

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

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18

19 **Abstract**

20 The burning of fossil fuels – especially coal, petrol, and diesel – is a major source of airborne fine  
21 particulate matter (PM<sub>2.5</sub>), and a key contributor to the global burden of mortality and disease.

22 Previous risk assessments have examined the health response to total PM<sub>2.5</sub>, not just PM<sub>2.5</sub> from  
23 fossil fuel combustion, and have used a concentration-response function with limited support from

24 the literature and data at both high and low concentrations. This assessment examines mortality  
25 associated with PM<sub>2.5</sub> from only fossil fuel combustion, making use of a recent meta-analysis of  
26 newer studies with a wider range of exposure. We also estimated mortality due to lower respiratory  
27 infections (LRI) among children under the age of five in the Americas and Europe, regions for  
28 which we have reliable data on the relative risk of this health outcome from PM<sub>2.5</sub> exposure. We  
29 used the chemical transport model GEOS-Chem to estimate global exposure levels to fossil-fuel  
30 related PM<sub>2.5</sub> in 2012. Relative risks of mortality were modeled using functions that link long-term  
31 exposure to PM<sub>2.5</sub> and mortality, incorporating nonlinearity in the concentration response. We  
32 estimate a global total of 10.2 (95% CI: -47.1 to 17.0) million premature deaths annually  
33 attributable to the fossil-fuel component of PM<sub>2.5</sub>. The greatest mortality impact is estimated over  
34 regions with substantial fossil fuel related PM<sub>2.5</sub>, notably China (3.9 million), India (2.5 million)  
35 and parts of eastern US, Europe and Southeast Asia. The estimate for China predates substantial  
36 decline in fossil fuel emissions and decreases to 2.4 million premature deaths due to 43.7%  
37 reduction in fossil fuel PM<sub>2.5</sub> from 2012 to 2018 bringing the global total to 8.7 (95% CI: -1.8 to  
38 14.0) million premature deaths. We also estimated excess annual deaths due to LRI in children (0-  
39 4 years old) of 876 in North America, 747 in South America, and 605 in Europe. This study  
40 demonstrates that the fossil fuel component of PM<sub>2.5</sub> contributes a large mortality burden. The  
41 steeper concentration-response function slope at lower concentrations leads to larger estimates  
42 than previously found in Europe and North America, and the slower drop-off in slope at higher  
43 concentrations results in larger estimates in Asia. Fossil fuel combustion can be more readily  
44 controlled than other sources and precursors of PM<sub>2.5</sub> such as dust or wildfire smoke, so this is a  
45 clear message to policymakers and stakeholders to further incentivize a shift to clean sources of  
46 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 key  
51 contributors to the global burden of mortality and disease (Apte et al., 2015; Dedoussi and Barrett,  
52 2014; Lim et al., 2013). A series of studies have reported an association between exposure to air  
53 pollution and adverse health outcomes (Brook et al., 2010), even at low exposure levels ( $< 10 \mu\text{g}$   
54  $\text{m}^{-3}$ , the current World Health Organization, WHO, guideline) (Di et al., 2017). The Global Burden  
55 of Diseases, Injuries, and Risk Factors Study 2015 (GBD 2015) identified ambient air pollution as  
56 a leading cause of the global disease burden, especially in low-income and middle-income  
57 countries (Forouzanfar et al., 2016). Recent estimates of the global burden of disease suggest that  
58 exposure to  $\text{PM}_{2.5}$  (particulate matter with an aerodynamic diameter  $< 2.5 \mu\text{m}$ ) causes 4.2 million  
59 deaths and 103.1 million disability-adjusted life-years (DALYs) in 2015, representing 7.6% of  
60 total global deaths and 4.2% of global DALYs, with 59% of these in east and south Asia (Cohen  
61 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 populations  
69 which incorporates those new findings at high and low  $\text{PM}_{2.5}$  concentrations (Vodonos et al.,

70 2018). We also focus our study on the health impacts of fossil-fuel derived PM<sub>2.5</sub>. In contrast, GBD  
71 reports only the health impacts of total PM<sub>2.5</sub> and does not distinguish mortality from fossil-fuel  
72 derived PM<sub>2.5</sub> and that from other kinds of PM<sub>2.5</sub>, including dust, wildfire smoke, and biogenically-  
73 sourced particles. We focus only on PM<sub>2.5</sub> since recent studies have provided mixed results on the  
74 link between ozone and mortality (Atkinson et al., 2016) and there does not exist a global coherent  
75 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 combustion  
78 than adults. This differential susceptibility to air pollution is due to their rapid growth, developing  
79 brain, and immature respiratory, detoxification, immune, and thermoregulatory systems (Bateson  
80 and Schwartz, 2008; Perera, 2018). Children also breathe more air per kilogram of body weight  
81 than adults, and are therefore more exposed to pollutants in air (WHO, 2006; Xu et al., 2012). The  
82 WHO estimated that in 2012, 169,000 global deaths among children under the age of 5 were  
83 attributable to ambient air pollution (WHO, 2016). Further estimation of the burden of mortality  
84 due to PM<sub>2.5</sub> (particularly from anthropogenic sources) among the young population would  
85 highlight the need for intervention aimed at reducing children's exposure.

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

92

93 **Materials and methods**

94 *Calculation of surface PM<sub>2.5</sub> concentrations*

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

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

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

### 131 *Population and Health data*

132 We used population data from the Center for International Earth Science Information  
133 Network (CIESIN) (CIESIN, 2018). The Gridded Population of the World, Version 4 Revision  
134 11 (GPWv4.11) is gridded with an output resolution of 30 arc-seconds (approximately 1 km at the  
135 equator). Since the population data are provided only at five-year intervals, we applied 2015  
136 population statistics to the results of our 2012 GEOS-Chem simulation. CIESIN population data

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

#### 143 *PM<sub>2.5</sub> mortality concentration–response model*

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

149 Global assessments of air pollution risk often use the Integrated Exposure-  
150 Response model (IER) (Burnett et al., 2014), which combined information on PM<sub>2.5</sub>–mortality  
151 associations from non-outdoor PM<sub>2.5</sub> sources, including secondhand smoke, household air  
152 pollution from use of solid fuels, and active smoking. The IER used data from active smoking and  
153 passive smoking to address the limited number of outdoor PM<sub>2.5</sub> epidemiologic studies at PM<sub>2.5</sub> >  
154 40 µg m<sup>-3</sup> available at the time. The IER formed the basis of the estimates of disease burden  
155 attributable to PM<sub>2.5</sub> (e.g., 4 million deaths in 2015 in GBD 2015). This function was then updated  
156 in 2018 using the Global Exposure Mortality Model (GEMM). In GEMM, data from 41  
157 epidemiological cohort studies were applied (Burnett et al., 2018). Independently conducted  
158 analyses were conducted on 15 of these cohorts to characterize the shapes of PM<sub>2.5</sub>–mortality  
159 associations in each cohort, using a specified functional form of the CRF. For the remaining 26

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

177

### 178 *Health impact calculations*

179 We estimated the number of premature deaths attributable to fossil fuel PM<sub>2.5</sub> using: (1)  
180 GEOS-Chem PM<sub>2.5</sub> estimated with all emission sources and GEOS-Chem PM<sub>2.5</sub> estimated without  
181 fossil fuel emissions, as a comparison against the first simulation, (2) total population above the  
182 age of 14 gridded to the GEOS-Chem grid resolution, (3) baseline all-cause mortality rates for



183 population above the age of 14 (per country or per state in the US and province in Canada), and  
 184 (4) the meta-analysis CRF (Vodonos et al., 2018). All health impacts were calculated on a per-grid  
 185 basis at the spatial resolution of the model. We applied the following health impact function to  
 186 estimate premature mortality related to exposure to fossil fuel PM<sub>2.5</sub> in each GEOS-Chem grid  
 187 cell:

188

$$189 \quad \sum \Delta y = y_0 * p * AF \tag{1}$$

$$190 \quad AF = \frac{\exp(\bar{\beta} * \Delta x) - 1}{\exp(\bar{\beta} * \Delta x)} \tag{2}$$

$$191 \quad \bar{\beta}(PM_{2.5}) = \int_{PM_{2.5} \text{ no fossil fuel}}^{PM_{2.5} \text{ all emissions}} \beta(PM_{2.5}) \tag{3}$$

192

193 where  $\Delta y$  is the change in the number of premature deaths due to exposure to fossil fuel PM<sub>2.5</sub>,  $y_0$   
 194 is the country/state/province specific baseline (all-cause) mortality rate,  $p$  is to the total population  
 195 above the age of 14,  $AF$  is the attributable fraction of deaths (the fraction of total deaths attributable  
 196 to PM<sub>2.5</sub> exposure),  $\bar{\beta}$  is the mean estimate for long-term PM<sub>2.5</sub> mortality concentration-response  
 197 over a range of concentrations from the penalized spline model in the recent meta-analysis, and  
 198  $\Delta x$  is the change in PM<sub>2.5</sub> concentration, calculated as the difference between GEOS-Chem PM<sub>2.5</sub>  
 199 with all emissions and GEOS-Chem PM<sub>2.5</sub> without fossil fuel emissions.

200

201 For each country, we summed the change in premature deaths ( $\Delta y$ ) in each grid cell over all grid  
 202 cells in that country. To estimate the change in deaths between the two scenarios (with and without  
 203 fossil fuel combustion), we computed the change in deaths in each grid cell, based on its

204 population, baseline rate, and exposure under the two scenarios (Equation (1)). The attributable  
205 fraction (AF), or proportion of deaths estimated as due to long-term exposure to PM<sub>2.5</sub> fossil fuel  
206 air pollution, was calculated using the concentration-response estimate, following the form shown  
207 in Equation (2) (Figure S5 in Supplemental material). Because these estimates of mortality  
208 concentration response ( $\beta$ ) are a nonlinear function of concentration, we used the penalized spline  
209 model predictions from this meta-analysis to integrate the concentration-specific  $\beta$  in each grid  
210 cell from the low PM<sub>2.5</sub> scenario (without fossil fuel emissions) to the high PM<sub>2.5</sub> scenario (with  
211 all emissions, including fossil fuel). In this way, we could calculate a mean value of  $\beta$  for each grid  
212 cell. There exist insufficient epidemiological data to calculate a robust health response function  
213 specific to fossil-fuel PM<sub>2.5</sub>. GEOS-Chem is a deterministic model. Therefore, our 95% confidence  
214 intervals (CI) for our estimates reflect only the 95% CI for the concentration response function.

### 215 *Secondary analysis among children <5 years old*

216 Lower respiratory infections (LRI), including pneumonia and bronchiolitis of bacterial and viral  
217 origin, are the largest single cause of mortality among young children worldwide and thus  
218 account for a significant global burden of disease worldwide (Nair et al., 2010). As mentioned  
219 previously, young children are more susceptible to the adverse effects of particulate air pollution  
220 than adults. Mehta et al. (2013) estimated the overall impact of PM<sub>2.5</sub> concentration with Relative  
221 Risk (RR) of 1.12 for LRI mortality per 10  $\mu\text{g m}^{-3}$  increase in annual average PM<sub>2.5</sub>  
222 concentration, as compared to RR of 1.04 for respiratory mortality among adults (Vodanos et al.,  
223 2018). We estimated the number of premature deaths attributable to PM<sub>2.5</sub> among children under  
224 the age of 5 years due to a range of LRI classifications (ICD-10, International Classification of  
225 Diseases codes: A48.1, A70, J09-J15.8, J16-J16.9, J20-J21.9, P23.0-P23.4). Baseline numbers of  
226 deaths due to LRI were obtained from the GBD for 2015 (IHME, 2017). We used the Relative

227 Risk (RR) of 1.12 (1.03-1.30) for LRI occurrence per  $10 \mu\text{g m}^{-3}$  increase in annual average  $\text{PM}_{2.5}$   
228 concentration (Mehta et al., 2013). Studies of longer-term exposure of  $\text{PM}_{2.5}$  and LRI in that  
229 meta-analysis were conducted in only a few developed countries with relatively low levels of  
230 annual mean  $\text{PM}_{2.5}$  ( $< 25 \mu\text{g m}^{-3}$ ), specifically the Netherlands, Czech Republic, Germany,  
231 Canada and USA. We therefore calculated the number of premature LRI deaths attributable to  
232  $\text{PM}_{2.5}$  only in North America, South America, and Europe.

233

## 234 **Results**

### 235 *Impact of fossil fuel use on $\text{PM}_{2.5}$*

236 Figure 1 shows the difference between global GEOS-Chem  $\text{PM}_{2.5}$  with and without fossil  
237 fuel emissions, plotted as the annual mean for 2012. Results show large contributions of 50-100  
238  $\mu\text{g m}^{-3}$  in  $\text{PM}_{2.5}$  over China and India, with smaller increments of 10-50  $\mu\text{g m}^{-3}$  over large swaths  
239 of the United States and Europe, industrialized countries in Africa (South Africa and Nigeria), and  
240 along the North African coastline due to European pollution.

### 241 *Global assessment of mortality attributable to $\text{PM}_{2.5}$*

242 Based on the annual  $\text{PM}_{2.5}$  simulation with and without global fossil fuel emissions, we  
243 estimated the excess deaths and attributable fraction (AF %) for the population above 14 years old.  
244 Figure 2 shows the simulated annual global premature mortality due to exposure to ambient  $\text{PM}_{2.5}$   
245 from fossil fuel emissions. Greatest mortality is simulated over regions with substantial influence  
246 of fossil-fuel related  $\text{PM}_{2.5}$ , notably parts of Eastern North America, western Europe, and South-  
247 East Asia.

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

267

#### 268 *Assessment of children (under the age of 5) LRI mortality attributable to PM<sub>2.5</sub>*

269 We estimated the number of premature deaths attributable to PM<sub>2.5</sub> among children under  
270 the age of 5 due to LRI only for those countries or regions with levels of annual PM<sub>2.5</sub>

271 concentrations below  $25 \mu\text{g m}^{-3}$ . These include North America, South America, and Europe. Based  
272 on the annual  $\text{PM}_{2.5}$  simulation with and without fossil fuel emissions, we calculated 876 excess  
273 deaths due to LRI in North and Central America, 747 in South America, and 605 in Europe (Table  
274 2). Using the GBD estimate of total deaths due to LRI (Institute for Health Metrics and Evaluation),  
275 we estimate that  $\text{PM}_{2.5}$  from fossil fuel combustion accounted on average for 7.2% of LRI mortality  
276 among children under the age of 5 in these regions, with the largest proportion of 13.6% in Europe  
277 (95% CI -0.4 to 25.3%) .

278

## 279 **Discussion**

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

288 Our estimate is for the year when fossil fuel emissions in China peaked and so predates  
289 large and dramatic reductions in fossil fuel emissions due to strict mitigation measures. These  
290 reductions led to a 30-50% decline in annual mean  $\text{PM}_{2.5}$  across the country from 2013 to 2018  
291 (Zhai et al., 2019). If we apply a 43.7% reduction in GEOS-Chem  $\text{PM}_{2.5}$  concentrations from the  
292 simulation with all emission sources, premature mortality in China decreases from 3.91 million to

293 2.36 million. India has recently imposed controls on pollution sources, but there is not yet evidence  
294 of air quality improvements in densely populated cities like Delhi (Vohra et al., 2020).  
295 Consideration of the 2012-2018 decrease in PM<sub>2.5</sub> exposure in China reduces the total global  
296 premature mortality due to fossil fuel PM<sub>2.5</sub> from 10.2 million premature deaths each year to 8.7  
297 (95% CI: -1.8 to 14.0) million.

298 In 2012, the population-weighted PM<sub>2.5</sub> is 72.8 µg m<sup>-3</sup> for China and 52.0 µg m<sup>-3</sup> for India  
299 from all sources and 9.9 µg m<sup>-3</sup> for China and 9.0 µg m<sup>-3</sup> for India without fossil fuel emissions.  
300 The low value of non-fossil fuel PM<sub>2.5</sub> is reasonable for southern India (Dey et al., 2012) but may  
301 be an underestimate in the Indo-Gangetic Plain where crop residue burning contributes to high  
302 levels of PM<sub>2.5</sub> (100-200 µg m<sup>-3</sup>) during the post-monsoon season (Ojha et al., 2020). An increase  
303 in the concentration of non-fossil-fuel PM<sub>2.5</sub> would decrease our estimate of the number of  
304 premature deaths due to fossil fuel PM<sub>2.5</sub> in India and China, as this would decrease the risk of  
305 premature mortality with a unit change in PM<sub>2.5</sub> (Figure S5).

306

#### 307 *Comparison with previous estimates of global mortality attributable to outdoor PM<sub>2.5</sub>*

308 Previous estimates of the GBD for 2015 suggest that exposure to total PM<sub>2.5</sub> causes 4.2  
309 million deaths (Cohen et al., 2017), whereas here we estimate more than double (10.2 million) the  
310 number of premature deaths from fossil fuel combustion alone in 2012. Differences between the  
311 current study and the 2015 GBD lower estimates are related mainly to the choice of the shape of  
312 the concentration-response function and the relative risk estimate. First, to provide information  
313 about exposure response at higher concentrations, the 2015 GBD study used the integrated  
314 exposure-response (IER) model in which active and second-hand smoking exposures were

315 converted to estimated annual PM<sub>2.5</sub> exposure equivalents using inhaled doses of particle mass  
316 (Burnett et al., 2014). Recent cohort studies from Asia indicate that this substantially  
317 underestimates the CRF at high concentrations. In contrast, in the current study we applied a CRF  
318 that was directly estimated from PM<sub>2.5</sub> studies alone, as described in a recent meta-analysis that  
319 included estimates from studies in countries like China with higher PM<sub>2.5</sub> concentrations than our  
320 included in previous derivations of CRFs (Vodonos et al., 2018). The CRF from this recent meta-  
321 analysis flattens out at higher concentrations, as does the IER curve. However, this flattening is  
322 not as great as in the IER, as Asian cohort studies at high PM<sub>2.5</sub> concentrations report larger effects  
323 than would be expected from the IER. Hence estimates of the global attributable fraction of deaths  
324 due to air pollution using the function from the recent meta-analysis are higher than the estimates  
325 using the IER function. In addition, at much lower concentrations ( $< 10 \mu\text{g m}^{-3}$ ), we applied higher  
326 slopes than assumed in the IER function. Recent studies at very low concentrations similarly show  
327 that the IER underestimated effects in this range (Pinault et al., 2016). Since GEOS-Chem  
328 estimated quite low concentrations in developed countries in Europe and North America, the  
329 number of premature deaths from PM<sub>2.5</sub> in these countries is greater than previous estimates.

330       Following an approach similar to the recent meta-analysis (Vodonos et al., 2018), Burnett  
331 et al. (2018) modeled the shape of the association between PM<sub>2.5</sub> and non-accidental mortality  
332 using data from 41 cohorts from 16 countries with GEMM. In that study, the uncertainty in a subset  
333 (15 cohorts) was characterized in the shape of the concentration-response parameter by calculating  
334 the Shape-Constrained Health Impact Function, a prespecified functional form. These estimated  
335 shapes varied across the cohorts included in the function. GEMM predicted 8.9 million (95% CI:  
336 7.5–10.3) deaths in 2015 attributable to long-term exposure to PM<sub>2.5</sub> from all sources; 120% higher  
337 excess deaths than previous estimates, but still lower than our estimate of mortality from exposure

338 to fossil-fuel derived PM<sub>2.5</sub> for 2012. Lelieveld et al. (2019) estimated the global and regional  
339 mortality burden of fossil fuel attributable PM<sub>2.5</sub> by applying the GEMM CRF to a global  
340 chemistry-climate model that is overall coarser (~1.9° latitude and longitude) than the model used  
341 in this work. The authors reported 3.61 million deaths per year attributable to pollution from fossil  
342 fuel combustion and 5.55 million deaths per year due to pollution from all anthropogenic sources.  
343 The estimated deaths from fossil fuel combustion are much lower than those in the current study  
344 for several reasons. First, the meta-analysis function used in our work includes 135 coefficients of  
345 all-cause mortality for adults aged 14-64 years old, together with cause-specific mortality and all-  
346 cause mortality among adults aged 65 and older, thus incorporating many more studies in a meta-  
347 regression framework than the 41 cohorts and coefficients in the GEMM function. Second, the  
348 approach used to estimate the CRF in Vodonos et al. (2018) allows for additional flexibility in the  
349 shape of the function because of its use of penalized splines. In contrast, the GEMM pooled CRF  
350 integrates a set of 26 log-linear functions and 15 functions characterized by three parameters  
351 governing the shape of the function. Third, while Cohen et al. (2017), Lelieveld et al. (2019) and  
352 Burnett et al. (2018) accounted for mortality from five specific causes (ischemic heart disease,  
353 stroke, chronic obstructive pulmonary disease, lung cancer and acute respiratory infections), in the  
354 current analysis we estimated changes in deaths from all causes. Fourth, some of the difference in  
355 the mortality estimates may come from differences in the age range. Our approach considers a  
356 wider population age range of over 14 years old (Vodonos et al., 2018) compared to the other  
357 studies, which considered a population age range of over 25 years (Burnett et al., 2018; Cohen et  
358 al., 2017; Lelieveld et al., 2019). Our approach has wider age range since the age range for the  
359 studies in the meta-analysis (Vodonos et al., 2018) included people younger than 25 years old  
360 (Hart et al., 2011; Pinault et al., 2016) . Finally, the finer spatial resolution that GEOS-Chem



361 utilizes over much of the globe improves co-location of PM hotspots and population centers,  
362 yielding higher estimates of excess mortality compared to Lelieveld et al. (2019).

363

#### 364 *Limitations*

365 There are a number of limitations that must be acknowledged. First, vulnerability to PM<sub>2.5</sub>  
366 exposure may vary by population characteristics such as ethnicity, socio-economic status (SES),  
367 risk behaviors such as smoking and underlying comorbidities (Krewski et al., 2000; Pope et al.,  
368 2004; Wang et al., 2017) and by different exposure characteristics. We were limited in our ability  
369 to undertake a comprehensive analysis of factors influencing the association between PM<sub>2.5</sub> and  
370 mortality since the global mortality data were not available by detailed age, ethnicity, SES,  
371 lifestyle, and underlying disease strata. In addition, the 95% CI of our estimates reflect the lower  
372 and upper bound of the CRF, which flattens out at higher concentrations. Regions with very high  
373 concentrations ( $>50 \mu\text{g m}^{-3}$ ) are beyond the data range in the meta-analysis; thus, the lower limit  
374 of the CI for those regions (China, West and North Africa; Table 1) are much less than zero.  
375 Second, for LRI in children, we have restricted our analysis to developed countries with annual  
376 PM<sub>2.5</sub>  $< 25 \mu\text{g m}^{-3}$ , in accordance with the geographical locations of the studies included in the  
377 meta-analysis by Mehta et al. (2013). Developing countries have much higher LRI mortality rates,  
378 and this restriction doubtless results in an underestimate. Finally, GEOS-Chem estimates of PM<sub>2.5</sub>  
379 concentrations almost certainly contains errors in estimates of emissions of pollution precursors,  
380 meteorological effects on air quality, and representation of the complex physical and chemical  
381 formation pathways. In the absence of systematic bias, such model error may not produce large

382 aggregate errors in the mortality burden of PM<sub>2.5</sub>, but bias may be present as well. In any event, it  
383 is challenging to estimate the true size of this error.

384

## 385 **Conclusions**

386 The effects of CO<sub>2</sub>-driven climate change on human health and welfare are complex, ranging from  
387 greater incidence of extreme weather events, more frequent storm-surge flooding, and increased  
388 risk of crop failure (Duffy et al., 2019). One consequence of increasing reliance on fossil fuel as  
389 an energy source that has thus far received comparatively little attention is the potential health  
390 impact of the pollutants co-emitted with the greenhouse gas CO<sub>2</sub>. Such pollutants include PM<sub>2.5</sub>  
391 and the gas-phase precursors of PM<sub>2.5</sub>. This study demonstrates that the fossil fuel component of  
392 PM<sub>2.5</sub> contributes a large global mortality burden. By quantifying this sometimes overlooked health  
393 consequence of fossil fuel combustion, a clear message is sent to policymakers and stakeholders  
394 of the co-benefits of a transition to alternative energy sources.

## 395 **Acknowledgments**

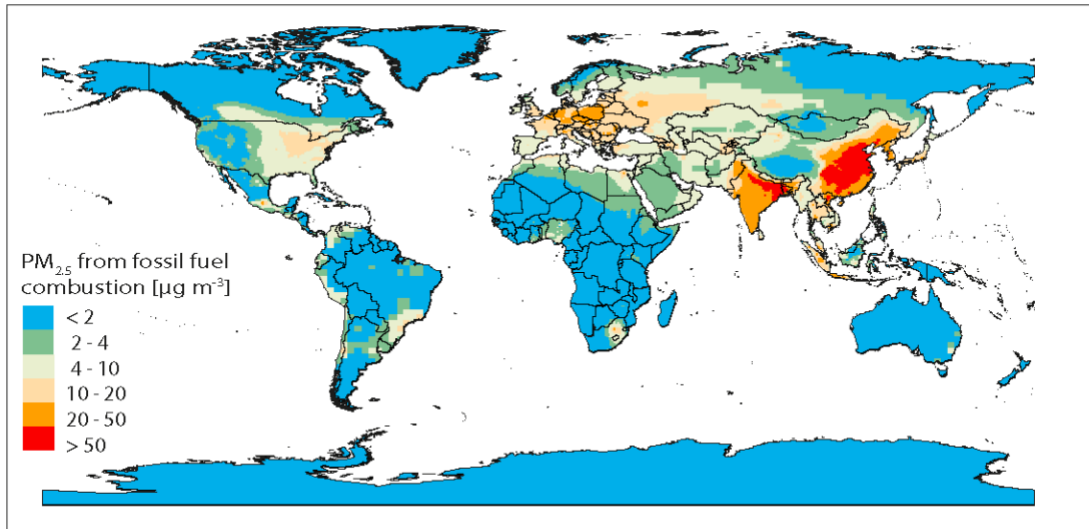
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## 399 **Declaration of interests**

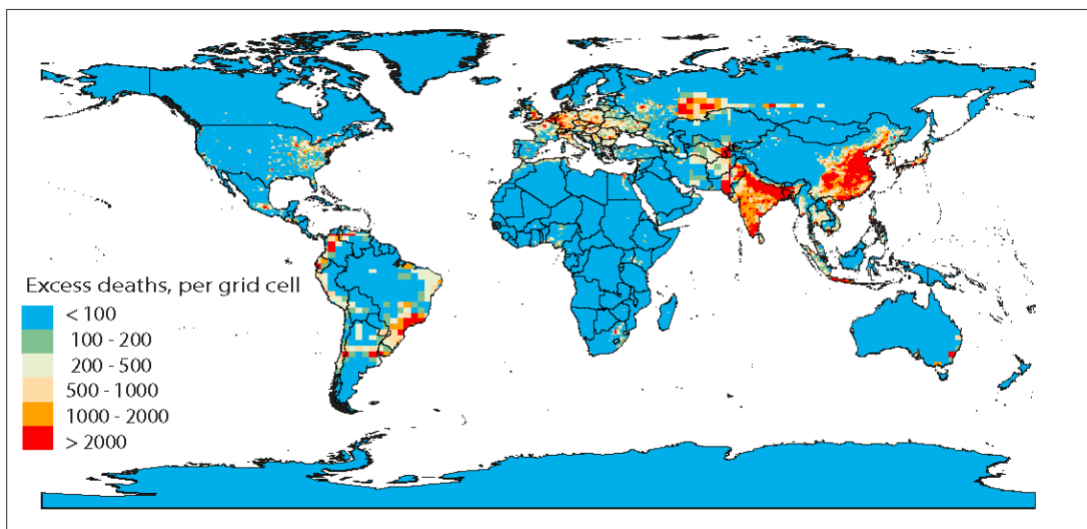
400 We declare no competing interests.

401 **Data availability.** GEOS-Chem code and output are available at the GEOS-Chem website  
402 ([http://acmg.seas.harvard.edu/geos\\_chem.html](http://acmg.seas.harvard.edu/geos_chem.html)) and upon request.

## Figures



**Figure 1: Contribution of fossil fuel combustion to surface PM<sub>2.5</sub>, as calculated by the chemical transport model GEOS-Chem.** The plot shows the difference in surface PM<sub>2.5</sub> concentrations from GEOS-Chem with and without fossil fuel emissions.



**Figure 2. Estimated annual excess deaths due to exposure to ambient PM<sub>2.5</sub> generated by fossil fuel combustion.**

**Table 1. Number of deaths attributable to exposure to fine particulate matter (PM<sub>2.5</sub>) generated by fossil fuel combustion for the population >14 years old**

| GEOS-Chem spatial grid resolution <sup>a</sup> | Region <sup>b</sup> |                                 | Total deaths >14 years old, in thousands | Population-weighted annual mean PM <sub>2.5</sub> concentration, µg m <sup>-3</sup> |                                       |   | Mean attributable fraction of deaths, % (95% CI) <sup>d</sup> | Deaths attributable to fossil-fuel related PM <sub>2.5</sub> , in thousands (95% CI) <sup>c</sup> | GEMM function deaths attributable to fossil-fuel related PM <sub>2.5</sub> , in thousands (95% CI) <sup>e</sup> |
|--|---------------------|---------------------------------|--|---|---------------------------------------|---|---|---|---|
|  |                     |                                 |  | PM <sub>2.5</sub> from all emission sources   | PM <sub>2.5</sub> without fossil fuel | Estimated PM <sub>2.5</sub> 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)   |

<sup>a</sup> Fine spatial scale is 0.5° × 0.67°, or about 50 km × 60 km. Coarse spatial scale is 2° × 2.5°, or about 200 km × 250 km

<sup>b</sup> List of countries for each region and subregion is provided in supplemental Table S2

<sup>c</sup> Annual number of deaths attributable to long-term exposure to PM<sub>2.5</sub> derived from fossil fuel combustion. CI is the confidence interval.

<sup>d</sup> Mean proportion of all deaths which can be attributed to long-term exposure to PM<sub>2.5</sub> generated by fossil fuel combustion, averaged over the country or region. CI; confidence interval.

<sup>e</sup> Attributable deaths calculated with the Global Exposure Mortality Model (GEMM) concentration-response function. <sup>44</sup>

**Table 2. Number of deaths due to lower respiratory infection (LRI) attributable to exposure to fine particulate matter (PM<sub>2.5</sub>) from fossil fuel combustion for the population <5 years old**

| <b>Region</b>                   | <b>Total deaths for children &lt;5 years old due to LRI</b> | <b>LRI deaths attributable to fossil-fuel PM<sub>2.5</sub> (95% CI)<sup>a</sup></b> | <b>Mean attributable fraction of deaths, % (95% CI)<sup>b</sup></b> |
|---------------------------------|---|---|---|
| <b>North America</b>            | <b>13,230</b>   | <b>876 (-26-1,657)</b>  | <b>6.6 (-0.2-12.5)</b>  |
| 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)  |
| <b>South America</b>            | <b>13,231</b>   | <b>747 (-21-1,443)</b>  | <b>5.7 (-0.2-10.9)</b>  |
| <b>Europe</b>                   | <b>4,446</b>  | <b>605 (-18-1,126)</b>  | <b>13.6 (-0.4-25.3)</b>   |

<sup>a</sup> Annual number of deaths attributed to long-term exposure to PM<sub>2.5</sub> derived from fossil fuel combustion.

<sup>b</sup> Mean proportion of deaths due to long-term exposure to PM<sub>2.5</sub> generated by fossil fuel combustion. CI is the confidence interval.

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