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The Contribution of Carbonaceous Aerosols to Air Pollution and Excess Mortality in Europe †

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Abstract: Air pollution is an important environmental risk factor associated with increased morbidity and excess mortality. Fine particulate matter (PM2.5) is a complex mixture of both organic and inorganic compounds, depending on emissions sources and atmospheric chemistry. According to toxicological studies, there is strong evidence that anthropogenic carbonaceous aerosols, especially those emitted from combustion sources, are more hazardous to human health than other types of fine particles. In this study, we use WRF-Chem to simulate PM2.5 and the carbonaceous sub-components (black carbon and organics from anthropogenic sources) over Europe. The excess mortality attributed to long-term exposure to these particles is quantified using the MR-BRT (meta-Regression–Bayesian, regularized, trimmed) and the Global Exposure Mortality Model (GEMM) exposure–response functions to assess the public health outcomes. Differential toxicity of carbonaceous aerosols is assumed to account for their potentially more pronounced effect on excess mortality.

Keywords: air pollution; PM2.5; black carbon; organic aerosols; health; mortality

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1. Introduction

Fine particulate matter (PM2.5), originating from various anthropogenic and, to a lesser degree, biogenic sources, is an important contributor to air pollution. PM2.5 consists of both organic (e.g., from aromatics) and inorganic (e.g., sulfates, nitrates) compounds, elemental or black carbon (BC), as well as transition metals (e.g., copper, chromium, iron, and zinc), often with a pronounced seasonal and spatial variation [1]. Exposure to PM2.5 has been associated with various cardiovascular, respiratory, and other diseases, being a significant environmental health risk factor [2,3]; however, with significant uncertainty in attributable mortality estimates. There is strong evidence from animal and in vitro toxicological studies that carbon-containing aerosols, primarily anthropogenic BC, primary and secondary organic aerosols from combustion sources, have a relatively strong ability to induce oxidative stress, which is a highly plausible disease-triggering mechanism [4,5]. Due to large differences in study outcomes, the evidence is inconclusive, and thus, health impact studies still rely on total PM2.5 mass concentration without discriminating for specific sub-components that might be of more or less toxicological importance. Furthermore, the different exposure risk functions that have been developed in recent years have resulted in varying excess mortality estimates adding to uncertainty among health impact studies [6]. Here, we focus on the wider European domain and use two exposure–response functions to calculate the excess mortality due to PM2.5. We estimate the contribution of anthropogenic carbonaceous aerosols to the estimated excess mortality and assume that these are up to two times more harmful than inorganic aerosols such as sulfates and nitrates.

2. Materials and Methods

WRF-CHEM-v.3.9.1 [7] is used to simulate annual concentrations of PM2.5 and carbonaceous constituents (black carbon, primary organic carbon, anthropogenic secondary organic carbon) over Europe at a horizontal resolution of 20 × 20 km [8]. Cause and age-specific Relative Risks (RRs) of death from exposure to ambient PM2.5 air pollution in adults (over 25 years old) are calculated using the meta-Regression–Bayesian, regularized, trimmed (MR-BRT) risk function [9]. We calculate RRs for ischemic heart disease (IHD), stroke, chronic obstructive pulmonary disease (COPD), lower respiratory infections (LRI), lung cancer (LC), and type II diabetes (T2D) among adults. Furthermore, RRs for non-communicable diseases and lower respiratory infections among adults are also calculated with the Global Exposure Mortality Model (GEMM) [10], which may be considered to provide upper limits [11]. The RRs are used to calculate the attributable fraction (AF), which expresses the fraction of total mortality that is attributed to ambient PM2.5 pollution and is expressed as (RR-1/RR). AF at each grid cell (calculated at 0.042° × 0.042° resolution) is combined with baseline mortality rates (BMR) and population (POP) per disease j and age k to calculate excess mortality attributable to PM2.5 long-term exposure (Equation (1)). The contribution of carbonaceous aerosols is estimated by multiplying the total excess mortality with the concentration ratio, as indicated in Equation (2). To account for the differential toxicity of carbonaceous aerosol components, we assume that the toxicity increases with inhaled dose (exposure). By considering carbonaceous aerosols twice more toxic than the other PM2.5 constituents (i.e., inorganic and natural), we enhance their relative contribution by a factor of two at the same PM2.5 concentration (Equation (3)) [12].

$$\mathrm{M_{PM2.5}(x,y)} = \sum_{\mathrm{j,k}} (\mathrm{BMR(x,y)} \times \mathrm{POP(x,y)} \times \mathrm{AFj,k}) \tag{1}$$

$$\mathrm{M_{CA}(x,y)} = \mathrm{M_{PM2.5}(x,y)} \times \left(\frac{\mathrm{CA(x,y)}}{\mathrm{PM2.5(x,y)}} \right) \tag{2}$$

$$\mathrm{M_{CA'}(x,y)} = \mathrm{M_{PM2.5}(x,y)} \times \left(\frac{2 \times \mathrm{CA(x,y)}}{\mathrm{PM2.5(x,y)}} \right) \tag{3}$$

where $\mathrm{M_{PM2.5}}$ and $\mathrm{M_{CA}}$ are the excess mortality due to total PM2.5 and carbonaceous aerosols, respectively, for each disease and age group, respectively.

3. Results

3.1. PM2.5 Exposure

Based on our model results, the populations of Serbia and the Czech Republic are exposed to the relatively highest PM2.5 concentrations (population-weighted, annual average 20.8 $\mu g/m^3$ and 19.3 $\mu g/m^3$) compared to other European countries, whereas that of Iceland to the lowest (3.2 $\mu g/m^3$) (Figure 1). The peoples of Hungary, Poland, and the Czech Republic are exposed to relatively high and, in particular, carbon-rich ambient PM2.5 concentrations (~20% of their mass) compared to other European countries such as Norway and Iceland (~3–6%). The citizens of the Czech Republic are found to have the highest annual exposure to carbonaceous aerosols (4.2 $\mu g/m^3$). Northern EU countries such as Sweden, Ireland, Norway, and Iceland have the lowest population-weighted, annual mean concentrations of carbonaceous aerosols (1 $\mu g/m^3$, 0.8 $\mu g/m^3$, 0.6 $\mu g/m^3$, 0.1 $\mu g/m^3$, respectively). This is reflected in the estimated excess mortality discussed in Section 3.2.

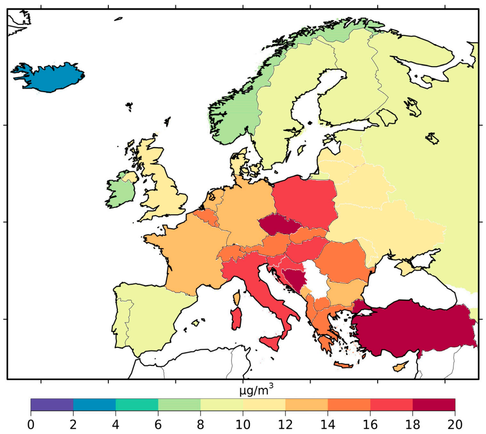
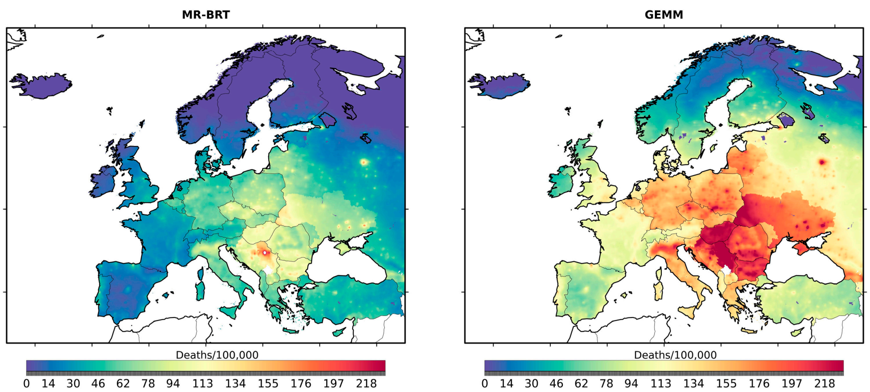


Figure 1. Population-weighted annual mean PM2.5 concentration for each country.

3.2. Excess Mortality

PM2.5 exposure over the wider European region is found to be responsible for about 316 (239–347) thousand excess deaths per year (with MR-BRT), of which up to 44% is due to the carbonaceous fraction, depending on the region and toxicity assumption. The highest excess mortality from PM2.5 occurs in Serbia (29 excess deaths per 100,000 persons) and the lowest in Norway (~2 excess deaths per 100,000 persons) (Table S1). The highest relative contribution of carbonaceous aerosols to excess mortality comes about in the Czech Republic, Poland, and Hungary (~21%) and the lowest in Norway (7%).

Calculations with the GEMM risk model result in 489 (355–554) thousand excess deaths per year for the five causes of death that are also included in MR-BRT (except Type II diabetes) (Table 1). When all non-communicable diseases are accounted for, GEMM results in 787 (647–927) thousand excess deaths per year, which is more than twice the estimates with MR-BRT, likely providing an upper limit [11] (Figure 2). The relative contributions of carbonaceous aerosols to excess mortality do not change significantly when we use different risk models because we attribute their mortality contribution to their concentration ratio. Thus, even though the absolute estimates between the MR-BRT and GEMM calculations differ considerably, the attribution to the sources is the same. On average, the contribution of anthropogenic carbonaceous aerosols is ~14%, assuming equal toxicity, and ~27%, assuming toxicity that is twice as high. At the country level and under the differential toxicity assumption, the relative contribution of the carbonaceous fraction to excess mortality can reach ~44%, for example, in Poland, Hungary, and the Czech Republic (Tables 1 and S2).



(**a**) (**b**)

Figure 2. (**a**) Total excess mortality due to PM2.5 with MR-BRT exposure risk function. (**b**) Total excess mortality due to PM2.5 with GEMM exposure risk function. The estimates are normalized to the adult population in each grid box.

Table 1. Adult excess mortality (mean, 95% confidence interval) expressed as the number of deaths per year with MR-BRT and GEMM, attributable to PM2.5 and carbonaceous aerosols (CA) long-term exposure in each country assuming equal (1) and differential toxicity (2).

	MR-BRT			GEMM		
Country	**PM25**	**CA**		**PM2.5**	**CA**	
		1	**2**		**1**	**2**
Austria	3200 (2300–3700)	534 (386–623)	1068 (772–1245)	8400 (6900–9900)	1300 (1100–1600)	2700 (2200–3100)
Belgium	4400 (3100–5200)	700 (752–540)	1505 (1079–1791)	11,600 (9500–13,700)	2000 (1600–2300)	3900 (3200–4600)
Bosnia and Herzegovina	2900 (2000–3500)	553 (376–666)	1106 (751–1333)	4700 (3900–5500)	900 (700–1000)	1800 (1400–2100)
Bulgaria	6100 (4500–6900)	988 (727–1130)	1977 (1454–2259)	11,700 (9600–13,800)	1800 (1500–2200)	3700 (3000–4300)
Belarus	4600 (3600–4800)	730 (559–754)	1461 (1118–1509)	10,300 (8500–12,200)	1600 (1300–1900)	3100 (2600–3700)
Croatia	3200 (2300–3700)	652 (478–766)	1304 (955–1532)	6400 (5200–7500)	1300 (1100–1500)	2600 (2100–3000)
Cyprus	400 (300–500)	40 (26–47)	80 (52–95)	700 (600–900)	100 (100–100)	100 (100–200)
Czech Republic	7500 (5400–8800)	1665 (1207–1956)	3330 (2414–3911)	14,200 (11,600–16,600)	3100 (2500–3600)	6100 (5100–7200)
Denmark	1400 (1100–1500)	202 (153–215)	403 (306–430)	4400 (3600–5300)	600 (500–700)	1200 (1000–1500)
France	14,500 (10,500–16,100)	2447 (1772)	4894 (3544–5507)	49,400 (40,600-)	8100 (6700–9500)	16,200 (13,300–19,100)
Germany	37,600 (27,300–43,400)	5936 (4298)	11,871 (8596–13,716)	99,900 (82,500–117,200)	15,300 (12,700–18,000)	30,700 (25,300–36,000)
Greece	5000 (3700–5800)	680 (497–788)	1361 (995–1575)	12,100 (9900–14,300)	1600 (1300–1900)	3200 (2600–3800)
Hungary	7800 (5700–9200)	1712 (1252–2014)	3425 (2503–4027)	16,200 (13,300–18,900)	3500 (2900–4100)	7000 (5700–8100)
Italy	29,300 (21,100–34,100)	5161 (3725–6033)	10,323 (7449–12,065)	71,800 (59,400–84,100)	12,300 (10,100–14,400)	24,500 (20,300–28,700)
Lithuania	1700 (500–1900)	306 (228–340)	612 (456–680)	3900 (3200–4600)	700 (600–800)	1300 (1100–1600)
Norway	400 (300–200)	41 (27–41)	82 (53–81)	2100 (1700–2500)	210 (172–247)	400 (300–500)
Portugal	2900 (1700–2900)	395 (301–398)	791 (603–795)	8300 (6800–9800)	1107 (904–1314)	2215 (1808–2628)
Serbia	8200 (3800–8700)	1524 (1116–1791)	3047 (2232–3581)	13,900 (11,500–16,300)	2520 (2079–2954)	5000 (4200–5900)
Slovakia	2900 (900–3500)	486 (342–593)	971 (683–1186)	6200 (5100–7300)	1000 (800–1200)	2000 (1700–2400)
Slovenia	800 (400–1000)	153 (108–191)	307 (216–383)	2200 (1800–2700)	400 (300–500)	800 (600–900)
Spain	9400 (5300–9200)	1508 (1163–1505)	3015 (2326–3011)	32,100 (26,400–37,800)	4900 (4100–5800)	9900 (8100–11,600)

Furthermore, we estimate (with MR-BRT) that in Europe, on average, the highest annual excess mortality is due to cardiovascular (CV) diseases (28 and 15 deaths per 100,000 for IHD and Stroke, respectively), followed by type II diabetes (7 per 100,000), lung cancer (5 per 100,000) and respiratory diseases (4 and 2 per 100,000 for COPD and LRI, respectively) (Table S1). The absolute mortality rates per disease category and risk model are summarized in Table 2. At the country level, we estimate that Serbia suffers the highest annual excess mortality from CV diseases and lung cancer (66 and 14 deaths per 100,000, respectively). Excess mortality from COPD is highest in Hungary (9 per 100,000) and from LRI in Belgium (5 per 100,000). Bosnia and Herzegovina has the highest excess mortality due to type II diabetes, followed by Cyprus (24 and 22 deaths per 100,000, respectively) (Table S1).

Table 2. Adult annual excess mortality (mean, 95% confidence interval) per disease category in the wider European domain attributable to PM2.5 and carbonaceous aerosols (CA) (equal toxicity assumption). The excess deaths are expressed in thousands/year.

Cause of Death	MR-BRT	GEMM (5COD)	MR-BRT	GEMM (5COD)
	PM2.5	PM2.5	CA	CA
IHD	145 (107–162)	299 (260–343)	26	51
Stroke	72 (59–76)	61 (28–97)	13	11
COPD	24 (19–27)	37 (18–22)	3	6
LC	29 (23–32)	44 (26–64)	4	8
LRI	12 (8–16)	48 (23–28)	2	8
Type II diabetes	34 (24–34)	-	5	-
Total	316 (239–347)	489 (355–554)	53 (16.7%)	84 (17.2%)

4. Discussion

The relationship between anthropogenic carbonaceous aerosols and morbidity is not well known due to a lack of (access to) public health data; thus, their contribution can be estimated only based on the available PM2.5 exposure risk functions. Depending on the exposure risk function and methodology, we estimate that excess mortality attributable to ambient PM2.5 exposure in the wider European domain can range between about 316 and 787 thousand excess deaths per year. The contribution of anthropogenic carbonaceous aerosols to the estimated mortality over the region is, on average, between ~14% and ~27% depending on the toxicity assumption. At the country level, their contribution can range between 4 and 27% of the total mortality, assuming equal toxicity, and 14–44%, assuming twice as high toxicity. These estimates are also subject to several other uncertainties that relate to model performance and emissions representation, as discussed elsewhere [8].

5. Conclusions

Excess mortality attributable to total PM2.5 and to the anthropogenic carbonaceous aerosol fraction can differ significantly between the two used risk models, countries, and toxicity assumptions. The contribution of carbonaceous aerosols to air pollution and excess mortality in Europe is significant and can contribute up to ~44% of the total excess mortality in some countries. Although these estimates are associated with several uncertainties, knowing that carbon-containing aerosols, especially from combustion sources, can be highly hazardous to human health, this study highlights the need for efficient air pollution mitigation strategies in Europe aimed at minimizing their emissions and personal exposure. Finally, it points out the necessity for consistent future epidemiological and toxicological studies that can provide quantifiable data on the relationship between personal exposure to carbonaceous aerosols and health effects.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/environsciproc2023026074/s1. Table S1: Annual adult excess mortality (with MR-BRT) per country and disease normalized to the population. The highest mortality per disease and country is highlighted with bold. Table S2: Excess adult mortality (with MR-BRT and GEMM risk models) attributable to PM2.5 and anthropogenic carbonaceous aerosols (CA) long-term exposure in each country assuming equal (1) and differential toxicity (2). The two last columns in each group indicate the relative contribution of CA (in percentages) to total excess mortality (from PM2.5).

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References

1. Borbon, A.; Gilman, J.B.; Kuster, W.C.; Grand, N.; Chevaillier, S.; Colomb, A.; Dolgorouky, C.; Gros, V.; Lopez, M.; Sarda-Esteve, R.; et al. Emission ratios of anthropogenic volatile organic compounds in northern mid-latitude megacities: Observations versus emission inventories in Los Angeles and Paris. *J. Geophys. Res. Atmos.* **2013**, *118*, 2041–2057. [CrossRef]
2. Pope, C.A., III; Dockery, D.W. Health Effects of Fine Particulate Air Pollution: Lines that Connect. *J. Air Waste Manag. Assoc.* **2006**, *56*, 709–742. [CrossRef] [PubMed]
3. World Health Organization. Review of evidence on health aspects of air pollution-REVIHAAP Project Technical Report. 2013. Available online: http://www.euro.who.int/pubrequest (accessed on 30 June 2023).
4. Bates, J.T.; Fang, T.; Verma, V.; Zeng, L.; Weber, R.J.; Tolbert, P.E.; Abrams, J.Y.; Sarnat, S.E.; Klein, M.; Mulholland, J.A.; et al. Review of acellular assays of ambient particulate matter oxidative potential: Methods and relationships with com- position, sources, and health effects. *Environ. Sci. Technol.* **2019**, *53*, 4003–4019. [CrossRef] [PubMed]
5. Daellenbach, K.R.; Uzu, G.; Jiang, J.; Cassagnes, L.-E.; Leni, Z.; Vlachou, A.; Stefenelli, G.; Canonaco, F.; Weber, S.; Segers, A.; et al. Sources of particulate-matter air pollution and its oxidative potential in Europe. *Nature* **2020**, *587*, 414–419. [CrossRef] [PubMed]
6. Pozzer, A.; Anenberg, S.C.; Dey, S.; Haines, A.; Lelieveld, J.; Chowdhury, S. Mortality Attributable to Ambient Air Pollution: A Review of Global Estimates. *Geohealth* **2023**, *7*, e2022GH000711. [CrossRef] [PubMed]
7. Grell, G.A.; Peckham, S.E.; Schmitz, R.; McKeen, S.A.; Frost, G.; Skamarock, W.C.; Eder, B. Fully coupled "online" chemistry within the WRF model. *Atmos. Environ.* **2005**, *39*, 6957–6975. [CrossRef]
8. Paisi, N.; Kushta, J.; Georgiou, G.; Zittis, G.; Pozzer, A.; Denier Van Der Gon, H.; Kuenen, J.; Christoudias, T.; Lelieveld, J. Modeling of carbonaceous aerosols for air pollution health impact studies in Europe. *Air Qual. Atmos. Health*, 2023; *Submitted*.
9. Global Burden of Disease Collaborative Network. *Global Burden of Disease Study 2019 (GBD 2019) Particulate Matter Risk Curves*; Institute for Health Metrics and Evaluation (IHME): Seattle, WA, USA, 2021.
10. Burnett, R.; Chen, H.; Szyszkowicz, M.; Fann, N.; Hubbell, B.; Pope, C.A., 3rd; Apte, J.S.; Brauer, M.; Cohen, A.; Weichenthal, S.; et al. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. *Proc. Natl. Acad. Sci. USA* **2018**, *115*, 9592–9597. [CrossRef] [PubMed]
11. Burnett, R.; Cohen, A. Relative Risk Functions for Estimating Excess Mortality Attributable to Outdoor $PM_{2.5}$ Air Pollution: Evolution and State-of-the-Art. *Atmosphere* **2020**, *11*, 589. [CrossRef]
12. Lelieveld, J.; Evans, J.S.; Fnais, M.; Giannadaki, D.; Pozzer, A. The Contribution of Outdoor Air Pollution Sources to Premature Mortality on a Global Scale. *Nature* **2015**, *525*, 367–371. [CrossRef] [PubMed]