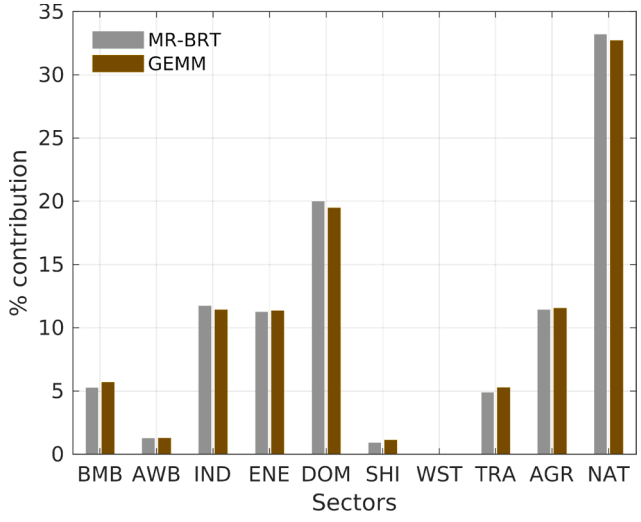

Fig. 8. Percentage contributions of source sectors to excess mortality burden from $PM_{2.5}$ calculated using the MR-BRT and GEMM exposure response functions. The sectors at the x-axis are BMB- Biomass burning; AWB- Agricultural waste burning; IND- Industries; ENE- Power generation; DOM- Domestic solid fuel burning and other commercial activities; SHI- Ships; WST- Waste Incineration; TRA- Transportation; AGR- Agricultural soils; NAT- Natural and biogenic emissions.

Another recent study (Vohra et al., 2021), using a concentration–response function built from a meta analyses of 53 studies, which provided 135 estimates of the quantitative association between the risk of mortality and exposure to $PM_{2.5}$ (Vodonos et al., 2018), estimated 8.7 (95% CI: −1.8 to 14.0) million premature deaths annually from the exposure to ambient $PM_{2.5}$ from fossil fuel use globally, which included recent declines in air pollution in China. Here, we chose to be consistent with the GBD, but emphasize that the continued incorporation of disease categories, following growing evidence for their relationship with air pollution, implies that estimates of the excess mortality rate will increase accordingly, as it has done since the previous GBD study (Cohen et al., 2017; Stanaway et al., 2018). It can be expected that future updates of the exposure–response functions of the GBD and the GEMM will converge with respect to excess mortality estimates (Burnett and Cohen, 2020). We find that the different functions (MR-BRT and GEMM) yield the same results in terms of the contributions of source sectors to $PM_{2.5}$ mortality, and the role of BC, POA and aSOA (SI Text, Fig. 8). This indicates that the policy implications of our sensitivity calculations are robust.

We estimated the source sectors and mortality attributable to $PM_{2.5}$, BC, POA and aSOA. Our results of source attribution of ambient PM2.5 exposure match with a recent global study (McDuffie et al., 2021), while here we additionally estimate the excess mortality from BC, POA and aSOA, the source sectors, and also present sensitivity studies where these components were assumed to be more toxic compared to other components in $PM_{2.5}$. We identified DOM as the globally largest source category of BC, POA and aSOA and the leading anthropogenic sector contributing to excess mortality from exposure to $PM_{2.5}$. The major sources of BC in our calculations are DOM and TRA, which concurs with previous findings (Anenberg et al., 2011; Janssen et al., 2011; Li et al., 2016). The current study provides the first global source attribution of POA and aSOA. Our estimates of excess deaths from aSOA are ~ 20% lower compared to a recent study (Nault et al., 2020) that used a different chemical transport model, carbonaceous aerosol routines and exposure–response functions.

As a sensitivity analysis, we consider BC, POA and aSOA to be more harmful compared to the other components of $PM_{2.5}$ based on toxicological and epidemiological studies of short-term health outcomes (Bates et al., 2019; Daellenbach et al., 2020; Janssen et al., 2011; Park et al., 2018; Verma et al., 2015; Yang et al., 2018), pending extensive epidemiological cohort studies of long-term impacts. The small number of cohort studies of chronic impact of BC have shown variable findings but a recent long term follow up has shown increased risk from BC exposure (Yang et al., 2021). Under the 2BSP assumption, anthropogenic carbonaceous particles would account for 364 (257–533) out of 1,000 excess deaths from the global exposure to $PM_{2.5}$ (i.e. 36 (26–53)%). If DOM emissions would indeed be more toxic than PM2.5 emissions from other sources, as we indicate in our sensitivity analyses, the health effects of air pollution originating in solid fuel using households may be even greater and more strongly coordinated efforts may be required to avert this burden. One way is to promote campaigns encouraging the use of

cleaner cookstoves in the low-and-middle-income countries; however, solid biomass is a difficult fuel to burn efficiently. Reducing or replacing household solid fuel use with cleaner fuels, like liquified petroleum gas (LPG), ethanol, or electricity, should be a high priority. Some countries like Brazil and India have substantially expanded the use of LPG for cooking in their households. Though the net climate impacts of LPG use, compared to improved biomass stoves, are uncertain and minimal, the social and health benefits are large.

Phasing out DOM emissions in South Asia, where interventions aim at replacing the domestic use of solid fuels for cooking and lighting, with LPG (Chowdhury et al., 2019) may help prevent up to 24% of global excess mortality from exposure to ambient $PM_{2.5}$. To some degree this will shift the mortality burden between bio- and fossil fuels (Table 1). The use of alternative, clean renewable energy technologies based on solar and wind power, for example, would remove this health burden altogether. For Western Europe we find AGR to be the largest contributor to ambient $PM_{2.5}$ (20%), however, more stringent policies will be effective by mitigating the use of solid fuels for heating, which will avert 27% of excess mortality if the 2BSP assumption would be valid. Despite the small share in total energy consumption, residential biomass and coal burning may cause ~ 40% of total primary emissions of $PM_{2.5}$ in Europe, which is about two times higher than emissions from TRA and AGR, which are often the targeted sector in policy making (Clean Heat, 2016; Crippa et al., 2018; Giannakis et al., 2019; Hoesly et al., 2018). Nevertheless, globally, contributions from fossil fuel use also increase by 15% under the 2BSP assumption, and by 18% and 16% in North America and Western Europe, respectively, which corroborates the importance of curbing fossil fuel use in power generation, industries, road transportation and shipping.

Thus, the relative contributions of sectors that use fossil fuels as well as DOM are expected to increase significantly under the 2BSP assumption (as well as other enhanced toxicity assumptions). Taken together the sectors that use fossil fuels and DOM then contribute about equally to excess mortality. These findings, which do not depend on the exposure–response functions applied, demonstrate the central importance of accounting for the relative harmfulness of $PM_{2.5}$ components in formulating air quality policies, which need to be tailored for different countries and regions. It may be expected that the actual relative health effects are within the range of these five assumptions, and further studies on the health effects of long term exposure to relatively toxic species and those with high oxidative potential are recommended. Even though current knowledge does not allow a more reliable quantitative assessment of the relative health outcomes, our results, based on the recent literature, suggest that anthropogenic, carbonaceous particles should receive greater weight in air pollution mitigation scenarios and policy measures. Our results generated at country-level (SI DataS3), or at higher resolution (on request), are available to study mitigation pathways to reduce the health burden of ambient $PM_{2.5}$, and capitalise on the potential health co-benefits from reducing greenhouse gas and air pollution emissions.

CRediT authorship contribution statement

Sourangsu Chowdhury: Conceptualization, Investigation, Methodology, Software, Formal analysis, Writing – original draft, Writing – review & editing. **Andrea Pozzer:** Conceptualization, Methodology, Software, Writing – review & editing. **Andy Haines:** Methodology, Writing – review & editing. **Klaus Klingmüller:** Software, Writing – review & editing. **Thomas Münzel:** Writing – review & editing. **Pauli Paasonen:** Data curation. **Arushi Sharma:** Data curation. **Chandra Venkataraman:** Data curation, Writing – review & editing. **Jos Lelieveld:** Conceptualization, Methodology, Software, Writing – original draft, Writing – review & editing, Supervision, Funding acquisition.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: The authors declare no competing interests.

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Appendix A. Supplementary data

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