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

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Abstract

The burning of fossil fuels – especially coal, petrol, and diesel – is a major source of airborne fine particulate matter (PM$_{2.5}$), and a key contributor to the global burden of mortality and disease. Previous risk assessments have examined the health response to total PM$_{2.5}$, not just PM$_{2.5}$ from fossil fuel combustion, and have used a concentration-response function with limited support from
the literature and data at both high and low concentrations. This assessment examines mortality associated with PM$_{2.5}$ from only fossil fuel combustion, making use of a recent meta-analysis of newer studies with a wider range of exposure. We also estimated mortality due to lower respiratory infections (LRI) among children under the age of five in the Americas and Europe, regions for which we have reliable data on the relative risk of this health outcome from PM$_{2.5}$ exposure. We used the chemical transport model GEOS-Chem to estimate global exposure levels to fossil-fuel related PM$_{2.5}$ in 2012. Relative risks of mortality were modeled using functions that link long-term exposure to PM$_{2.5}$ and mortality, incorporating nonlinearity in the concentration response. We estimate a global total of 10.2 (95% CI: -47.1 to 17.0) million premature deaths annually attributable to the fossil-fuel component of PM$_{2.5}$. The greatest mortality impact is estimated over regions with substantial fossil fuel related PM$_{2.5}$, notably China (3.9 million), India (2.5 million) and parts of eastern US, Europe and Southeast Asia. The estimate for China predates substantial decline in fossil fuel emissions and decreases to 2.4 million premature deaths due to 43.7% reduction in fossil fuel PM$_{2.5}$ from 2012 to 2018 bringing the global total to 8.7 (95% CI: -1.8 to 14.0) million premature deaths. We also estimated excess annual deaths due to LRI in children (0-4 years old) of 876 in North America, 747 in South America, and 605 in Europe. This study demonstrates that the fossil fuel component of PM$_{2.5}$ contributes a large mortality burden. The steeper concentration-response function slope at lower concentrations leads to larger estimates than previously found in Europe and North America, and the slower drop-off in slope at higher concentrations results in larger estimates in Asia. Fossil fuel combustion can be more readily controlled than other sources and precursors of PM$_{2.5}$ such as dust or wildfire smoke, so this is a clear message to policymakers and stakeholders to further incentivize a shift to clean sources of energy.
Introduction

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

A series of newer studies conducted at lower concentrations and at higher concentrations have reported higher slopes than incorporated into the GBD using the integrated exposure–response (IER) curve (Burnett et al., 2014). These studies examined mortality due to exposure to PM$_{2.5}$ at concentrations below 10 μg m⁻³ in North America (Di et al., 2017; Pinault et al., 2016) and above 40 μg m⁻³ in Asia (Katanoda et al., 2011; Tseng et al., 2015; Ueda et al., 2012; Wong et al., 2015; 2016; Yin et al., 2017). Here we have used a concentration-response curve from a recently published meta-analysis of long-term PM$_{2.5}$ mortality association among adult populations which incorporates those new findings at high and low PM$_{2.5}$ concentrations (Vodonos et al.,
We also focus our study on the health impacts of fossil-fuel derived PM$_{2.5}$. In contrast, GBD reports only the health impacts of total PM$_{2.5}$ and does not distinguish mortality from fossil-fuel derived PM$_{2.5}$ and that from other kinds of PM$_{2.5}$, including dust, wildfire smoke, and biogenically-sourced particles. We focus only on PM$_{2.5}$ since recent studies have provided mixed results on the link between ozone and mortality (Atkinson et al., 2016) and there does not exist a global coherent concentration-response function (CRF) for ozone.

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

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

Calculation of surface PM\(_{2.5}\) concentrations

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

In this analysis we used GEOS-Chem with fossil fuel emissions from multiple sectors (power generation, industry, ships, aircraft, ground transportation, backup generators, kerosene, oil/gas extraction), detailed oxidant-aerosol chemistry, and reanalysis meteorology from the NASA Global Modeling and Assimilation Office. Fossil fuel emissions are from regional inventories where these are available for the US, Europe, Asia, and Africa, and from global
inventories everywhere else (such as Mexico, Australia, South America and Canada). More details of the specific fossil fuel inventories used in GEOS-Chem are in Table S1. Global-scale simulations in GEOS-Chem were carried out on a coarse spatial grid (2° × 2.5°, about 200 km × 250 km). Four regional simulations were also performed at fine spatial scale (0.5° × 0.67°, about 50 km × 60 km) for North America, Europe, Asia, and Africa using boundary conditions from the global model. The regional simulations allow for a better match with the spatial distribution of population, thus enhancing the accuracy of the estimates of health impacts. All simulations were set up to replicate 2012 pollution conditions. As described in the Supplemental Material, we find that globally, GEOS-Chem captures observed annual mean PM$_{2.5}$ concentrations with a spatial correlation of 0.70 and mean absolute error of 3.4 µg m$^{-3}$, values which compare well with those from other models (Shindell et al., 2018; Xing et al., 2015). We performed two sets of simulations: one set with fossil fuel emissions turned on and the other with such emissions turned off. We then assumed that the difference between the two sets of simulations represents the contribution of fossil fuel combustion to surface PM$_{2.5}$. More information on our choice of GEOS-Chem, the model setup, details of relevant anthropogenic emissions, and model validation is described in the Supplemental material.

Population and Health data

We used population data from the Center for International Earth Science Information Network (CIESIN) (CIESIN, 2018). The Gridded Population of the World, Version 4 Revision 11 (GPWv4.11) is gridded with an output resolution of 30 arc-seconds (approximately 1 km at the equator). Since the population data are provided only at five-year intervals, we applied 2015 population statistics to the results of our 2012 GEOS-Chem simulation. CIESIN population data
was then aggregated to the spatial scale of the model for the exposure estimates. Country/region level data on baseline mortality rates were from GBD data for 2015 (based on the 2017 iteration) (IHME, 2017). USA state-specific mortality rates were obtained from the CDC Wide-ranging Online Data for Epidemiologic Research (WONDER) compressed mortality files (WONDER). Canada death estimates by province were obtained from Statistics Canada, CANSIM (Canada, 2018).

PM$_{2.5}$ mortality concentration–response model

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

Global assessments of air pollution risk often use the Integrated Exposure-Response model (IER) (Burnett et al., 2014), which combined information on PM$_{2.5}$–mortality associations from non-outdoor PM$_{2.5}$ sources, including secondhand smoke, household air pollution from use of solid fuels, and active smoking. The IER used data from active smoking and passive smoking to address the limited number of outdoor PM$_{2.5}$ epidemiologic studies at PM$_{2.5} > 40$ µg m$^{-3}$ available at the time. The IER formed the basis of the estimates of disease burden attributable to PM$_{2.5}$ (e.g., 4 million deaths in 2015 in GBD 2015). This function was then updated in 2018 using the Global Exposure Mortality Model (GEMM). In GEMM, data from 41 epidemiological cohort studies were applied (Burnett et al., 2018). Independently conducted analyses were conducted on 15 of these cohorts to characterize the shapes of PM$_{2.5}$–mortality associations in each cohort, using a specified functional form of the CRF. For the remaining 26
cohort, the concentration-response was examined with a linear concentration hazard ratio model. A recent meta-analysis of the association between long-term PM$_{2.5}$ and mortality (Vodonos et al., 2018) applied techniques involving flexible penalized spline CRF in a multivariate random effects and meta-regression model. This approach allows the data to specify the shape of the CRF. The meta-regression pooled 135 estimates from 53 studies examining long-term PM$_{2.5}$ and mortality of cohorts aged 15 years and older. The estimate of the confidence intervals about the CRF includes a random variance component. This meta-analysis provided evidence of a nonlinear association between PM$_{2.5}$ exposure and mortality in which the exposure-mortality slopes decreases at higher concentrations (Figure S5 in Supplemental Material). We have chosen to use the dose-response function from the meta-analysis rather than the GEMM function as the meta-regression approach is more flexible and does not constrain the CRF to a specific functional form, it incorporates a random variance component in estimating the uncertainty around that curve, it is derived with more studies than previous approaches, and its estimates at high and low exposures are closer to the estimates in cohorts restricted to only very high and very low exposures. To ensure consistency with the concentration-response curve, premature mortality rates for the portion of the population >14 years of age were determined using the population and baseline mortality rates for different age groups from GBD data for 2015.

Health impact calculations

We estimated the number of premature deaths attributable to fossil fuel PM$_{2.5}$ using: (1) GEOS-Chem PM$_{2.5}$ estimated with all emission sources and GEOS-Chem PM$_{2.5}$ estimated without fossil fuel emissions, as a comparison against the first simulation, (2) total population above the age of 14 gridded to the GEOS-Chem grid resolution, (3) baseline all-cause mortality rates for
population above the age of 14 (per country or per state in the US and province in Canada), and (4) the meta-analysis CRF (Vodonos et al., 2018). All health impacts were calculated on a per-grid basis at the spatial resolution of the model. We applied the following health impact function to estimate premature mortality related to exposure to fossil fuel PM$_{2.5}$ in each GEOS-Chem grid cell:

\[
\sum \Delta y = y_o \ast p \ast AF
\]  

(1)

\[
AF = \frac{\exp(\bar{\beta}\Delta x)-1}{\exp(\bar{\beta}\Delta x)}
\]  

(2)

\[
\bar{\beta}(PM_{2.5}) = \int_{PM_{2.5} \text{ all emissions}}^{PM_{2.5} \text{ no fossil fuel}} \beta(PM_{2.5})
\]  

(3)

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

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

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

Lower respiratory infections (LRI), including pneumonia and bronchiolitis of bacterial and viral
origin, are the largest single cause of mortality among young children worldwide and thus
account for a significant global burden of disease worldwide (Nair et al., 2010). As mentioned
previously, young children are more susceptible to the adverse effects of particulate air pollution
than adults. Mehta et al. (2013) estimated the overall impact of PM$_{2.5}$ concentration with Relative
Risk (RR) of 1.12 for LRI mortality per 10 $\mu$g m$^{-3}$ increase in annual average PM$_{2.5}$
concentration, as compared to RR of 1.04 for respiratory mortality among adults (Vodonos et al.,
2018). We estimated the number of premature deaths attributable to PM$_{2.5}$ among children under
the age of 5 years due to a range of LRI classifications (ICD-10, International Classification of
Diseases codes: A48.1, A70, J09-J15.8, J16-J16.9, J20-J21.9, P23.0-P23.4). Baseline numbers of
deaths due to LRI were obtained from the GBD for 2015 (IHME, 2017). We used the Relative
Risk (RR) of 1.12 (1.03-1.30) for LRI occurrence per 10 µg m\(^{-3}\) increase in annual average PM\(_{2.5}\) concentration (Mehta et al., 2013). Studies of longer-term exposure of PM\(_{2.5}\) and LRI in that meta-analysis were conducted in only a few developed countries with relatively low levels of annual mean PM\(_{2.5}\) (< 25 µg m\(^{-3}\)), specifically the Netherlands, Czech Republic, Germany, Canada and USA. We therefore calculated the number of premature LRI deaths attributable to PM\(_{2.5}\) only in North America, South America, and Europe.

Results

Impact of fossil fuel use on PM\(_{2.5}\)

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

Global assessment of mortality attributable to PM\(_{2.5}\)

Based on the annual PM\(_{2.5}\) simulation with and without global fossil fuel emissions, we estimated the excess deaths and attributable fraction (AF %) for the population above 14 years old. Figure 2 shows the simulated annual global premature mortality due to exposure to ambient PM\(_{2.5}\) from fossil fuel emissions. Greatest mortality is simulated over regions with substantial influence of fossil-fuel related PM\(_{2.5}\), notably parts of Eastern North America, western Europe, and South-East Asia.
We estimated a total global annual burden premature mortality due to fossil fuel combustion in 2012 of 10.2 million (95% CI: -47.1 to 17.0 million). Table 1 reports the baseline number of deaths for people >14 years old, the annual PM$_{2.5}$ simulation with and without global fossil fuel emissions, the estimated excess deaths, and the attributable fraction for the populated continents. As shown in Table 1, we calculated 483,000 premature deaths in North America (95% CI: 284,000-670,000), 187,000 deaths in South America (95% CI: 107,000-263,000), 1,447,000 deaths in Europe (95% CI: 896,000-1,952,000), 7,916,000 deaths in Asia (95% CI: -48,106,000 to 13,622,000), and 194,000 deaths in Africa (95% CI: -237,000 to 457,000). The wide confidence intervals in Asia and Africa are due to the lack of data for areas where the exposure remains outside the range of the concentration response curve (PM$_{2.5}$ > 50 μg m$^{-3}$; Figure S5). The population-weighted pollution concentrations presented in Table 1 are higher than the average PM$_{2.5}$ concentrations for each country, since fossil-fuel PM$_{2.5}$ is mainly emitted in populous areas. The two countries with the highest premature mortality are China with 3.91 million and India with 2.46 million. Supplemental Table S2 provides extended data of the health impact calculations for each country. For comparison, Table 1 also reports the number of premature deaths attributable to fossil fuel PM$_{2.5}$ when the GEMM function is applied to the GEOS-Chem output. For most regions, the number of premature deaths calculated with GEMM is significantly lower than that calculated with the new function from Vodonos et al. (2018). Globally, the GEMM function yields 6.7 million deaths in 2012 due to fossil fuel combustion.

Assessment of children (under the age of 5) LRI mortality attributable to PM$_{2.5}$

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

**Discussion**

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

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

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

Comparison with previous estimates of global mortality attributable to outdoor PM$_{2.5}$

Previous estimates of the GBD for 2015 suggest that exposure to total PM$_{2.5}$ causes 4.2 million deaths (Cohen et al., 2017), whereas here we estimate more than double (10.2 million) the number of premature deaths from fossil fuel combustion alone in 2012. Differences between the current study and the 2015 GBD lower estimates are related mainly to the choice of the shape of the concentration-response function and the relative risk estimate. First, to provide information about exposure response at higher concentrations, the 2015 GBD study used the integrated exposure–response (IER) model in which active and second-hand smoking exposures were
converted to estimated annual PM$_{2.5}$ exposure equivalents using inhaled doses of particle mass (Burnett et al., 2014). Recent cohort studies from Asia indicate that this substantially underestimates the CRF at high concentrations. In contrast, in the current study we applied a CRF that was directly estimated from PM$_{2.5}$ studies alone, as described in a recent meta-analysis that included estimates from studies in countries like China with higher PM$_{2.5}$ concentrations than our included in previous derivations of CRFs (Vodonos et al., 2018). The CRF from this recent meta-analysis flattens out at higher concentrations, as does the IER curve. However, this flattening is not as great as in the IER, as Asian cohort studies at high PM$_{2.5}$ concentrations report larger effects than would be expected from the IER. Hence estimates of the global attributable fraction of deaths due to air pollution using the function from the recent meta-analysis are higher than the estimates using the IER function. In addition, at much lower concentrations (< 10 $\mu$g m$^{-3}$), we applied higher slopes than assumed in the IER function. Recent studies at very low concentrations similarly show that the IER underestimated effects in this range (Pinault et al., 2016). Since GEOS-Chem estimated quite low concentrations in developed countries in Europe and North America, the number of premature deaths from PM$_{2.5}$ in these countries is greater than previous estimates.

Following an approach similar to the recent meta-analysis (Vodonos et al., 2018), Burnett et al. (2018) modeled the shape of the association between PM$_{2.5}$ and non-accidental mortality using data from 41 cohorts from 16 countries with GEMM. In that study, the uncertainty in a subset (15 cohorts) was characterized in the shape of the concentration-response parameter by calculating the Shape-Constrained Health Impact Function, a prespecified functional form. These estimated shapes varied across the cohorts included in the function. GEMM predicted 8.9 million (95% CI: 7.5–10.3) deaths in 2015 attributable to long-term exposure to PM$_{2.5}$ from all sources; 120% higher excess deaths than previous estimates, but still lower than our estimate of mortality from exposure
to fossil-fuel derived PM$_{2.5}$ for 2012. Lelieveld et al. (2019) estimated the global and regional
mortality burden of fossil fuel attributable PM$_{2.5}$ by applying the GEMM CRF to a global
chemistry-climate model that is overall coarser (~1.9° latitude and longitude) than the model used
in this work. The authors reported 3.61 million deaths per year attributable to pollution from fossil
fuel combustion and 5.55 million deaths per year due to pollution from all anthropogenic sources.
The estimated deaths from fossil fuel combustion are much lower than those in the current study
for several reasons. First, the meta-analysis function used in our work includes 135 coefficients of
all-cause mortality for adults aged 14-64 years old, together with cause-specific mortality and all-
cause mortality among adults aged 65 and older, thus incorporating many more studies in a meta-
regression framework than the 41 cohorts and coefficients in the GEMM function. Second, the
approach used to estimate the CRF in Vodonos et al. (2018) allows for additional flexibility in the
shape of the function because of its use of penalized splines. In contrast, the GEMM pooled CRF
integrates a set of 26 log-linear functions and 15 functions characterized by three parameters
governing the shape of the function. Third, while Cohen et al. (2017), Lelieveld et al. (2019) and
Burnett et al. (2018) accounted for mortality from five specific causes (ischemic heart disease,
stroke, chronic obstructive pulmonary disease, lung cancer and acute respiratory infections), in the
current analysis we estimated changes in deaths from all causes. Fourth, some of the difference in
the mortality estimates may come from differences in the age range. Our approach considers a
wider population age range of over 14 years old (Vodonos et al., 2018) compared to the other
studies, which considered a population age range of over 25 years (Burnett et al., 2018; Cohen et
al., 2017; Lelieveld et al., 2019). Our approach has wider age range since the age range for the
studies in the meta-analysis (Vodonos et al., 2018) included people younger than 25 years old
(Hart et al., 2011; Pinault et al., 2016). Finally, the finer spatial resolution that GEOS-Chem
utilizes over much of the globe improves co-location of PM hotspots and population centers, yielding higher estimates of excess mortality compared to Lelieveld et al. (2019).

Limitations

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

Conclusions

The effects of CO$_2$-driven climate change on human health and welfare are complex, ranging from greater incidence of extreme weather events, more frequent storm-surge flooding, and increased risk of crop failure (Duffy et al., 2019). One consequence of increasing reliance on fossil fuel as an energy source that has thus far received comparatively little attention is the potential health impact of the pollutants co-emitted with the greenhouse gas CO$_2$. Such pollutants include PM$_{2.5}$ and the gas-phase precursors of PM$_{2.5}$. This study demonstrates that the fossil fuel component of PM$_{2.5}$ contributes a large global mortality burden. By quantifying this sometimes overlooked health consequence of fossil fuel combustion, a clear message is sent to policymakers and stakeholders of the co-benefits of a transition to alternative energy sources.

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Declaration of interests

We declare no competing interests.
Data availability. GEOS-Chem code and output are available at the GEOS-Chem website (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.
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

<table>
<thead>
<tr>
<th>GEOS-Chem spatial grid resolution$^a$</th>
<th>Region$^b$</th>
<th>Total deaths &gt;14 years old, in thousands</th>
<th>Population-weighted annual mean PM$_{2.5}$ concentration, $\mu g m^{-3}$</th>
<th>Mean attributable fraction of deaths, % (95% CI)$^d$</th>
<th>Deaths attributable to fossil-fuel related PM$_{2.5}$, in thousands (95% CI)$^e$</th>
<th>GEMM function deaths attributable to fossil-fuel related PM$_{2.5}$, in thousands (95% CI)$^e$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fine</td>
<td>North America</td>
<td>Central America &amp; the Caribbean</td>
<td>1,148</td>
<td>10.06</td>
<td>3.03</td>
<td>7.03 (69.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>USA</td>
<td>2,705</td>
<td>11.81</td>
<td>2.15</td>
<td>9.66 (81.8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Canada</td>
<td>250</td>
<td>12.01</td>
<td>1.76</td>
<td>10.25 (85.4)</td>
</tr>
<tr>
<td>Coarse</td>
<td>South America</td>
<td>2,389</td>
<td>8.66</td>
<td>3.02</td>
<td>5.65 (65.2)</td>
<td>7.8 (4.5-11.0)</td>
</tr>
<tr>
<td>Fine</td>
<td>Europe</td>
<td>8,626</td>
<td>19.22</td>
<td>4.68</td>
<td>14.54 (75.7)</td>
<td>16.8 (10.4-22.6)</td>
</tr>
<tr>
<td>Fine</td>
<td>Asia</td>
<td>Eastern Asia</td>
<td>25,468</td>
<td>51.72</td>
<td>8.68</td>
<td>43.05 (83.2)</td>
</tr>
<tr>
<td>Coarse</td>
<td>Western Asia &amp; the Middle East</td>
<td>1,456</td>
<td>26.95</td>
<td>20.73</td>
<td>6.22 (23.1)</td>
<td>6.5 (3.0-9.9)</td>
</tr>
<tr>
<td>Fine</td>
<td>Africa</td>
<td>5,274</td>
<td>32.98</td>
<td>28.98</td>
<td>4.00 (12.1)</td>
<td>3.7 (-4.5-8.7)</td>
</tr>
<tr>
<td>Coarse</td>
<td>Australia &amp; Oceania</td>
<td>189</td>
<td>4.17</td>
<td>2.19</td>
<td>1.98 (47.4)</td>
<td>3.2 (1.6-4.8)</td>
</tr>
<tr>
<td>Global</td>
<td></td>
<td></td>
<td>47,506</td>
<td>38.01</td>
<td>11.14</td>
<td>26.87 (70.7)</td>
</tr>
</tbody>
</table>

$^a$ 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

$^b$ 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

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

$^a$ 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


51. WONDER, C., Centers for Disease Control and Prevention Wide-ranging ONline Data for Epidemiologic Research https://wonder.cdc.gov/.


