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.,

2018). 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 $\mu g\ m^{-3}$ 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

cohorts, 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:

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

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

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

where Δy 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 Δx 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 (Δy) 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 (β) are a nonlinear function of concentration, we used the penalized spline model predictions from this meta-analysis to integrate the concentration-specific β 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 β 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 μg 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 μg 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

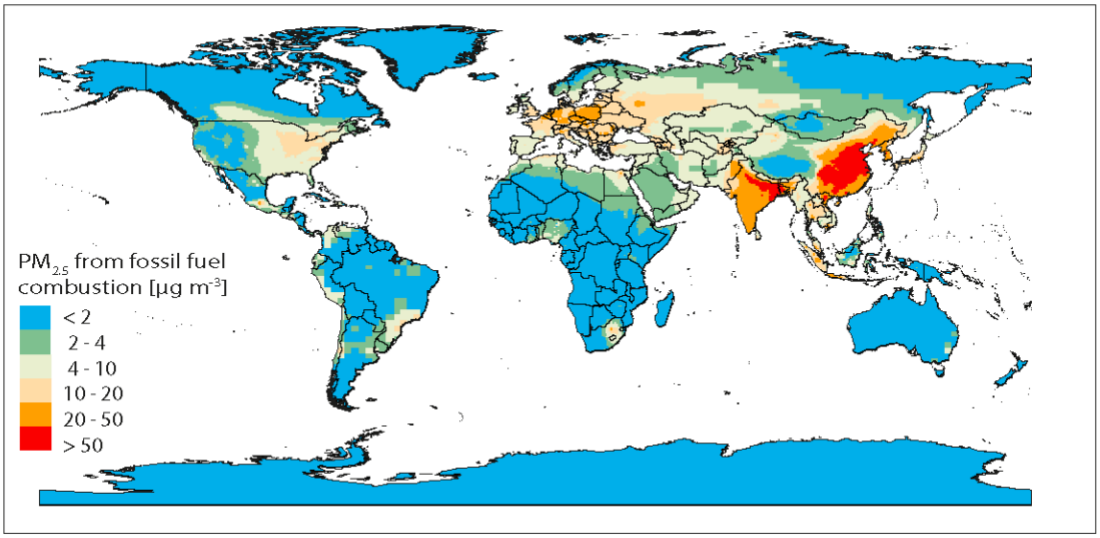

Figure 1: Contribution of fossil fuel combustion to surface $PM_{2.5}$, as calculated by the chemical transport model GEOS-Chem. The plot shows the difference in surface $PM_{2.5}$ concentrations from GEOS-Chem with and without fossil fuel emissions.

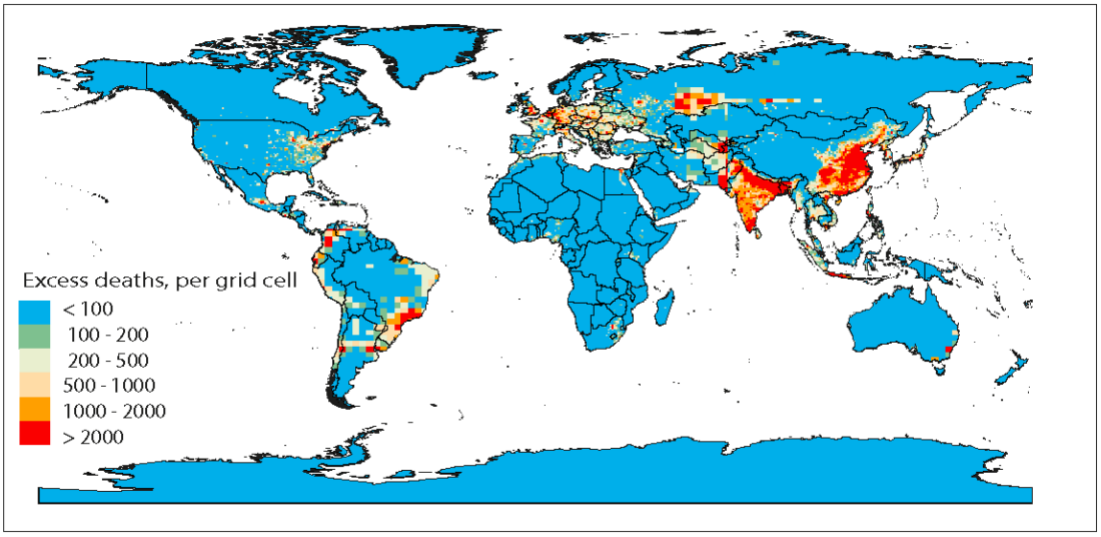

Figure 2. Estimated annual excess deaths due to exposure to ambient $PM_{2.5}$ generated by fossil fuel combustion.

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

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

[a] Fine spatial scale is 0.5° × 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

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

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

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

References

1. Apte, J. S., Marshall, J. D., Cohen, A. J., et al., Addressing Global Mortality from Ambient $PM_{2.5}$, Environ Sci Technol, 49, 8057-8066, doi:10.1021/acs.est.5b01236, 2015.

2. Atkinson, R. W., Butland, B. K., Dimitroulopoulou, C., et al., Long-term exposure to ambient ozone and mortality: a quantitative systematic review and meta-analysis of evidence from cohort studies, Bmj Open, 6, doi:10.1136/bmjopen-2015-009493, 2016.

3. Bateson, T. F., Schwartz, J., Children's response to air pollutants, J Toxicol Env Heal A, 71, 238-243, doi:10.1080/15287390701598234, 2008.

4. Brauer, M., Freedman, G., Frostad, J., et al., Ambient Air Pollution Exposure Estimation for the Global Burden of Disease 2013, Environ Sci Technol, 50, 79-88, doi:10.1021/acs.est.5b03709, 2016.

5. Brook, R. D., Rajagopalan, S., Pope, C. A., et al., Particulate Matter Air Pollution and Cardiovascular Disease An Update to the Scientific Statement From the American Heart Association, Circulation, 121, 2331-2378, doi:10.1161/CIR.0b013e3181dbece1, 2010.

6. Burnett, R., Chen, H., Szyszkowicz, M., et al., Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter, P Natl Acad Sci USA, 115, 9592-9597, doi:10.1073/pnas.1803222115, 2018.

7. Burnett, R., Pope, C. A., Ezzati, M., et al., An Integrated Risk Function for Estimating the Global Burden of Disease Attributable to Ambient Fine Particulate Matter Exposure, Environ Health Persp, 122, 397-403, doi:10.1289/ehp.1307049, 2014.

8. Canada, S., Government of Canada. https://www150.statcan.gc.ca/n1/en/type/data, 2018.

9. CIESIN, Center for International Earth Science Information Network - Columbia University; Gridded Population of the World, Version 4 (GPWv4): Population Count Adjusted to Match 2015 Revision of UN WPP Country Totals, Revision 11. NASA Socioeconomic Data and Applications Center (SEDAC), Palisades, NY, https://doi.org/10.7927/H4PN93PB, 2018.

10. Cohen, A. J., Brauer, M., Burnett, R., et al., Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015, Lancet, 389, 1907-1918, doi:10.1016/S0140-6736(17)30505-6, 2017.

11. Dedoussi, I. C., Barrett, S. R. H., Air pollution and early deaths in the United States. Part II: Attribution of $PM_{2.5}$ exposure to emissions species, time, location and sector, Atmos Environ, 99, 610-617, doi:10.1016/j.atmosenv.2014.10.033, 2014.

12. Dey, S., Di Girolamo, L., van Donkelaar, A., et al., Variability of outdoor fine particulate ($PM_{2.5}$) concentration in the Indian Subcontinent: A remote sensing approach, Remote Sens Environ, 127, 153-161, doi:10.1016/j.rse.2012.08.021, 2012.

13. Di, Q., Wang, Y., Zanobetti, A., et al., Air Pollution and Mortality in the Medicare Population, N Engl J Med, 376, 2513-2522, doi:10.1056/NEJMoa1702747, 2017.

14. Drury, E., Jacob, D. J., Spurr, R. J. D., et al., Synthesis of satellite (MODIS), aircraft (ICARTT), and surface (IMPROVE, EPA-AQS, AERONET) aerosol observations over eastern North America to improve MODIS aerosol retrievals and constrain surface aerosol concentrations and sources, J Geophys Res-Atmos, 115, doi:10.1029/2009jd012629, 2010.

15. Duffy, P. B., Field, C. B., Diffenbaugh, N. S., et al., Strengthened scientific support for the Endangerment Finding for atmospheric greenhouse gases, Science, 363, 597-+, doi:10.1126/science.aat5982, 2019.

16. Ford, B., Heald, C. L., Aerosol loading in the Southeastern United States: reconciling surface and satellite observations, Atmos Chem Phys, 13, 9269-9283, doi:10.5194/acp-13-9269-2013, 2013.

17. Forouzanfar, M. H., Afshin, A., Alexander, L. T., et al., Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015, Lancet, 388, 1659-1724, doi:10.1016/S0140-6736(16)31679-8, 2016.

18. Hart, J. E., Garshick, E., Dockery, D. W., et al., Long-Term Ambient Multipollutant Exposures and Mortality, Am J Resp Crit Care, 183, 73-78, doi:10.1164/rccm.200912-1903OC, 2011.

19. Heald, C. L., Collett, J. L., Lee, T., et al., Atmospheric ammonia and particulate inorganic nitrogen over the United States, Atmos Chem Phys, 12, 10295-10312, doi:10.5194/acp-12-10295-2012, 2012.

20. IHME, Institute for Health Metrics and Evaluation. http://ghdx.healthdata.org/gbd-results-tool, 2017.

21. Katanoda, K., Sobue, T., Satoh, H., et al., An Association Between Long-Term Exposure to Ambient Air Pollution and Mortality From Lung Cancer and Respiratory Diseases in Japan, J Epidemiol, 21, 132-143, doi:10.2188/jea.JE20100098, 2011.

22. Koplitz, S. N., Mickley, L. J., Marlier, M. E., et al., Public health impacts of the severe haze in Equatorial Asia in September-October 2015: demonstration of a new framework for informing fire management strategies to reduce downwind smoke exposure, Environ Res Lett, 11, doi:10.1088/1748-9326/11/9/094023, 2016.

23. Krewski, D., Burnett, R. T., Goldberg, M. S., et al., Special report reanalysis of the Harvard six cities study and the American Cancer Society Study of particulate air pollution and mortality part II: Sensitivity Analyses Appendix C. Flexible Modeling of the Effects of Fine Particles and Sulphate on Mortality, Health Effects Institute, https://www.healtheffects.org/system/files/SR-PartIIAppC.pdf, 2000.

24. Lacey, F. G., Marais, E. A., Henze, D. K., et al., Improving present day and future estimates of anthropogenic sectoral emissions and the resulting air quality impacts in Africa, Faraday Discuss, 200, 397-412, doi:10.1039/c7fd00011a, 2017.

25. Leibensperger, E. M., Mickley, L. J., Jacob, D. J., et al., Climatic effects of 1950-2050 changes in US anthropogenic aerosols - Part 1: Aerosol trends and radiative forcing, Atmos Chem Phys, 12, 3333-3348, doi:10.5194/acp-12-3333-2012, 2012.

26. Lelieveld, J., Klingmuller, K., Pozzer, A., et al., Effects of fossil fuel and total anthropogenic emission removal on public health and climate, P Natl Acad Sci USA, 116, 7192-7197, doi:10.1073/pnas.1819989116, 2019.

27. Lim, S. S., Vos, T., Flaxman, A. D., et al., A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010 (vol 380, pg 2224, 2012), Lancet, 381, 628-628, <Go to ISI>://WOS:000315189300032, 2013.

28. Lin, J. T., van Donkelaar, A., Xin, J. Y., et al., Clear-sky aerosol optical depth over East China estimated from visibility measurements and chemical transport modeling, Atmos Environ, 95, 258-267, doi:10.1016/j.atmosenv.2014.06.044, 2014.

29. Marais, E. A., Jacob, D. J., Guenther, A., et al., Improved model of isoprene emissions in Africa using Ozone Monitoring Instrument (OMI) satellite observations of formaldehyde: implications for oxidants and particulate matter, Atmos Chem Phys, 14, 7693-7703, doi:10.5194/acp-14-7693-2014, 2014a.

30. Marais, E. A., Jacob, D. J., Jimenez, J. L., et al., Aqueous-phase mechanism for secondary organic aerosol formation from isoprene: application to the southeast United States and co-benefit of SO_2 emission controls, Atmos Chem Phys, 16, 1603-1618, doi:10.5194/acp-16-1603-2016, 2016.

31. Marais, E. A., Jacob, D. J., Wecht, K., et al., Anthropogenic emissions in Nigeria and implications for atmospheric ozone pollution: A view from space, Atmos Environ, 99, 32-40, doi:10.1016/j.atmosenv.2014.09.055, 2014b.

32. Marais, E. A., Silvern, R. F., Vodonos, A., et al., Air Quality and Health Impact of Future Fossil Fuel Use for Electricity Generation and Transport in Africa, Environ Sci Technol, 53, 13524-13534, doi:10.1021/acs.est.9b04958, 2019.

33. Marais, E. A., Wiedinmyer, C., Air Quality Impact of Diffuse and Inefficient Combustion Emissions in Africa (DICE-Africa), Environ Sci Technol, 50, 10739-10745, doi:10.1021/acs.est.6b02602, 2016.

34. Mehta, S., Shin, H., Burnett, R., et al., Ambient particulate air pollution and acute lower respiratory infections: a systematic review and implications for estimating the global burden of disease, Air Qual Atmos Hlth, 6, 69-83, doi:10.1007/s11869-011-0146-3, 2013.

35. Nair, H., Nokes, D. J., Gessner, B. D., et al., Global burden of acute lower respiratory infections due to respiratory syncytial virus in young children: a systematic review and meta-analysis, Lancet, 375, 1545-1555, doi:10.1016/S0140-6736(10)60206-1, 2010.

36. Ojha, N., Sharma, A., Kumar, M., et al., On the widespread enhancement in fine particulate matter across the Indo-Gangetic Plain towards winter, Sci Rep-Uk, 10, doi:10.1038/s41598-020-62710-8, 2020.

37. Perera, F., Pollution from Fossil-Fuel Combustion is the Leading Environmental Threat to Global Pediatric Health and Equity: Solutions Exist, Int J Env Res Pub He, 15, doi:10.3390/ijerph15010016, 2018.

38. Pinault, L., Tjepkema, M., Crouse, D. L., et al., Risk estimates of mortality attributed to low concentrations of ambient fine particulate matter in the Canadian community health survey cohort, Environ Health-Glob, 15, doi:10.1186/s12940-016-0111-6, 2016.

39. Pope, C. A., Burnett, R. T., Thurston, G. D., et al., Cardiovascular mortality and long-term exposure to particulate air pollution - Epidemiological evidence of general pathophysiological pathways of disease, Circulation, 109, 71-77, doi:10.1161/01.Cir.0000108927.80044.7f, 2004.

40. Protonotariou, A. P., Bossioli, E., Tombrou, M., et al., Air Pollution in Eastern Mediterranean: Nested-Grid GEOS-CHEM Model Results and Airborne Observations. Advances in Meteorology, Climatology and Atmospheric Physics. Springer Atmospheric Sciences, Springer, Berlin, Heidelberg, 2013, pp. 1203-1209.

41. Shaddick, G., Thomas, M. L., Green, A., et al., Data integration model for air quality: a hierarchical approach to the global estimation of exposures to ambient air pollution, J R Stat Soc C-Appl, 67, 231-253, doi:10.1111/rssc.12227, 2018.

42. Shindell, D., Faluvegi, G., Seltzer, K., et al., Quantified, localized health benefits of accelerated carbon dioxide emissions reductions, Nat Clim Change, 8, doi:10.1038/s41558-018-0108-y, 2018.

43. Tseng, E., Ho, W. C., Lin, M. H., et al., Chronic exposure to particulate matter and risk of cardiovascular mortality: cohort study from Taiwan, Bmc Public Health, 15, doi:10.1186/s12889-015-2272-6, 2015.

44. Ueda, K., Nagasawa, S., Nitta, H., et al., Exposure to Particulate Matter and Long-term Risk of Cardiovascular Mortality in Japan: NIPPON DATA80, J Atheroscler Thromb, 19, 246-254, doi:10.5551/jat.9506, 2012.

45. Veefkind, J. P., Boersma, K. F., Wang, J., et al., Global satellite analysis of the relation between aerosols and short-lived trace gases, Atmos Chem Phys, 11, 1255-1267, doi:10.5194/acp-11-1255-2011, 2011.

46. Vodonos, A., Abu Awad, Y., Schwartz, J., The concentration-response between long-term $PM_{2.5}$ exposure and mortality; A meta-regression approach, Environ Res, 166, 677-689, doi:10.1016/j.envres.2018.06.021, 2018.

47. Vohra, K., Marais, E. A., Suckra, S., et al., Long-term trends in air quality in major cities in the UK and India: A view from space, Atmospheric Chemistry and Physics Discussions, doi:10.5194/acp-2020-342, 2020.

48. Wang, Y., Shi, L. H., Lee, M., et al., Long-term Exposure to $PM_{2.5}$ and Mortality Among Older Adults in the Southeastern US, Epidemiology, 28, 207-214, doi:10.1097/Ede.0000000000000614, 2017.

49. WHO, World Health Organization; Principles for evaluating health risks in children associated with exposure to chemicals. https://apps.who.int/iris/handle/10665/43604, 2006.

50. WHO, World Health Organization; Ambient air pollution: A global assessment of exposure and burden of disease. https://www.who.int/phe/publications/air-pollution-global-assessment/en/, 2016.

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

52. Wong, C. M., Lai, H. K., Tsang, H., et al., Satellite-Based Estimates of Long-Term Exposure to Fine Particles and Association with Mortality in Elderly Hong Kong Residents, Environ Health Persp, 123, 1167-1172, doi:10.1289/ehp.1408264, 2015.

53. Wong, C. M., Tsang, H., Lai, H. K., et al., Cancer Mortality Risks from Long-term Exposure to Ambient Fine Particle, Cancer Epidem Biomar, 25, 839-845, doi:10.1158/1055-9965.Epi-15-0626, 2016.

54. Xing, J., Mathur, R., Pleim, J., et al., Can a coupled meteorology-chemistry model reproduce the historical trend in aerosol direct radiative effects over the Northern Hemisphere?, Atmos Chem Phys, 15, 9997-10018, doi:10.5194/acp-15-9997-2015, 2015.

55. Xu, Z. W., Sheffield, P. E., Hu, W. B., et al., Climate Change and Children's Health-A Call for Research on What Works to Protect Children, Int J Env Res Pub He, 9, 3298-3316, doi:10.3390/ijerph9093298, 2012.

56. Yin, P., Brauer, M., Cohen, A., et al., Long-term Fine Particulate Matter Exposure and Nonaccidental and Cause-specific Mortality in a Large National Cohort of Chinese Men, Environ Health Persp, 125, doi:10.1289/Ehp1673, 2017.

57. Zhai, S. X., Jacob, D. J., Wang, X., et al., Fine particulate matter ($PM_{2.5}$) trends in China, 2013-2018: separating contributions from anthropogenic emissions and meteorology, Atmos Chem Phys, 19, 11031-11041, doi:10.5194/acp-19-11031-2019, 2019.

58. Zhang, L., Jacob, D. J., Knipping, E. M., et al., Nitrogen deposition to the United States: distribution, sources, and processes, Atmos Chem Phys, 12, 4539-4554, doi:10.5194/acp-12-4539-2012, 2012.