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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.

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

3  
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16  
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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<sub>2.5</sub>), and a key contributor to the global burden of mortality and  
22 disease. Previous risk assessments have examined the health response to total PM<sub>2.5</sub>, not just  
23 PM<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> exposure. We used the chemical transport model GEOS-Chem to estimate  
30 global exposure levels to fossil-fuel related PM<sub>2.5</sub> in 2012. Relative risks of mortality were  
31 modeled using functions that link long-term exposure to PM<sub>2.5</sub> 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<sub>2.5</sub>. The  
34 greatest mortality impact is estimated over regions with substantial fossil fuel related PM<sub>2.5</sub>,  
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<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> 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  $\mu\text{m}$ ) 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<sub>2.5</sub> 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<sub>2.5</sub> (Shaddick et al., 2018). However, the goal of such studies  
99 was to quantify the health response to PM<sub>2.5</sub> 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<sub>2.5</sub>  
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<sub>2.5</sub>. 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  $250 \text{ km}$ ). 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<sub>2.5</sub> 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<sub>2.5</sub>–mortality  
152 associations from non-outdoor PM<sub>2.5</sub> 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<sub>2.5</sub> epidemiologic studies at  
155 PM<sub>2.5</sub> > 40 µg m<sup>-3</sup> available at the time. The IER formed the basis of the estimates of disease  
156 burden attributable to PM<sub>2.5</sub> (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<sub>2.5</sub>–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<sub>2.5</sub> 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  $\Delta y$  is the change in the number of premature deaths due to exposure to fossil fuel PM<sub>2.5</sub>,  
 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<sub>2.5</sub> exposure),  $\bar{\beta}$  is the mean estimate for long-term PM<sub>2.5</sub> mortality  
 199 concentration-response over a range of concentrations from the penalized spline model in the  
 200 recent meta-analysis, and  $\Delta x$  is the change in PM<sub>2.5</sub> concentration, calculated as the difference  
 201 between GEOS-Chem PM<sub>2.5</sub> with all emissions and GEOS-Chem PM<sub>2.5</sub> without fossil fuel  
 202 emissions.

203

204 For each country, we summed the change in premature deaths ( $\Delta y$ ) 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 ( $\beta$ ) are a nonlinear function of concentration, we used the  
212 penalized spline model predictions from this meta-analysis to integrate the concentration-specific  
213  $\beta$  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  $\beta$  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  $10 \mu\text{g m}^{-3}$  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  $PM_{2.5}$  ( $< 25 \mu\text{g m}^{-3}$ ), 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  $\mu\text{g m}^{-3}$  in  $PM_{2.5}$  over China and India, with smaller increments of 10-50  $\mu\text{g m}^{-3}$  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<sub>2.5</sub>*

273 We estimated the number of premature deaths attributable to PM<sub>2.5</sub> among children under  
274 the age of 5 due to LRI only for those countries or regions with levels of annual PM<sub>2.5</sub>  
275 concentrations below 25 µg m<sup>-3</sup>. These include North America, South America, and Europe.  
276 Based on the annual PM<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> air pollution from fossil fuel combustion. Using the updated concentration  
286 response relationship between relative mortality and airborne PM<sub>2.5</sub>, 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<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> studies alone, as described in a recent meta-analysis  
323 that included estimates from studies in countries like China with higher PM<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> for 2012. Lelieveld et al. (2019) estimated the global  
344 and regional mortality burden of fossil fuel attributable PM<sub>2.5</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> 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.

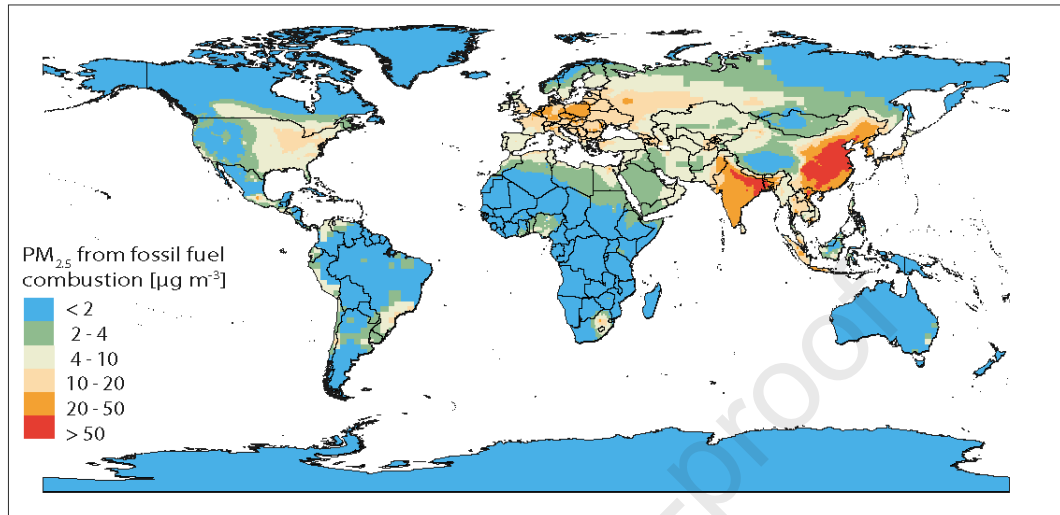
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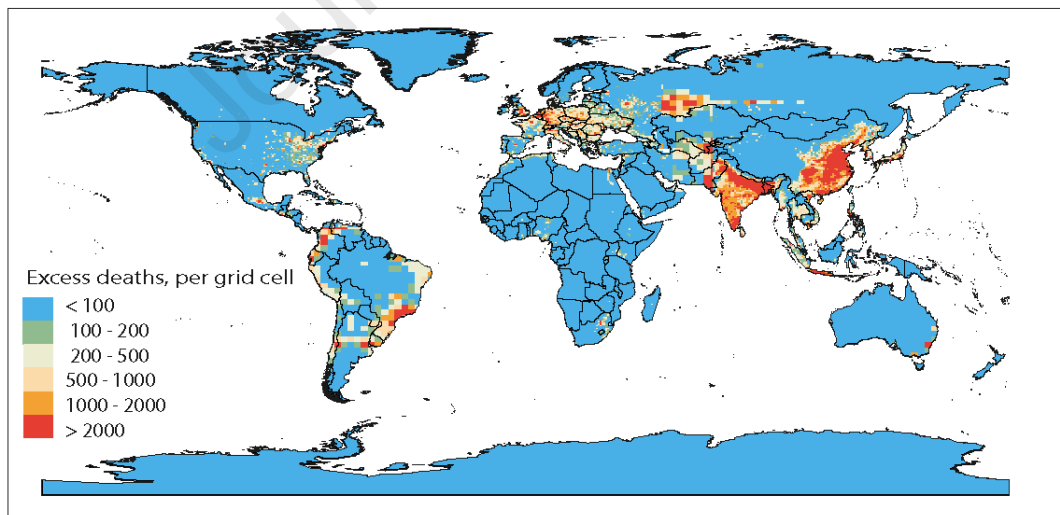
405   **Declaration of interests**

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](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.**

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**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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## Highlights

- Fossil fuel combustion emits particulate matter (PM<sub>2.5</sub>) 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<sub>2.5</sub> 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:

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