

Journal Pre-proof

Global mortality from outdoor fine particle pollution generated by fossil fuel combustion: Results from GEOS-Chem

Karn Vohra, Alina Vodonos, Joel Schwartz, Eloise A. Marais, Melissa P. Sulprizio, Loretta J. Mickley

PII: S0013-9351(21)00048-7

DOI: <https://doi.org/10.1016/j.envres.2021.110754>

Reference: YENRS 110754

To appear in: *Environmental Research*

Received Date: 29 July 2019

Revised Date: 12 January 2021

Accepted Date: 14 January 2021

Please cite this article as: Vohra, K., Vodonos, A., Schwartz, J., Marais, E.A., Sulprizio, M.P., Mickley, L.J., Global mortality from outdoor fine particle pollution generated by fossil fuel combustion: Results from GEOS-Chem, *Environmental Research*, <https://doi.org/10.1016/j.envres.2021.110754>.

This is a PDF file of an article that has undergone enhancements after acceptance, such as the addition of a cover page and metadata, and formatting for readability, but it is not yet the definitive version of record. This version will undergo additional copyediting, typesetting and review before it is published in its final form, but we are providing this version to give early visibility of the article. Please note that, during the production process, errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

© 2021 Published by Elsevier Inc.



K. Vohra and A. Vodonos carried out the health impact calculations guided by J. Schwartz. E. A. Marais and M. P. Sulprizio performed GEOS-Chem simulations. L. J. Mickley oversaw the project. All authors contributed to writing the manuscript.

Journal Pre-proof

1 **Global mortality from outdoor fine particle pollution generated by**
2 **fossil fuel combustion: Results from GEOS-Chem**

3
4 **Karn Vohra^{1*}, Alina Vodonos², Joel Schwartz², Eloise A. Marais^{3,a}, Melissa P. Sulprizio⁴,**
5 **Loretta J. Mickley⁴**

6 ¹ School of Geography, Earth and Environmental Sciences, University of Birmingham,
7 Birmingham, UK

8 ² Harvard T.H. Chan School of Public Health, Department of Environmental Health, Harvard
9 University, Boston, MA, USA

10 ³ Department of Physics and Astronomy, University of Leicester, Leicester, UK

11 ⁴ John A. Paulson School of Engineering and Applied Sciences, Harvard University, Cambridge,
12 MA, USA

13 ^a Now at: Department of Geography, University College London, London, UK

14 * Corresponding author: Karn Vohra, Phone: +44 7716 496 867,

15 Email: kxv745@student.bham.ac.uk

16
17 **Keywords;** particulate matter, fossil fuel, mortality, health impact assessment

18
19 **Abstract**

20 The burning of fossil fuels – especially coal, petrol, and diesel – is a major source of airborne
21 fine particulate matter (PM_{2.5}), and a key contributor to the global burden of mortality and
22 disease. Previous risk assessments have examined the health response to total PM_{2.5}, not just
23 PM_{2.5} from fossil fuel combustion, and have used a concentration-response function with limited

24 support from the literature and data at both high and low concentrations. This assessment
25 examines mortality associated with $PM_{2.5}$ from only fossil fuel combustion, making use of a
26 recent meta-analysis of newer studies with a wider range of exposure. We also estimated
27 mortality due to lower respiratory infections (LRI) among children under the age of five in the
28 Americas and Europe, regions for which we have reliable data on the relative risk of this health
29 outcome from $PM_{2.5}$ exposure. We used the chemical transport model GEOS-Chem to estimate
30 global exposure levels to fossil-fuel related $PM_{2.5}$ in 2012. Relative risks of mortality were
31 modeled using functions that link long-term exposure to $PM_{2.5}$ and mortality, incorporating
32 nonlinearity in the concentration response. We estimate a global total of 10.2 (95% CI: -47.1 to
33 17.0) million premature deaths annually attributable to the fossil-fuel component of $PM_{2.5}$. The
34 greatest mortality impact is estimated over regions with substantial fossil fuel related $PM_{2.5}$,
35 notably China (3.9 million), India (2.5 million) and parts of eastern US, Europe and Southeast
36 Asia. The estimate for China predates substantial decline in fossil fuel emissions and decreases
37 to 2.4 million premature deaths due to 43.7% reduction in fossil fuel $PM_{2.5}$ from 2012 to 2018
38 bringing the global total to 8.7 (95% CI: -1.8 to 14.0) million premature deaths. We also
39 estimated excess annual deaths due to LRI in children (0-4 years old) of 876 in North America,
40 747 in South America, and 605 in Europe. This study demonstrates that the fossil fuel
41 component of $PM_{2.5}$ contributes a large mortality burden. The steeper concentration-response
42 function slope at lower concentrations leads to larger estimates than previously found in Europe
43 and North America, and the slower drop-off in slope at higher concentrations results in larger
44 estimates in Asia. Fossil fuel combustion can be more readily controlled than other sources and
45 precursors of $PM_{2.5}$ such as dust or wildfire smoke, so this is a clear message to policymakers
46 and stakeholders to further incentivize a shift to clean sources of energy.

47

48 **Introduction**

49 The burning of fossil fuels – especially coal, petrol, and diesel – is a major source of
50 airborne particulate matter (PM) and ground-level ozone, which have both been implicated as
51 key contributors to the global burden of mortality and disease (Apte et al., 2015; Dedoussi and
52 Barrett, 2014; Lim et al., 2013). A series of studies have reported an association between
53 exposure to air pollution and adverse health outcomes (Brook et al., 2010), even at low exposure
54 levels ($< 10 \mu\text{g m}^{-3}$, the current World Health Organization, WHO, guideline) (Di et al., 2017).
55 The Global Burden of Diseases, Injuries, and Risk Factors Study 2015 (GBD 2015) identified
56 ambient air pollution as a leading cause of the global disease burden, especially in low-income
57 and middle-income countries (Forouzanfar et al., 2016). Recent estimates of the global burden
58 of disease suggest that exposure to $\text{PM}_{2.5}$ (particulate matter with an aerodynamic diameter < 2.5
59 μm) causes 4.2 million deaths and 103.1 million disability-adjusted life-years (DALYs) in 2015,
60 representing 7.6% of total global deaths and 4.2% of global DALYs, with 59% of these in east
61 and south Asia (Cohen et al., 2017).

62 A series of newer studies conducted at lower concentrations and at higher concentrations
63 have reported higher slopes than incorporated into the GBD using the integrated exposure–
64 response (IER) curve (Burnett et al., 2014). These studies examined mortality due to exposure to
65 $\text{PM}_{2.5}$ at concentrations below $10 \mu\text{g m}^{-3}$ in North America (Di et al., 2017; Pinault et al., 2016)
66 and above $40 \mu\text{g m}^{-3}$ in Asia (Katanoda et al., 2011; Tseng et al., 2015; Ueda et al., 2012; Wong
67 et al., 2015; 2016; Yin et al., 2017). Here we have used a concentration-response curve from a
68 recently published meta-analysis of long-term $\text{PM}_{2.5}$ mortality association among adult
69 populations which incorporates those new findings at high and low $\text{PM}_{2.5}$ concentrations

70 (Vodonos et al., 2018). We also focus our study on the health impacts of fossil-fuel derived
71 $PM_{2.5}$. In contrast, GBD reports only the health impacts of total $PM_{2.5}$ and does not distinguish
72 mortality from fossil-fuel derived $PM_{2.5}$ and that from other kinds of $PM_{2.5}$, including dust,
73 wildfire smoke, and biogenically-sourced particles. We focus only on $PM_{2.5}$ since recent studies
74 have provided mixed results on the link between ozone and mortality (Atkinson et al., 2016) and
75 there does not exist a global coherent concentration-response function (CRF) for ozone.

76 The developing fetus and children younger than 5 years of age are more biologically and
77 neurologically susceptible to the many adverse effects of air pollutants from fossil-fuel
78 combustion than adults. This differential susceptibility to air pollution is due to their rapid
79 growth, developing brain, and immature respiratory, detoxification, immune, and
80 thermoregulatory systems (Bateson and Schwartz, 2008; Perera, 2018). Children also breathe
81 more air per kilogram of body weight than adults, and are therefore more exposed to pollutants
82 in air (WHO, 2006; Xu et al., 2012). The WHO estimated that in 2012, 169,000 global deaths
83 among children under the age of 5 were attributable to ambient air pollution (WHO, 2016).
84 Further estimation of the burden of mortality due to $PM_{2.5}$ (particularly from anthropogenic
85 sources) among the young population would highlight the need for intervention aimed at
86 reducing children's exposure.

87 Using the chemical transport model GEOS-Chem, we quantified the number of premature
88 deaths attributable to ambient air pollution from fossil fuel combustion. Improved knowledge of
89 this very immediate and direct consequence of fossil fuel use provides evidence of the benefits to
90 current efforts to cut greenhouse gas emissions and invest in alternative sources of energy. It also
91 helps quantify the magnitude of the health impacts of a category of $PM_{2.5}$ that can be more
92 readily controlled than other kinds of $PM_{2.5}$ such as dust or wildfire smoke.

93

94 **Materials and methods**95 *Calculation of surface PM_{2.5} concentrations*

96 Previous studies examining the global burden of disease from outdoor air pollution have
97 combined satellite and surface observations with models to obtain improved estimates of global
98 annual mean concentrations of PM_{2.5} (Shaddick et al., 2018). However, the goal of such studies
99 was to quantify the health response to PM_{2.5} from all sources, both natural and anthropogenic
100 (Brauer et al., 2016; Cohen et al., 2017). Here the focus of our study is on surface ambient PM_{2.5}
101 generated by fossil fuel combustion, and for that we rely solely on the chemical transport model
102 GEOS-Chem since current satellite and surface measurements cannot readily distinguish
103 between the sources of PM_{2.5}. Results from GEOS-Chem have been extensively validated against
104 surface, aircraft, and space-based observations around the world, including simulation of surface
105 pollution over the United States (Drury et al., 2010; Ford and Heald, 2013; Heald et al., 2012;
106 Leibensperger et al., 2012; Marais et al., 2016; Zhang et al., 2012), Asia (Kopplitz et al., 2016;
107 Lin et al., 2014), Europe (Protonotariou et al., 2013; Veeffkind et al., 2011), and Africa (Lacey et
108 al., 2017; Marais et al., 2014a; 2014b; 2016; 2019). The model has also been applied to previous
109 studies quantifying the global burden of disease from particulate matter from all sources (Brauer
110 et al., 2016; Cohen et al., 2017).

111 In this analysis we used GEOS-Chem with fossil fuel emissions from multiple sectors
112 (power generation, industry, ships, aircraft, ground transportation, backup generators, kerosene,
113 oil/gas extraction), detailed oxidant-aerosol chemistry, and reanalysis meteorology from the
114 NASA Global Modeling and Assimilation Office. Fossil fuel emissions are from regional

115 inventories where these are available for the US, Europe, Asia, and Africa, and from global
116 inventories everywhere else (such as Mexico, Australia, South America and Canada). More
117 details of the specific fossil fuel inventories used in GEOS-Chem are in Table S1. Global-scale
118 simulations in GEOS-Chem were carried out on a coarse spatial grid ($2^\circ \times 2.5^\circ$, about $200 \text{ km} \times$
119 250 km). Four regional simulations were also performed at fine spatial scale ($0.5^\circ \times 0.67^\circ$, about
120 $50 \text{ km} \times 60 \text{ km}$) for North America, Europe, Asia, and Africa using boundary conditions from
121 the global model. The regional simulations allow for a better match with the spatial distribution
122 of population, thus enhancing the accuracy of the estimates of health impacts. All simulations
123 were set up to replicate 2012 pollution conditions. As described in the Supplemental Material,
124 we find that globally, GEOS-Chem captures observed annual mean $\text{PM}_{2.5}$ concentrations with a
125 spatial correlation of 0.70 and mean absolute error of $3.4 \mu\text{g m}^{-3}$, values which compare well
126 with those from other models (Shindell et al., 2018; Xing et al., 2015). We performed two sets of
127 simulations: one set with fossil fuel emissions turned on and the other with such emissions
128 turned off. We then assumed that the difference between the two sets of simulations represents
129 the contribution of fossil fuel combustion to surface $\text{PM}_{2.5}$. More information on our choice of
130 GEOS-Chem, the model setup, details of relevant anthropogenic emissions, and model validation
131 is described in the Supplemental material.

132 *Population and Health data*

133 We used population data from the Center for International Earth Science Information
134 Network (CIESIN) (CIESIN, 2018). The Gridded Population of the World, Version 4 Revision
135 11 (GPWv4.11) is gridded with an output resolution of 30 arc-seconds (approximately 1 km at
136 the equator). Since the population data are provided only at five-year intervals, we applied 2015

137 population statistics to the results of our 2012 GEOS-Chem simulation. CIESIN population data
138 was then aggregated to the spatial scale of the model for the exposure estimates. Country/region
139 level data on baseline mortality rates were from GBD data for 2015 (based on the 2017 iteration)
140 (IHME, 2017). USA state-specific mortality rates were obtained from the CDC Wide-ranging
141 Online Data for Epidemiologic Research (WONDER) compressed mortality files (WONDER).
142 Canada death estimates by province were obtained from Statistics Canada, CANSIM (Canada,
143 2018).

144 *PM_{2.5} mortality concentration –response model*

145 The risk of air pollution to health in a population is usually estimated by applying a
146 concentration–response function (CRF), which is typically based on Relative Risk (RR)
147 estimates derived from epidemiological studies. CRFs are necessary elements for the
148 quantification of health impacts due to air pollution and require regular evaluation and update to
149 incorporate new developments in the literature.

150 Global assessments of air pollution risk often use the Integrated Exposure-
151 Response model (IER) (Burnett et al., 2014), which combined information on PM_{2.5}–mortality
152 associations from non-outdoor PM_{2.5} sources, including secondhand smoke, household air
153 pollution from use of solid fuels, and active smoking. The IER used data from active smoking
154 and passive smoking to address the limited number of outdoor PM_{2.5} epidemiologic studies at
155 PM_{2.5} > 40 µg m⁻³ available at the time. The IER formed the basis of the estimates of disease
156 burden attributable to PM_{2.5} (e.g., 4 million deaths in 2015 in GBD 2015). This function was then
157 updated in 2018 using the Global Exposure Mortality Model (GEMM). In GEMM, data from 41
158 epidemiological cohort studies were applied (Burnett et al., 2018). Independently conducted
159 analyses were conducted on 15 of these cohorts to characterize the shapes of PM_{2.5}–mortality

160 associations in each cohort, using a specified functional form of the CRF. For the remaining 26
161 cohorts, the concentration-response was examined with a linear concentration hazard ratio
162 model. A recent meta-analysis of the association between long-term $PM_{2.5}$ and mortality
163 (Vodonos et al., 2018) applied techniques involving flexible penalized spline CRF in a
164 multivariate random effects and meta-regression model. This approach allows the data to specify
165 the shape of the CRF. The meta-regression pooled 135 estimates from 53 studies examining
166 long-term $PM_{2.5}$ and mortality of cohorts aged 15 years and older. The estimate of the confidence
167 intervals about the CRF includes a random variance component. This meta-analysis provided
168 evidence of a nonlinear association between $PM_{2.5}$ exposure and mortality in which the exposure-
169 mortality slopes decreases at higher concentrations (Figure S5 in Supplemental Material). We
170 have chosen to use the dose-response function from the meta-analysis rather than the GEMM
171 function as the meta-regression approach is more flexible and does not constrain the CRF to a
172 specific functional form, it incorporates a random variance component in estimating the
173 uncertainty around that curve, it is derived with more studies than previous approaches, and its
174 estimates at high and low exposures are closer to the estimates in cohorts restricted to only very
175 high and very low exposures. To ensure consistency with the concentration-response curve,
176 premature mortality rates for the portion of the population >14 years of age were determined
177 using the population and baseline mortality rates for different age groups from GBD data for
178 2015.

179

180 *Health impact calculations*

181 We estimated the number of premature deaths attributable to fossil fuel $PM_{2.5}$ using: (1)
182 GEOS-Chem $PM_{2.5}$ estimated with all emission sources and GEOS-Chem $PM_{2.5}$ estimated

183 without fossil fuel emissions, as a comparison against the first simulation, (2) total population
 184 above the age of 14 gridded to the GEOS-Chem grid resolution, (3) baseline all-cause mortality
 185 rates for population above the age of 14 (per country or per state in the US and province in
 186 Canada), and (4) the meta-analysis CRF (Vodonos et al., 2018). All health impacts were
 187 calculated on a per-grid basis at the spatial resolution of the model. We applied the following
 188 health impact function to estimate premature mortality related to exposure to fossil fuel PM_{2.5} in
 189 each GEOS-Chem grid cell:

190

$$191 \quad \Sigma \Delta y = y_o * p * AF \quad (1)$$

$$192 \quad AF = \frac{\exp(\bar{\beta} * \Delta x) - 1}{\exp(\bar{\beta} * \Delta x)} \quad (2)$$

$$193 \quad \bar{\beta}(PM_{2.5}) = \int_{PM_{2.5} \text{ no fossil fuel}}^{PM_{2.5} \text{ all emissions}} \beta(PM_{2.5}) \quad (3)$$

194

195 where Δy is the change in the number of premature deaths due to exposure to fossil fuel PM_{2.5},
 196 y_o is the country/state/province specific baseline (all-cause) mortality rate, p is to the total
 197 population above the age of 14, AF is the attributable fraction of deaths (the fraction of total
 198 deaths attributable to PM_{2.5} exposure), $\bar{\beta}$ is the mean estimate for long-term PM_{2.5} mortality
 199 concentration-response over a range of concentrations from the penalized spline model in the
 200 recent meta-analysis, and Δx is the change in PM_{2.5} concentration, calculated as the difference
 201 between GEOS-Chem PM_{2.5} with all emissions and GEOS-Chem PM_{2.5} without fossil fuel
 202 emissions.

203

204 For each country, we summed the change in premature deaths (Δy) in each grid cell over all grid
205 cells in that country. To estimate the change in deaths between the two scenarios (with and
206 without fossil fuel combustion), we computed the change in deaths in each grid cell, based on its
207 population, baseline rate, and exposure under the two scenarios (Equation (1)). The attributable
208 fraction (AF), or proportion of deaths estimated as due to long-term exposure to $PM_{2.5}$ fossil fuel
209 air pollution, was calculated using the concentration-response estimate, following the form
210 shown in Equation (2) (Figure S5 in Supplemental material). Because these estimates of
211 mortality concentration response (β) are a nonlinear function of concentration, we used the
212 penalized spline model predictions from this meta-analysis to integrate the concentration-specific
213 β in each grid cell from the low $PM_{2.5}$ scenario (without fossil fuel emissions) to the high $PM_{2.5}$
214 scenario (with all emissions, including fossil fuel). In this way, we could calculate a mean value
215 of β for each grid cell. There exist insufficient epidemiological data to calculate a robust health
216 response function specific to fossil-fuel $PM_{2.5}$. GEOS-Chem is a deterministic model. Therefore,
217 our 95% confidence intervals (CI) for our estimates reflect only the 95% CI for the concentration
218 response function.

219 *Secondary analysis among children <5 years old*

220 Lower respiratory infections (LRI), including pneumonia and bronchiolitis of bacterial and viral
221 origin, are the largest single cause of mortality among young children worldwide and thus
222 account for a significant global burden of disease worldwide (Nair et al., 2010). As mentioned
223 previously, young children are more susceptible to the adverse effects of particulate air pollution
224 than adults. Mehta et al. (2013) estimated the overall impact of $PM_{2.5}$ concentration with Relative
225 Risk (RR) of 1.12 for LRI mortality per $10 \mu g m^{-3}$ increase in annual average $PM_{2.5}$
226 concentration, as compared to RR of 1.04 for respiratory mortality among adults (Vodonos et al.,

227 2018). We estimated the number of premature deaths attributable to PM_{2.5} among children under
228 the age of 5 years due to a range of LRI classifications (ICD-10, International Classification of
229 Diseases codes: A48.1, A70, J09-J15.8, J16-J16.9, J20-J21.9, P23.0-P23.4). Baseline numbers of
230 deaths due to LRI were obtained from the GBD for 2015 (IHME, 2017). We used the Relative
231 Risk (RR) of 1.12 (1.03-1.30) for LRI occurrence per 10 µg m⁻³ increase in annual average PM_{2.5}
232 concentration (Mehta et al., 2013). Studies of longer-term exposure of PM_{2.5} and LRI in that
233 meta-analysis were conducted in only a few developed countries with relatively low levels of
234 annual mean PM_{2.5} (< 25 µg m⁻³), specifically the Netherlands, Czech Republic, Germany,
235 Canada and USA. We therefore calculated the number of premature LRI deaths attributable to
236 PM_{2.5} only in North America, South America, and Europe.

237

238 **Results**

239 *Impact of fossil fuel use on PM_{2.5}*

240 Figure 1 shows the difference between global GEOS-Chem PM_{2.5} with and without fossil
241 fuel emissions, plotted as the annual mean for 2012. Results show large contributions of 50-100
242 µg m⁻³ in PM_{2.5} over China and India, with smaller increments of 10-50 µg m⁻³ over large swaths
243 of the United States and Europe, industrialized countries in Africa (South Africa and Nigeria),
244 and along the North African coastline due to European pollution.

245 *Global assessment of mortality attributable to PM_{2.5}*

246 Based on the annual PM_{2.5} simulation with and without global fossil fuel emissions, we
247 estimated the excess deaths and attributable fraction (AF %) for the population above 14 years

248 old. Figure 2 shows the simulated annual global premature mortality due to exposure to ambient
249 $PM_{2.5}$ from fossil fuel emissions. Greatest mortality is simulated over regions with substantial
250 influence of fossil-fuel related $PM_{2.5}$, notably parts of Eastern North America, western Europe,
251 and South-East Asia.

252 We estimated a total global annual burden premature mortality due to fossil fuel
253 combustion in 2012 of 10.2 million (95% CI: -47.1 to 17.0 million). Table 1 reports the baseline
254 number of deaths for people >14 years old, the annual $PM_{2.5}$ simulation with and without global
255 fossil fuel emissions, the estimated excess deaths, and the attributable fraction for the populated
256 continents. As shown in Table 1, we calculated 483,000 premature deaths in North America
257 (95% CI: 284,000-670,000), 187,000 deaths in South America (95% CI: 107,000-263,000),
258 1,447,000 deaths in Europe (95% CI: 896,000-1,952,000), 7,916,000 deaths in Asia (95% CI: -
259 48,106,000 to 13,622,000), and 194,000 deaths in Africa (95% CI: -237,000 to 457,000). The
260 wide confidence intervals in Asia and Africa are due to the lack of data for areas where the
261 exposure remains outside the range of the concentration response curve ($PM_{2.5} > 50 \mu g m^{-3}$;
262 Figure S5). The population-weighted pollution concentrations presented in Table 1 are higher
263 than the average $PM_{2.5}$ concentrations for each country, since fossil-fuel $PM_{2.5}$ is mainly emitted
264 in populous areas. The two countries with the highest premature mortality are China with 3.91
265 million and India with 2.46 million. Supplemental Table S2 provides extended data of the health
266 impact calculations for each country. For comparison, Table 1 also reports the number of
267 premature deaths attributable to fossil fuel $PM_{2.5}$ when the GEMM function is applied to the
268 GEOS-Chem output. For most regions, the number of premature deaths calculated with GEMM
269 is significantly lower than that calculated with the new function from Vodonos et al. (2018).
270 Globally, the GEMM function yields 6.7 million deaths in 2012 due to fossil fuel combustion.

271

272 *Assessment of children (under the age of 5) LRI mortality attributable to PM_{2.5}*

273 We estimated the number of premature deaths attributable to PM_{2.5} among children under
274 the age of 5 due to LRI only for those countries or regions with levels of annual PM_{2.5}
275 concentrations below 25 µg m⁻³. These include North America, South America, and Europe.
276 Based on the annual PM_{2.5} simulation with and without fossil fuel emissions, we calculated 876
277 excess deaths due to LRI in North and Central America, 747 in South America, and 605 in
278 Europe (Table 2). Using the GBD estimate of total deaths due to LRI (Institute for Health
279 Metrics and Evaluation), we estimate that PM_{2.5} from fossil fuel combustion accounted on
280 average for 7.2% of LRI mortality among children under the age of 5 in these regions, with the
281 largest proportion of 13.6% in Europe (95% CI -0.4 to 25.3%) .

282

283 **Discussion**

284 We used the chemical transport model GEOS-Chem to quantify the global mortality
285 attributed to PM_{2.5} air pollution from fossil fuel combustion. Using the updated concentration
286 response relationship between relative mortality and airborne PM_{2.5}, we estimated global
287 premature mortality in 2012 of 10.2 million per year from fossil fuel combustion alone. China
288 has the highest burden of 3.91 million per year, followed by India with 2.46 million per year.
289 These estimates carry large uncertainty (e.g., 95% CI of -47.1 to 17.0 million for the global
290 estimate) from the concentration-response curve, as it is an improved function that provides a
291 more realistic picture of the health consequences of PM_{2.5} compared to previous studies.

292 Our estimate is for the year when fossil fuel emissions in China peaked and so predates
293 large and dramatic reductions in fossil fuel emissions due to strict mitigation measures. These
294 reductions led to a 30-50% decline in annual mean $PM_{2.5}$ across the country from 2013 to 2018
295 (Zhai et al., 2019). If we apply a 43.7% reduction in GEOS-Chem $PM_{2.5}$ concentrations from the
296 simulation with all emission sources, premature mortality in China decreases from 3.91 million
297 to 2.36 million. India has recently imposed controls on pollution sources, but there is not yet
298 evidence of air quality improvements in densely populated cities like Delhi (Vohra et al., 2020).
299 Consideration of the 2012-2018 decrease in $PM_{2.5}$ exposure in China reduces the total global
300 premature mortality due to fossil fuel $PM_{2.5}$ from 10.2 million premature deaths each year to 8.7
301 (95% CI: -1.8 to 14.0) million.

302 In 2012, the population-weighted $PM_{2.5}$ is $72.8 \mu\text{g m}^{-3}$ for China and $52.0 \mu\text{g m}^{-3}$ for India
303 from all sources and $9.9 \mu\text{g m}^{-3}$ for China and $9.0 \mu\text{g m}^{-3}$ for India without fossil fuel emissions.
304 The low value of non-fossil fuel $PM_{2.5}$ is reasonable for southern India (Dey et al., 2012) but
305 may be an underestimate in the Indo-Gangetic Plain where crop residue burning contributes to
306 high levels of $PM_{2.5}$ ($100\text{-}200 \mu\text{g m}^{-3}$) during the post-monsoon season (Ojha et al., 2020). An
307 increase in the concentration of non-fossil-fuel $PM_{2.5}$ would decrease our estimate of the number
308 of premature deaths due to fossil fuel $PM_{2.5}$ in India and China, as this would decrease the risk of
309 premature mortality with a unit change in $PM_{2.5}$ (Figure S5).

310

311 *Comparison with previous estimates of global mortality attributable to outdoor $PM_{2.5}$*

312 Previous estimates of the GBD for 2015 suggest that exposure to total $PM_{2.5}$ causes 4.2
313 million deaths (Cohen et al., 2017), whereas here we estimate more than double (10.2 million)

314 the number of premature deaths from fossil fuel combustion alone in 2012. Differences between
315 the current study and the 2015 GBD lower estimates are related mainly to the choice of the shape
316 of the concentration-response function and the relative risk estimate. First, to provide
317 information about exposure response at higher concentrations, the 2015 GBD study used the
318 integrated exposure–response (IER) model in which active and second-hand smoking exposures
319 were converted to estimated annual PM_{2.5} exposure equivalents using inhaled doses of particle
320 mass (Burnett et al., 2014). Recent cohort studies from Asia indicate that this substantially
321 underestimates the CRF at high concentrations. In contrast, in the current study we applied a
322 CRF that was directly estimated from PM_{2.5} studies alone, as described in a recent meta-analysis
323 that included estimates from studies in countries like China with higher PM_{2.5} concentrations
324 than our included in previous derivations of CRFs (Vodonos et al., 2018). The CRF from this
325 recent meta-analysis flattens out at higher concentrations, as does the IER curve. However, this
326 flattening is not as great as in the IER, as Asian cohort studies at high PM_{2.5} concentrations
327 report larger effects than would be expected from the IER. Hence estimates of the global
328 attributable fraction of deaths due to air pollution using the function from the recent meta-
329 analysis are higher than the estimates using the IER function. In addition, at much lower
330 concentrations ($< 10 \mu\text{g m}^{-3}$), we applied higher slopes than assumed in the IER function. Recent
331 studies at very low concentrations similarly show that the IER underestimated effects in this
332 range (Pinault et al., 2016). Since GEOS-Chem estimated quite low concentrations in developed
333 countries in Europe and North America, the number of premature deaths from PM_{2.5} in these
334 countries is greater than previous estimates.

335 Following an approach similar to the recent meta-analysis (Vodonos et al., 2018), Burnett
336 et al. (2018) modeled the shape of the association between PM_{2.5} and non-accidental mortality

337 using data from 41 cohorts from 16 countries with GEMM. In that study, the uncertainty in a
338 subset (15 cohorts) was characterized in the shape of the concentration-response parameter by
339 calculating the Shape-Constrained Health Impact Function, a prespecified functional form. These
340 estimated shapes varied across the cohorts included in the function. GEMM predicted 8.9 million
341 (95% CI: 7.5–10.3) deaths in 2015 attributable to long-term exposure to PM_{2.5} from all sources;
342 120% higher excess deaths than previous estimates, but still lower than our estimate of mortality
343 from exposure to fossil-fuel derived PM_{2.5} for 2012. Lelieveld et al. (2019) estimated the global
344 and regional mortality burden of fossil fuel attributable PM_{2.5} by applying the GEMM CRF to a
345 global chemistry-climate model that is overall coarser (~1.9° latitude and longitude) than the
346 model used in this work. The authors reported 3.61 million deaths per year attributable to
347 pollution from fossil fuel combustion and 5.55 million deaths per year due to pollution from all
348 anthropogenic sources. The estimated deaths from fossil fuel combustion are much lower than
349 those in the current study for several reasons. First, the meta-analysis function used in our work
350 includes 135 coefficients of all-cause mortality for adults aged 14-64 years old, together with
351 cause-specific mortality and all-cause mortality among adults aged 65 and older, thus
352 incorporating many more studies in a meta-regression framework than the 41 cohorts and
353 coefficients in the GEMM function. Second, the approach used to estimate the CRF in Vodonos
354 et al. (2018) allows for additional flexibility in the shape of the function because of its use of
355 penalized splines. In contrast, the GEMM pooled CRF integrates a set of 26 log-linear functions
356 and 15 functions characterized by three parameters governing the shape of the function. Third,
357 while Cohen et al. (2017), Lelieveld et al. (2019) and Burnett et al. (2018) accounted for
358 mortality from five specific causes (ischemic heart disease, stroke, chronic obstructive
359 pulmonary disease, lung cancer and acute respiratory infections), in the current analysis we

360 estimated changes in deaths from all causes. Fourth, some of the difference in the mortality
361 estimates may come from differences in the age range. Our approach considers a wider
362 population age range of over 14 years old (Vodonos et al., 2018) compared to the other studies,
363 which considered a population age range of over 25 years (Burnett et al., 2018; Cohen et al.,
364 2017; Lelieveld et al., 2019). Our approach has wider age range since the age range for the
365 studies in the meta-analysis (Vodonos et al., 2018) included people younger than 25 years old
366 (Hart et al., 2011; Pinault et al., 2016) . Finally, the finer spatial resolution that GEOS-Chem
367 utilizes over much of the globe improves co-location of PM hotspots and population centers,
368 yielding higher estimates of excess mortality compared to Lelieveld et al. (2019).

369

370 *Limitations*

371 There are a number of limitations that must be acknowledged. First, vulnerability to
372 PM_{2.5} exposure may vary by population characteristics such as ethnicity, socio-economic status
373 (SES), risk behaviors such as smoking and underlying comorbidities (Krewski et al., 2000; Pope
374 et al., 2004; Wang et al., 2017) and by different exposure characteristics. We were limited in our
375 ability to undertake a comprehensive analysis of factors influencing the association between
376 PM_{2.5} and mortality since the global mortality data were not available by detailed age, ethnicity,
377 SES, lifestyle, and underlying disease strata. In addition, the 95% CI of our estimates reflect the
378 lower and upper bound of the CRF, which flattens out at higher concentrations. Regions with
379 very high concentrations ($>50 \mu\text{g m}^{-3}$) are beyond the data range in the meta-analysis; thus, the
380 lower limit of the CI for those regions (China, West and North Africa; Table 1) are much less
381 than zero. Second, for LRI in children, we have restricted our analysis to developed countries

382 with annual $PM_{2.5} < 25 \mu g m^{-3}$, in accordance with the geographical locations of the studies
383 included in the meta-analysis by Mehta et al. (2013). Developing countries have much higher
384 LRI mortality rates, and this restriction doubtless results in an underestimate. Finally, GEOS-
385 Chem estimates of $PM_{2.5}$ concentrations almost certainly contains errors in estimates of
386 emissions of pollution precursors, meteorological effects on air quality, and representation of the
387 complex physical and chemical formation pathways. In the absence of systematic bias, such
388 model error may not produce large aggregate errors in the mortality burden of $PM_{2.5}$, but bias
389 may be present as well. In any event, it is challenging to estimate the true size of this error.

390

391 **Conclusions**

392 The effects of CO_2 -driven climate change on human health and welfare are complex, ranging
393 from greater incidence of extreme weather events, more frequent storm-surge flooding, and
394 increased risk of crop failure (Duffy et al., 2019). One consequence of increasing reliance on
395 fossil fuel as an energy source that has thus far received comparatively little attention is the
396 potential health impact of the pollutants co-emitted with the greenhouse gas CO_2 . Such
397 pollutants include $PM_{2.5}$ and the gas-phase precursors of $PM_{2.5}$. This study demonstrates that the
398 fossil fuel component of $PM_{2.5}$ contributes a large global mortality burden. By quantifying this
399 sometimes overlooked health consequence of fossil fuel combustion, a clear message is sent to
400 policymakers and stakeholders of the co-benefits of a transition to alternative energy sources.

401 **Acknowledgments**

402 This study was funded by the Wallace Global Fund, the Environment and Health Fund
403 (EHF) Israel, and a University of Birmingham Global Challenges Fund PhD studentship awarded
404 to KV.

405 **Declaration of interests**

406 We declare no competing interests.

407 **Data availability.** GEOS-Chem code and output are available at the GEOS-Chem website
408 (http://acmg.seas.harvard.edu/geos_chem.html) and upon request.

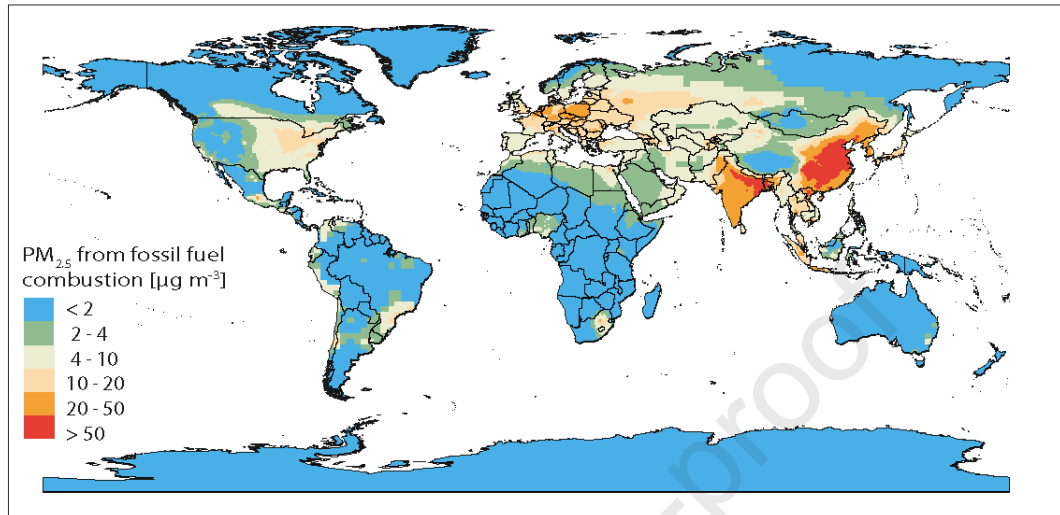
Figures

Figure 1: Contribution of fossil fuel combustion to surface PM_{2.5}, as calculated by the chemical transport model GEOS-Chem. The plot shows the difference in surface PM_{2.5} concentrations from GEOS-Chem with and without fossil fuel emissions.

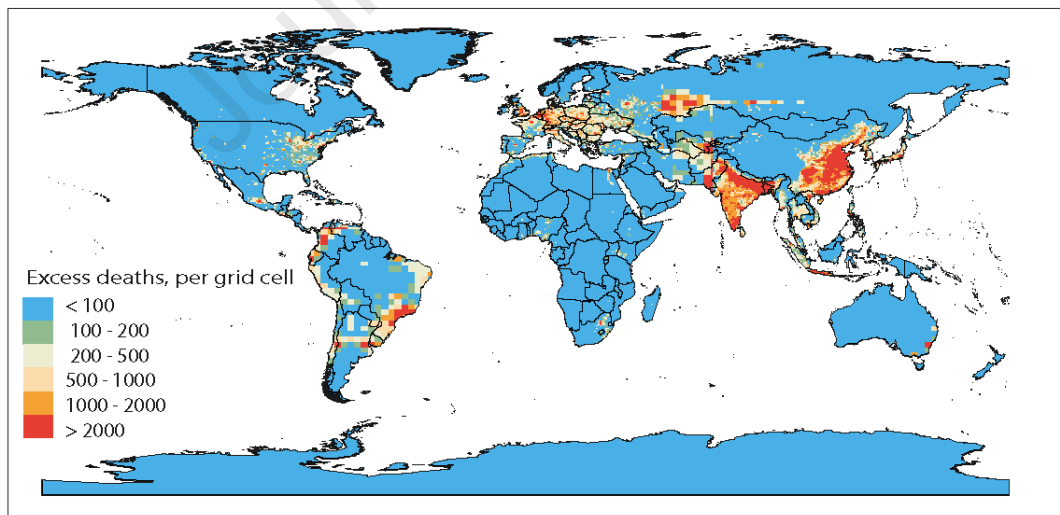


Figure 2. Estimated annual excess deaths due to exposure to ambient PM_{2.5} generated by fossil fuel combustion.

Journal Pre-proof

Table 1. Number of deaths attributable to exposure to fine particulate matter (PM_{2.5}) generated by fossil fuel combustion for the population >14 years old

GEOS-Chem spatial grid resolution ^a	Region ^b		Total deaths >14 years old, in thousands	Population-weighted annual mean PM _{2.5} concentration, $\mu\text{g m}^{-3}$			Mean attributable fraction of deaths, % (95% CI) ^d	Deaths attributable to fossil-fuel related PM _{2.5} , in thousands (95% CI) ^c	GEMM function deaths attributable to fossil-fuel related PM _{2.5} , in thousands (95% CI) ^e
				PM _{2.5} from all emission sources	PM _{2.5} without fossil fuel	Estimated PM _{2.5} from fossil fuel, %			
Fine	North America	Central America & the Caribbean	1,148	10.06	3.03	7.03 (69.9)	8.2 (4.5-11.6)	94 (52-133)	80 (62-98)
		USA	2,705	11.81	2.15	9.66 (81.8)	13.1 (7.8-18.1)	355 (212-490)	305 (233-375)
		Canada	250	12.01	1.76	10.25 (85.4)	13.6 (8.0-18.7)	34 (20-47)	28 (22-35)
Coarse	South America		2,389	8.66	3.02	5.65 (65.2)	7.8 (4.5-11.0)	187 (107-263)	159 (121-195)
Fine	Europe		8,626	19.22	4.68	14.54 (75.7)	16.8 (10.4-22.6)	1,447 (896-1,952)	1,033 (798-1,254)
Fine	Asia	Eastern Asia	25,468	51.72	8.68	43.05 (83.2)	30.7 (-189.1-52.9)	7,821 (-48,150-13,478)	4,945 (3,943-5,826)
Coarse		Western Asia & the Middle East	1,456	26.95	20.73	6.22 (23.1)	6.5 (3.0-9.9)	95 (44-144)	54 (43-65)
Fine	Africa		5,274	32.98	28.98	4.00 (12.1)	3.7 (-4.5-8.7)	194 (-237-457)	102 (81-121)
Coarse	Australia & Oceania		189	4.17	2.19	1.98 (47.4)	3.2 (1.6-4.8)	6.0 (2.9-9.0)	6.4 (4.8-7.9)
	Global		47,506	38.01	11.14	26.87 (70.7)	21.5 (-99.0-35.7)	10,235 (-47,054-16,972)	6,713 (5,308-7,976)

^a Fine spatial scale is $0.5^\circ \times 0.67^\circ$, or about $50 \text{ km} \times 60 \text{ km}$. Coarse spatial scale is $2^\circ \times 2.5^\circ$, or about $200 \text{ km} \times 250 \text{ km}$

^b List of countries for each region and subregion is provided in supplemental Table S2

^c Annual number of deaths attributable to long-term exposure to PM_{2.5} derived from fossil fuel combustion. CI is the confidence interval.

^d Mean proportion of all deaths which can be attributed to long-term exposure to PM_{2.5} generated by fossil fuel combustion, averaged over the country or region. CI; confidence interval.

^e Attributable deaths calculated with the Global Exposure Mortality Model (GEMM) concentration-response function.⁴⁴

Table 2. Number of deaths due to lower respiratory infection (LRI) attributable to exposure to fine particulate matter (PM_{2.5}) from fossil fuel combustion for the population <5 years old

Region	Total deaths for children <5 years old due to LRI	LRI deaths attributable to fossil-fuel PM_{2.5} (95% CI)^a	Mean attributable fraction of deaths, % (95% CI)^b
North America	13,230	876 (-26-1,657)	6.6 (-0.2-12.5)
Central America & the Caribbean	12,507	802 (-23-1,516)	6.4 (-0.2-12.1)
USA	672	69 (-2-131)	10.2 (-0.3-19.5)
Canada	50	5 (0-10)	10.8 (-0.3-20.5)
South America	13,231	747 (-21-1,443)	5.7 (-0.2-10.9)
Europe	4,446	605 (-18-1,126)	13.6 (-0.4-25.3)

^a Annual number of deaths attributed to long-term exposure to PM_{2.5} derived from fossil fuel combustion.

^b Mean proportion of deaths due to long-term exposure to PM_{2.5} generated by fossil fuel combustion. CI is the confidence interval.

References

1. Apte, J. S., Marshall, J. D., Cohen, A. J., et al., Addressing Global Mortality from Ambient PM_{2.5}, *Environ Sci Technol*, 49, 8057-8066, doi:10.1021/acs.est.5b01236, 2015.
2. Atkinson, R. W., Butland, B. K., Dimitroulopoulou, C., et al., Long-term exposure to ambient ozone and mortality: a quantitative systematic review and meta-analysis of evidence from cohort studies, *Bmj Open*, 6, doi:10.1136/bmjopen-2015-009493, 2016.
3. Bateson, T. F., Schwartz, J., Children's response to air pollutants, *J Toxicol Env Heal A*, 71, 238-243, doi:10.1080/15287390701598234, 2008.
4. Brauer, M., Freedman, G., Frostad, J., et al., Ambient Air Pollution Exposure Estimation for the Global Burden of Disease 2013, *Environ Sci Technol*, 50, 79-88, doi:10.1021/acs.est.5b03709, 2016.
5. Brook, R. D., Rajagopalan, S., Pope, C. A., et al., Particulate Matter Air Pollution and Cardiovascular Disease An Update to the Scientific Statement From the American Heart Association, *Circulation*, 121, 2331-2378, doi:10.1161/CIR.0b013e3181d8e1, 2010.
6. Burnett, R., Chen, H., Szyszkowicz, M., et al., Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter, *P Natl Acad Sci USA*, 115, 9592-9597, doi:10.1073/pnas.1803222115, 2018.
7. Burnett, R., Pope, C. A., Ezzati, M., et al., An Integrated Risk Function for Estimating the Global Burden of Disease Attributable to Ambient Fine Particulate Matter Exposure, *Environ Health Persp*, 122, 397-403, doi:10.1289/ehp.1307049, 2014.
8. Canada, S., Government of Canada. <https://www150.statcan.gc.ca/n1/en/type/data>, 2018.
9. CIESIN, Center for International Earth Science Information Network - Columbia University; Gridded Population of the World, Version 4 (GPWv4): Population Count Adjusted to Match 2015 Revision of UN WPP Country Totals, Revision 11. NASA Socioeconomic Data and Applications Center (SEDAC), Palisades, NY, <https://doi.org/10.7927/H4PN93PB>, 2018.
10. Cohen, A. J., Brauer, M., Burnett, R., et al., Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015, *Lancet*, 389, 1907-1918, doi:10.1016/S0140-6736(17)30505-6, 2017.

11. Dedoussi, I. C., Barrett, S. R. H., Air pollution and early deaths in the United States. Part II: Attribution of PM_{2.5} exposure to emissions species, time, location and sector, *Atmos Environ*, 99, 610-617, doi:10.1016/j.atmosenv.2014.10.033, 2014.
12. Dey, S., Di Girolamo, L., van Donkelaar, A., et al., Variability of outdoor fine particulate (PM_{2.5}) concentration in the Indian Subcontinent: A remote sensing approach, *Remote Sens Environ*, 127, 153-161, doi:10.1016/j.rse.2012.08.021, 2012.
13. Di, Q., Wang, Y., Zanobetti, A., et al., Air Pollution and Mortality in the Medicare Population, *N Engl J Med*, 376, 2513-2522, doi:10.1056/NEJMoa1702747, 2017.
14. Drury, E., Jacob, D. J., Spurr, R. J. D., et al., Synthesis of satellite (MODIS), aircraft (ICARTT), and surface (IMPROVE, EPA-AQS, AERONET) aerosol observations over eastern North America to improve MODIS aerosol retrievals and constrain surface aerosol concentrations and sources, *J Geophys Res-Atmos*, 115, doi:10.1029/2009jd012629, 2010.
15. Duffy, P. B., Field, C. B., Diffenbaugh, N. S., et al., Strengthened scientific support for the Endangerment Finding for atmospheric greenhouse gases, *Science*, 363, 597-+, doi:10.1126/science.aat5982, 2019.
16. Ford, B., Heald, C. L., Aerosol loading in the Southeastern United States: reconciling surface and satellite observations, *Atmos Chem Phys*, 13, 9269-9283, doi:10.5194/acp-13-9269-2013, 2013.
17. Forouzanfar, M. H., Afshin, A., Alexander, L. T., et al., Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015, *Lancet*, 388, 1659-1724, doi:10.1016/S0140-6736(16)31679-8, 2016.
18. Hart, J. E., Garshick, E., Dockery, D. W., et al., Long-Term Ambient Multipollutant Exposures and Mortality, *Am J Resp Crit Care*, 183, 73-78, doi:10.1164/rccm.200912-1903OC, 2011.
19. Heald, C. L., Collett, J. L., Lee, T., et al., Atmospheric ammonia and particulate inorganic nitrogen over the United States, *Atmos Chem Phys*, 12, 10295-10312, doi:10.5194/acp-12-10295-2012, 2012.
20. IHME, Institute for Health Metrics and Evaluation. <http://ghdx.healthdata.org/gbd-results-tool>, 2017.

21. Katanoda, K., Sobue, T., Satoh, H., et al., An Association Between Long-Term Exposure to Ambient Air Pollution and Mortality From Lung Cancer and Respiratory Diseases in Japan, *J Epidemiol*, 21, 132-143, doi:10.2188/jea.JE20100098, 2011.
22. Koplitz, S. N., Mickley, L. J., Marlier, M. E., et al., Public health impacts of the severe haze in Equatorial Asia in September-October 2015: demonstration of a new framework for informing fire management strategies to reduce downwind smoke exposure, *Environ Res Lett*, 11, doi:10.1088/1748-9326/11/9/094023, 2016.
23. Krewski, D., Burnett, R. T., Goldberg, M. S., et al., Special report reanalysis of the Harvard six cities study and the American Cancer Society Study of particulate air pollution and mortality part II: Sensitivity Analyses Appendix C. Flexible Modeling of the Effects of Fine Particles and Sulphate on Mortality, Health Effects Institute, <https://www.healtheffects.org/system/files/SR-PartIIAppC.pdf>, 2000.
24. Lacey, F. G., Marais, E. A., Henze, D. K., et al., Improving present day and future estimates of anthropogenic sectoral emissions and the resulting air quality impacts in Africa, *Faraday Discuss*, 200, 397-412, doi:10.1039/c7fd00011a, 2017.
25. Leibensperger, E. M., Mickley, L. J., Jacob, D. J., et al., Climatic effects of 1950-2050 changes in US anthropogenic aerosols - Part 1: Aerosol trends and radiative forcing, *Atmos Chem Phys*, 12, 3333-3348, doi:10.5194/acp-12-3333-2012, 2012.
26. Lelieveld, J., Klingmuller, K., Pozzer, A., et al., Effects of fossil fuel and total anthropogenic emission removal on public health and climate, *P Natl Acad Sci USA*, 116, 7192-7197, doi:10.1073/pnas.1819989116, 2019.
27. Lim, S. S., Vos, T., Flaxman, A. D., et al., A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010 (vol 380, pg 2224, 2012), *Lancet*, 381, 628-628, <Go to ISI>://WOS:000315189300032, 2013.
28. Lin, J. T., van Donkelaar, A., Xin, J. Y., et al., Clear-sky aerosol optical depth over East China estimated from visibility measurements and chemical transport modeling, *Atmos Environ*, 95, 258-267, doi:10.1016/j.atmosenv.2014.06.044, 2014.
29. Marais, E. A., Jacob, D. J., Guenther, A., et al., Improved model of isoprene emissions in Africa using Ozone Monitoring Instrument (OMI) satellite observations of formaldehyde: implications for oxidants and particulate matter, *Atmos Chem Phys*, 14, 7693-7703, doi:10.5194/acp-14-7693-2014, 2014a.

30. Marais, E. A., Jacob, D. J., Jimenez, J. L., et al., Aqueous-phase mechanism for secondary organic aerosol formation from isoprene: application to the southeast United States and co-benefit of SO₂ emission controls, *Atmos Chem Phys*, 16, 1603-1618, doi:10.5194/acp-16-1603-2016, 2016.
31. Marais, E. A., Jacob, D. J., Wecht, K., et al., Anthropogenic emissions in Nigeria and implications for atmospheric ozone pollution: A view from space, *Atmos Environ*, 99, 32-40, doi:10.1016/j.atmosenv.2014.09.055, 2014b.
32. Marais, E. A., Silvern, R. F., Vodonos, A., et al., Air Quality and Health Impact of Future Fossil Fuel Use for Electricity Generation and Transport in Africa, *Environ Sci Technol*, 53, 13524-13534, doi:10.1021/acs.est.9b04958, 2019.
33. Marais, E. A., Wiedinmyer, C., Air Quality Impact of Diffuse and Inefficient Combustion Emissions in Africa (DICE-Africa), *Environ Sci Technol*, 50, 10739-10745, doi:10.1021/acs.est.6b02602, 2016.
34. Mehta, S., Shin, H., Burnett, R., et al., Ambient particulate air pollution and acute lower respiratory infections: a systematic review and implications for estimating the global burden of disease, *Air Qual Atmos Hlth*, 6, 69-83, doi:10.1007/s11869-011-0146-3, 2013.
35. Nair, H., Nokes, D. J., Gessner, B. D., et al., Global burden of acute lower respiratory infections due to respiratory syncytial virus in young children: a systematic review and meta-analysis, *Lancet*, 375, 1545-1555, doi:10.1016/S0140-6736(10)60206-1, 2010.
36. Ojha, N., Sharma, A., Kumar, M., et al., On the widespread enhancement in fine particulate matter across the Indo-Gangetic Plain towards winter, *Sci Rep-Uk*, 10, doi:10.1038/s41598-020-62710-8, 2020.
37. Perera, F., Pollution from Fossil-Fuel Combustion is the Leading Environmental Threat to Global Pediatric Health and Equity: Solutions Exist, *Int J Env Res Pub He*, 15, doi:10.3390/ijerph15010016, 2018.
38. Pinault, L., Tjepkema, M., Crouse, D. L., et al., Risk estimates of mortality attributed to low concentrations of ambient fine particulate matter in the Canadian community health survey cohort, *Environ Health-Glob*, 15, doi:10.1186/s12940-016-0111-6, 2016.

39. Pope, C. A., Burnett, R. T., Thurston, G. D., et al., Cardiovascular mortality and long-term exposure to particulate air pollution - Epidemiological evidence of general pathophysiological pathways of disease, *Circulation*, 109, 71-77, doi:10.1161/01.Cir.0000108927.80044.7f, 2004.
40. Protonotariou, A. P., Bossioli, E., Tombrou, M., et al., Air Pollution in Eastern Mediterranean: Nested-Grid GEOS-CHEM Model Results and Airborne Observations. *Advances in Meteorology, Climatology and Atmospheric Physics*. Springer Atmospheric Sciences, Springer, Berlin, Heidelberg, 2013, pp. 1203-1209.
41. Shaddick, G., Thomas, M. L., Green, A., et al., Data integration model for air quality: a hierarchical approach to the global estimation of exposures to ambient air pollution, *J R Stat Soc C-Appl*, 67, 231-253, doi:10.1111/rssc.12227, 2018.
42. Shindell, D., Faluvegi, G., Seltzer, K., et al., Quantified, localized health benefits of accelerated carbon dioxide emissions reductions, *Nat Clim Change*, 8, doi:10.1038/s41558-018-0108-y, 2018.
43. Tseng, E., Ho, W. C., Lin, M. H., et al., Chronic exposure to particulate matter and risk of cardiovascular mortality: cohort study from Taiwan, *Bmc Public Health*, 15, doi:10.1186/s12889-015-2272-6, 2015.
44. Ueda, K., Nagasawa, S., Nitta, H., et al., Exposure to Particulate Matter and Long-term Risk of Cardiovascular Mortality in Japan: NIPPON DATA80, *J Atheroscler Thromb*, 19, 246-254, doi:10.5551/jat.9506, 2012.
45. Veefkind, J. P., Boersma, K. F., Wang, J., et al., Global satellite analysis of the relation between aerosols and short-lived trace gases, *Atmos Chem Phys*, 11, 1255-1267, doi:10.5194/acp-11-1255-2011, 2011.
46. Vodonos, A., Abu Awad, Y., Schwartz, J., The concentration-response between long-term PM_{2.5} exposure and mortality; A meta-regression approach, *Environ Res*, 166, 677-689, doi:10.1016/j.envres.2018.06.021, 2018.
47. Vohra, K., Marais, E. A., Suckra, S., et al., Long-term trends in air quality in major cities in the UK and India: A view from space, *Atmospheric Chemistry and Physics Discussions*, doi:10.5194/acp-2020-342, 2020.
48. Wang, Y., Shi, L. H., Lee, M., et al., Long-term Exposure to PM_{2.5} and Mortality Among Older Adults in the Southeastern US, *Epidemiology*, 28, 207-214, doi:10.1097/Ede.0000000000000614, 2017.

49. WHO, World Health Organization; Principles for evaluating health risks in children associated with exposure to chemicals. <https://apps.who.int/iris/handle/10665/43604>, 2006.
50. WHO, World Health Organization; Ambient air pollution: A global assessment of exposure and burden of disease. <https://www.who.int/phe/publications/air-pollution-global-assessment/en/>, 2016.
51. WONDER, C., Centers for Disease Control and Prevention Wide-ranging ONline Data for Epidemiologic Research <https://wonder.cdc.gov/>.
52. Wong, C. M., Lai, H. K., Tsang, H., et al., Satellite-Based Estimates of Long-Term Exposure to Fine Particles and Association with Mortality in Elderly Hong Kong Residents, *Environ Health Persp*, 123, 1167-1172, doi:10.1289/ehp.1408264, 2015.
53. Wong, C. M., Tsang, H., Lai, H. K., et al., Cancer Mortality Risks from Long-term Exposure to Ambient Fine Particle, *Cancer Epidem Biomar*, 25, 839-845, doi:10.1158/1055-9965.Epi-15-0626, 2016.
54. Xing, J., Mathur, R., Pleim, J., et al., Can a coupled meteorology-chemistry model reproduce the historical trend in aerosol direct radiative effects over the Northern Hemisphere?, *Atmos Chem Phys*, 15, 9997-10018, doi:10.5194/acp-15-9997-2015, 2015.
55. Xu, Z. W., Sheffield, P. E., Hu, W. B., et al., Climate Change and Children's Health-A Call for Research on What Works to Protect Children, *Int J Env Res Pub He*, 9, 3298-3316, doi:10.3390/ijerph9093298, 2012.
56. Yin, P., Brauer, M., Cohen, A., et al., Long-term Fine Particulate Matter Exposure and Nonaccidental and Cause-specific Mortality in a Large National Cohort of Chinese Men, *Environ Health Persp*, 125, doi:10.1289/Ehp1673, 2017.
57. Zhai, S. X., Jacob, D. J., Wang, X., et al., Fine particulate matter (PM_{2.5}) trends in China, 2013-2018: separating contributions from anthropogenic emissions and meteorology, *Atmos Chem Phys*, 19, 11031-11041, doi:10.5194/acp-19-11031-2019, 2019.
58. Zhang, L., Jacob, D. J., Knipping, E. M., et al., Nitrogen deposition to the United States: distribution, sources, and processes, *Atmos Chem Phys*, 12, 4539-4554, doi:10.5194/acp-12-4539-2012, 2012.

Highlights

- Fossil fuel combustion emits particulate matter (PM_{2.5}) harmful to public health
- We use a re-evaluated concentration-response function (CRF)
- We estimate 10.2 million global excess deaths in 2012 due to PM_{2.5} from this source
- 62% of deaths are in China (3.9 million) and India (2.5 million)
- Our estimate is more than double the GBD reports, due to the updated CRF we use

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Journal Pre-proof