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Global and regional trends in particulate air pollution and attributable health burden over the past 50 years

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Global and regional trends in particulate air pollution and attributable health burden over the past 50 years

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Supplementary material for this article is available [online](#)

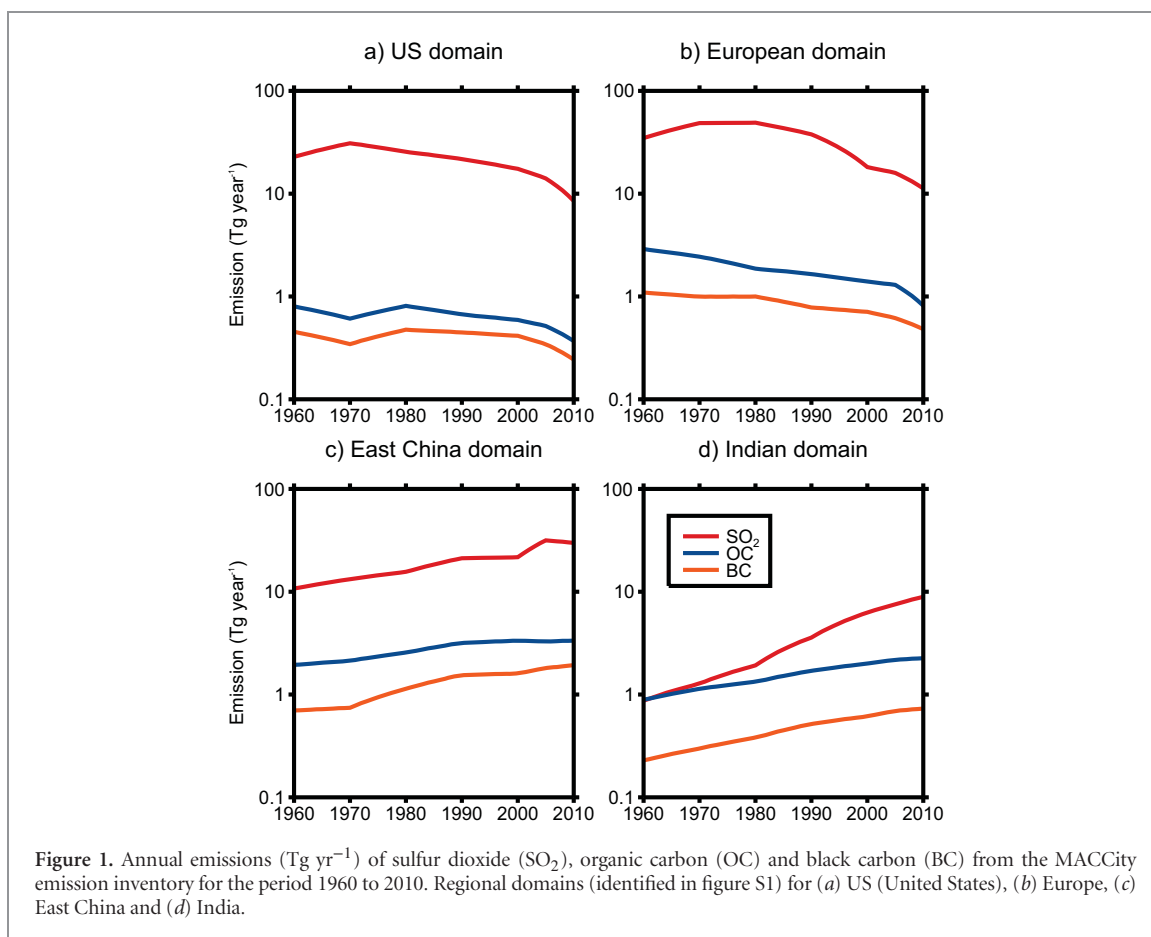
Abstract

Long-term exposure to ambient particulate matter (PM_{2.5}, mass of particles with an aerodynamic dry diameter of < 2.5 μm) is a major risk factor to the global burden of disease. Previous studies have focussed on present day or future health burdens attributed to ambient PM_{2.5}. Few studies have estimated changes in PM_{2.5} and attributable health burdens over the last few decades, a period where air quality has changed rapidly. Here we used the HadGEM3-UKCA coupled chemistry-climate model, integrated exposure-response relationships, demographic and background disease data to provide the first estimate of the changes in global and regional ambient PM_{2.5} concentrations and attributable health burdens over the period 1960 to 2009. Over this period, global mean population-weighted PM_{2.5} concentrations increased by 38%, dominated by increases in China and India. Global attributable deaths increased by 89% to 124% over the period 1960 to 2009, dominated by large increases in China and India. Population growth and ageing contributed mostly to the increases in attributable deaths in China and India, highlighting the importance of demographic trends. In contrast, decreasing PM_{2.5} concentrations and background disease dominated the reduction in attributable health burden in Europe and the United States. Our results shed light on how future projected trends in demographics and uncertainty in the exposure-response relationship may provide challenges for future air quality policy in Asia.

1. Introduction

Long-term exposure to ambient concentration of particles with an aerodynamic dry diameter of < 2.5 μm (PM_{2.5}) is associated with mortality and morbidity and shortens life expectancy (Dockery *et al* 1993, Pope and Dockery 2006, Pope *et al* 1995). It is estimated that ~87% of the global population live in areas exceeding the World Health Organisation's (WHO) air quality guidelines for annual mean ambient PM_{2.5} (10 μg m⁻³) (Apte *et al* 2015). Recent assessments of the Global Burden of Disease (GBD) estimate that exposure to ambient PM_{2.5} is a major contributing risk factor to regional and global burden of disease (Forouzanfar *et al* 2016, Forouzanfar *et al* 2015, Lim *et al* 2013).

Previous studies have reported present day and future (Lelieveld *et al* 2015, Silva *et al* 2016b) attributable health burdens assessments. Few studies have estimated changes in PM_{2.5} attributable health burdens over the last few decades (e.g. Wang *et al* 2017), a period where widespread implementation of air quality regulation and emission controls in North America and Europe coincided with extensive economic growth and limited emission controls across Asia. Over Europe and the United States, emissions of sulphur dioxide (SO₂) have decreased by more than 70% over the last few decades (Leibensperger *et al* 2012, Vestreng *et al* 2007), resulting in substantial reductions in PM_{2.5} concentrations (Leibensperger *et al* 2012, Tørseth *et al* 2012, Turnock *et al* 2015). In contrast, SO₂ emissions over Asia have increased by a



factor of 7 between 1960 and 2005 (Smith *et al* 2011), resulting in increased PM_{2.5} concentrations (Brauer *et al* 2015). Understanding these historical changes in PM_{2.5} concentrations and attributable burdens across these regions is vital to inform future air quality policy design.

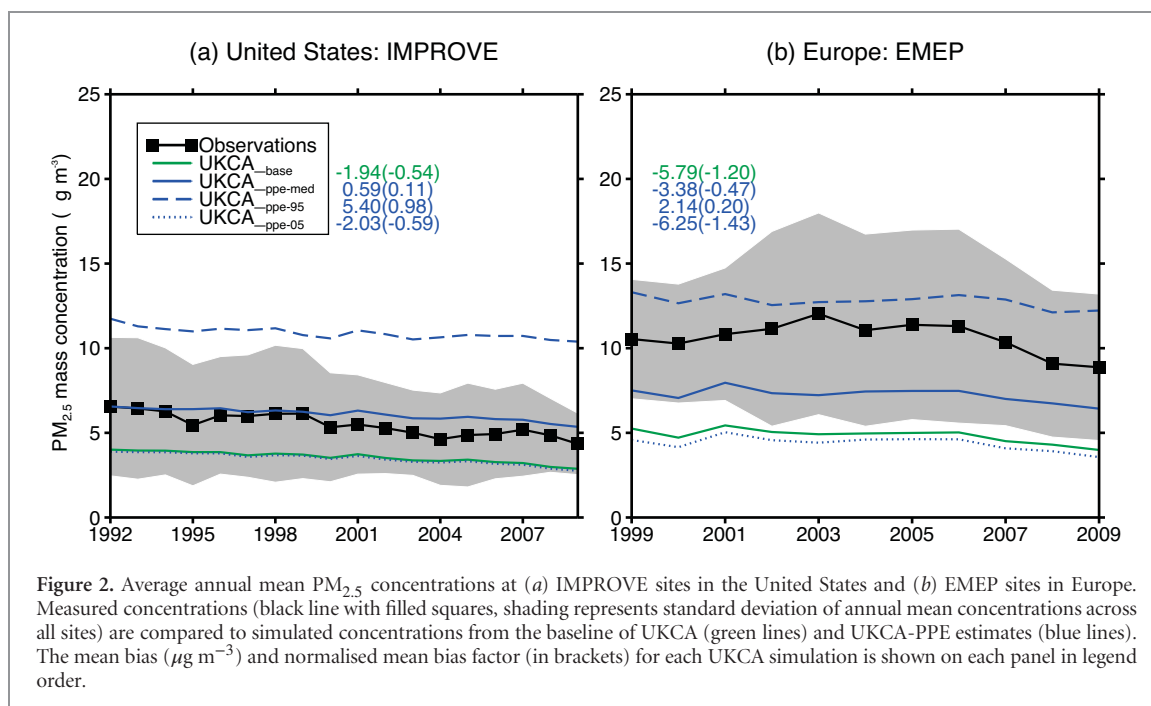
Estimating the health burden attributable to long-term exposure to PM_{2.5} requires an understanding of the exposure-response relationship, an accurate representation of PM_{2.5} concentrations and demographic and background disease trends. PM_{2.5} concentrations can be simulated using global chemical transport models (Anenberg *et al* 2010, Lelieveld *et al* 2013, Silva *et al* 2016a), or through a combination of modelling, satellite remote sensing data, ground-based observations, and land-use regression (Brauer *et al* 2012, Brauer *et al* 2015, Jerrett *et al* 2016, van Donkelaar *et al* 2010). Global health assessments (Forouzanfar *et al* 2016, Forouzanfar *et al* 2015, Lim *et al* 2013, Wang *et al* 2017) are restricted to the last few decades (1990 onwards), when satellite and ground-based observations are typically available. For this reason, little is known about how PM_{2.5} attributable burden changed prior to 1990. Here we combine a global climate model, with exposure-response relationships, demographic and background disease data to provide the first estimate of the changes in global and regional PM_{2.5} attributable health burdens over the period 1960 to 2009.

2. Methods

2.1. PM_{2.5} concentrations

We used the coupled chemistry–climate model HadGEM-3-UKCA, known hereafter as ‘UKCA’, to simulate PM_{2.5} concentrations for the period 1960 to 2009. We use the same model setup described in detail in Turnock *et al* (2015) with a horizontal resolution of 1.875° × 1.25° (approximately 140 km at mid latitudes). Meteorological fields were nudged at 6 h intervals to the European Centre for Medium-Range Weather Forecasts Reanalysis (ERA-40) (Uppala *et al* 2005) for the years 1960 to 2000 and ERA-Interim (Dee *et al* 2011) for 2001 to 2009.

UKCA simulates sulfate (SO₄), black carbon (BC), organic carbon (OC) and sea salt aerosol in five log-normal modes (four soluble modes and one insoluble Aitken mode) (Mann *et al* 2010) (see supplementary information (SI) 1.1 available at stacks.iop.org/ERL/12/104017/mmedia). Monthly mean anthropogenic emissions of CO, SO₂, NO_x, OC and BC from 1960 to 2009 are taken from the MACCity emission inventory (Granier *et al* 2011). Figure 1 shows the 1960 to 2010 trends in SO₂, BC and OC. Emissions in Europe and the United States (US) have declined from a maximum in the 1970s due to the implementation of air quality regulation and emission controls, while emissions have increased substantially in China and India. SO₂ emissions in the region of East China



in the 2000s were a similar magnitude to US emissions in the 1970s, but less than European emissions during the same period. Other emission sources are described in detail in Turnock *et al* (2015). Mineral dust concentrations are taken from a 10 year GLOMAP-mode climatology taken from Reddington *et al* (2015). We therefore assume no interannual variability or trends in dust over the study period. There is no representation of ammonium nitrate in this version of the model.

To account for uncertainty in $\text{PM}_{2.5}$ estimated by UKCA we used a perturbed parameter ensemble (PPE) of 235 UKCA simulations for the year 2008 where 26 aerosol related parameters were perturbed simultaneously. The PPE represents the aerosol parametric uncertainty in the model (see SI 1.3 and figure S2). We use the median value of $\text{PM}_{2.5}$ simulated across the PPE as our best estimate of $\text{PM}_{2.5}$ and the 5th and 95th percentile values as an indication of uncertainty in our estimate. We increment the baseline model (UKCA_{base}) by the absolute difference in the year 2008 between the baseline and the median (UKCA_{ppe-med}), 5th (UKCA_{ppe-05}) and 95th (UKCA_{ppe-95}) percentile of the PPE (see SI 1.3 and figure S3). We assume that the same increment across the entire 1960 to 2009 simulation period.

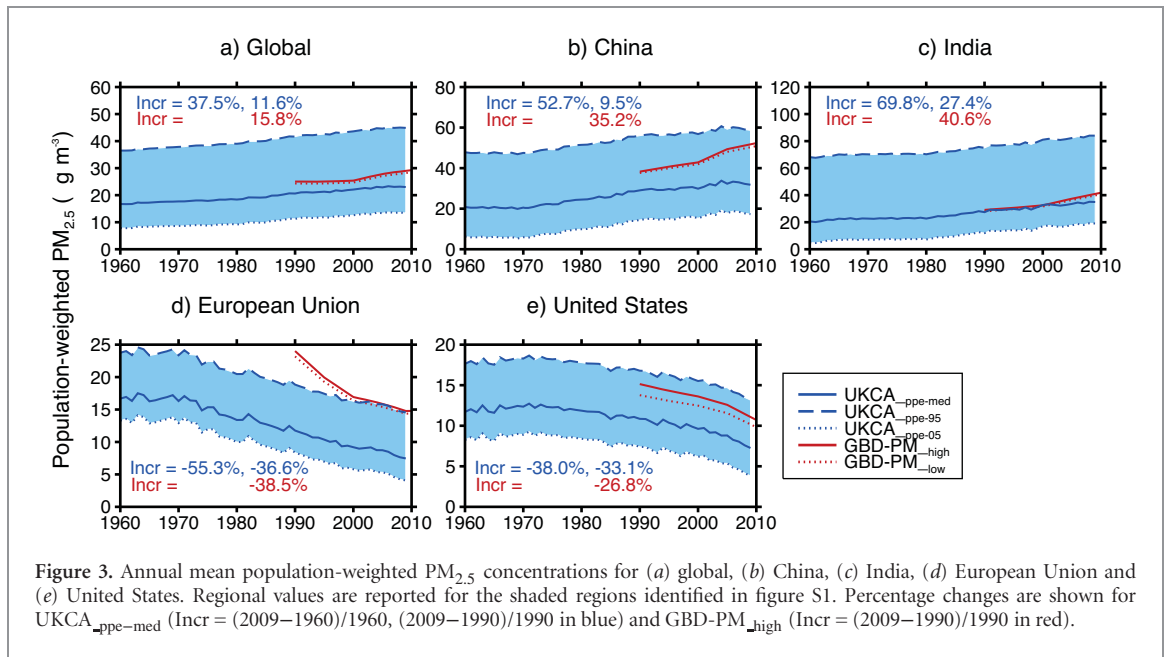
We also used satellite-derived $\text{PM}_{2.5}$ estimates (Brauer *et al* 2015) known hereafter as ‘GBD-PM’. This dataset provides annual mean $\text{PM}_{2.5}$ concentrations at $0.1^\circ \times 0.1^\circ$ horizontal resolution for the period 1990 to 2010 at five year intervals. In this dataset, $\text{PM}_{2.5}$ is estimated through a $\text{PM}_{2.5}$ to aerosol optical depth (AOD) relationship using the GEOS-Chem model and satellite remote sensing products calibrated to ground-based measurements (Brauer *et al* 2015). This dataset was used in the GBD2013 (Forouzanfar *et al* 2015) and we use it compare with UKCA.

Figure 2 compares simulated $\text{PM}_{2.5}$ against measurements at surface sites in the US and Europe (see figure S1). In the US we use observations from the Interagency Monitoring of Protected Visual Environments (IMPROVE) and in Europe we use observations from the European Monitoring and Evaluation Programme (EMEP) network (see SI 1.2). There are few long-term observations of $\text{PM}_{2.5}$ outside of Europe and the US with which to evaluate UKCA. The baseline UKCA model (UKCA_{base}) underestimates observed concentrations in both Europe (normalised mean bias factor (NMBF) = -1.2) and the US (NMBF = -0.54). Similarly, Turnock *et al* (2015) found UKCA underestimated total suspended particles and PM_{10} observed over Europe using the same model setup. We find that UKCA_{ppe-med} better matches observations in both Europe (NMBF = -0.47) and the US (NMBF = 0.11). The 5th to 95th percentile of the PPE brackets surface observations in both Europe and the US. We therefore report the results from these three simulations for the rest of the paper.

2.2. Background disease and demographic data

We used national level population and age group distribution data from the United Nations (UN) Population Division (UN 2015), which are available for the period 1960 to 2010 (see figure S3). We used gridded population from the Gridded Population of the World v3 (GPWv3) (CIESIN 2015), at a resolution of 2.5 arc-minutes for the period 1990 to 2010. We extrapolated the GPWv3 to 1960 applying the rate of change observed in the UN national level data (see SI 1.4).

We used age and cause-specific background disease data for the period 1980 to 2010 from the Institute for Health Metrics and Evaluation (IHME 2014). This



dataset provides national level background disease endpoint data for cardiovascular ischemic heart disease (IHD) and stroke (cerebrovascular disease), lung cancer (LungC), chronic obstructive pulmonary disease (COPD) and lower respiratory infections (LRI). Disease data is not available prior to 1980, so we take a conservative assumption and assume that background disease rates remain constant at 1980 levels. In a sensitivity study, we assume that background disease rates between 1960 and 1980 follow the same trend as that between the period 1980 to 1990 (see SI 1.4).

2.3. Attributable health burden calculation

We calculate relative risk (RR) due to long-term exposure to $PM_{2.5}$ using the integrated exposure-response (IER) relationship (Burnett *et al* 2014), which compiles epidemiological evidence across a wide range of $PM_{2.5}$ exposures from different combustion sources. The IER allows for age-dependent (i.e. ≥ 25 years of age at five year intervals to age 80+) calculation of RR for IHD and stroke, adult (≥ 25 years of age) for LC and COPD, and all ages for lower respiratory infections (LRI). We develop a lookup table compatible with the IER used in GBD2013 (Forouzanfar *et al* 2015) (see SI 1.5). This lookup table is provided in SI data 1. IER relationships are non-linear with respect to $PM_{2.5}$ exposure (figure S4), with reduced sensitivity of RR to $PM_{2.5}$ at higher concentrations (Pope *et al* 2009a, Pope *et al* 2011), particularly for IHD, stroke and LRI.

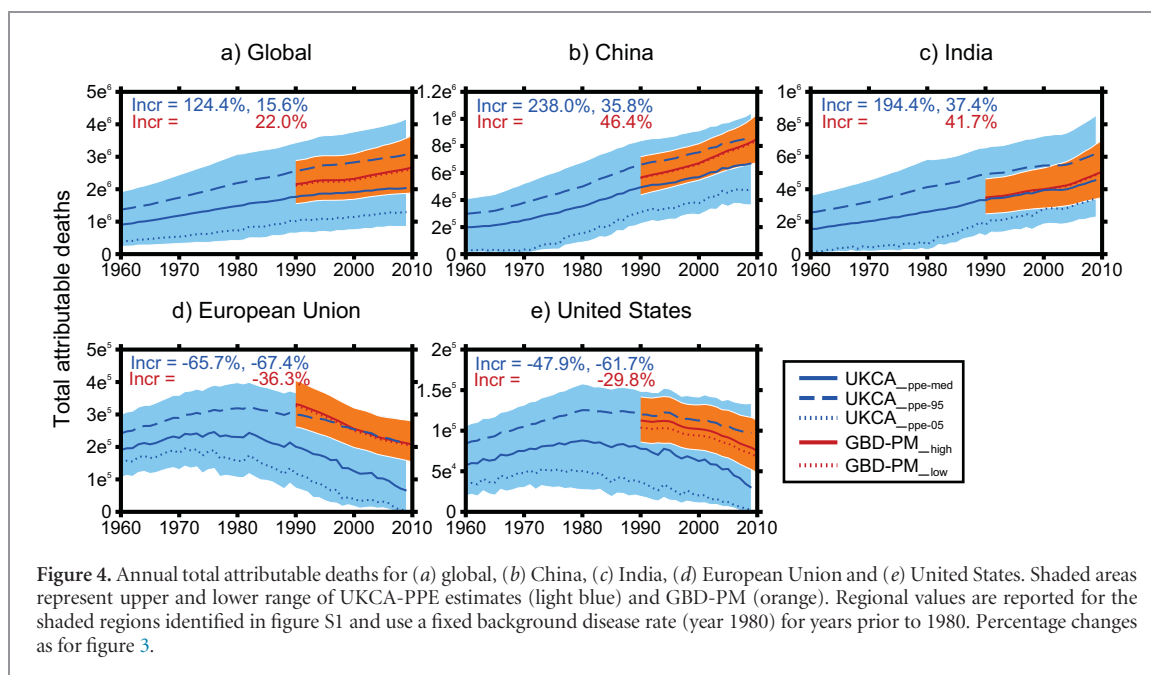
We use IER derived RRs to estimate attributable deaths at the grid cell level using attributable fraction type relationship described in Apte *et al* (2015) (see SI 1.5). Years of lost life (YLLs) are calculated by summing attributable deaths in each age group and multiplying by the associated expected life expectancy taken from the standard life table provide by Murray *et al* (2013). We estimate attributable deaths

using $PM_{2.5}$ concentrations from UKCA for the period 1960 to 2009 and from GBD-PM for the period 1990 to 2010. We calculated attributable deaths at original resolution of the GBD-PM data ($0.1^\circ \times 0.1^\circ$) (GBD-PM_{high}) and at the same resolution of UKCA ($1.875^\circ \times 1.25^\circ$) (GBD-PM_{low}). We find that attributable deaths estimated using GBD-PM closely match GBD2013, within 3%–4% globally, with similar regional mortalities (Forouzanfar *et al* 2015). We also explored the relative contribution of estimated attributable deaths over the period 1980 to 2009 to changing $PM_{2.5}$ concentrations, population demographics and background disease (see SI 1.6).

3. Results and discussion

Figure 3 shows annual mean population-weighted $PM_{2.5}$ concentrations over the period 1960 to 2009. Population-weighted $PM_{2.5}$ concentrations simulated by the median PPE (UKCA_{ppe-med}) closely match those from GBD-PM over India but are lower in other regions. We explored whether the coarser spatial resolution of UKCA ($1.875^\circ \times 1.25^\circ$) compared to GBD-PM_{high} ($0.1^\circ \times 0.1^\circ$) is responsible for this difference. When GBD-PM_{high} (Brauer *et al* 2015) is averaged to the same spatial resolution as UKCA (GBD-PM_{low}), mean population-weighted $PM_{2.5}$ typically decreased by less than $\sim 4\%$ ($\sim 1 \mu\text{g m}^{-3}$), showing that lower $PM_{2.5}$ concentrations simulated by UKCA_{ppe-med} is not entirely due to the coarse resolution of UKCA. The upper (UKCA_{ppe95}) and lower (UKCA_{ppe05}) range of UKCA bracket values from GBD-PM, except over Europe.

During the period 1990 to 2009, global population-weighted $PM_{2.5}$ concentrations simulated by UKCA_{ppe-med} increased by 11.6%, smaller than the 15.8% increase estimated by GBD-PM. At the



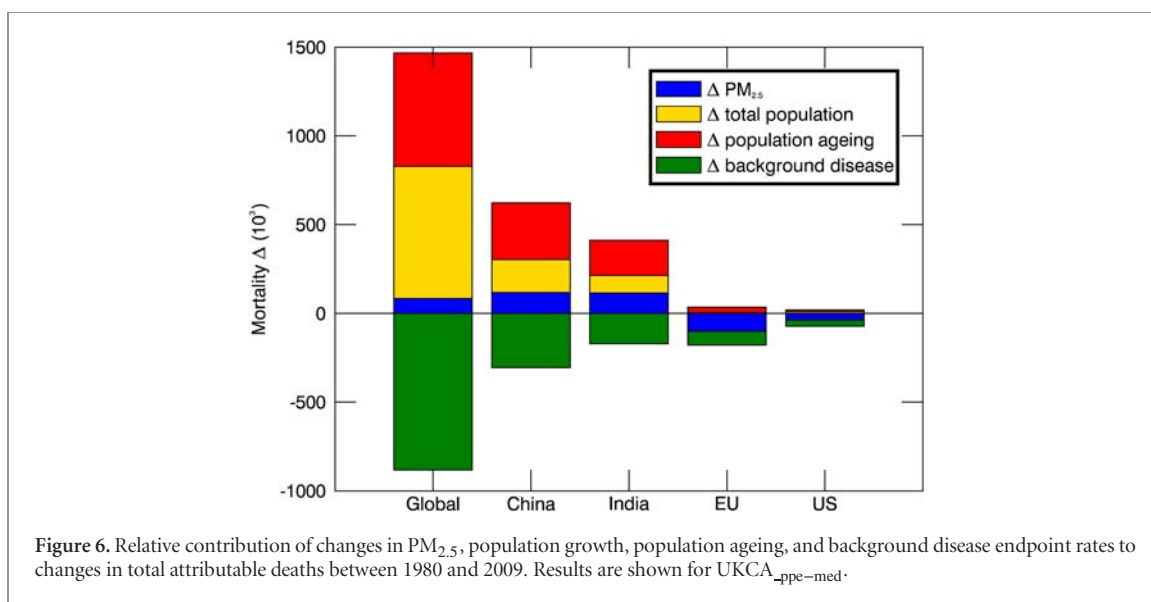
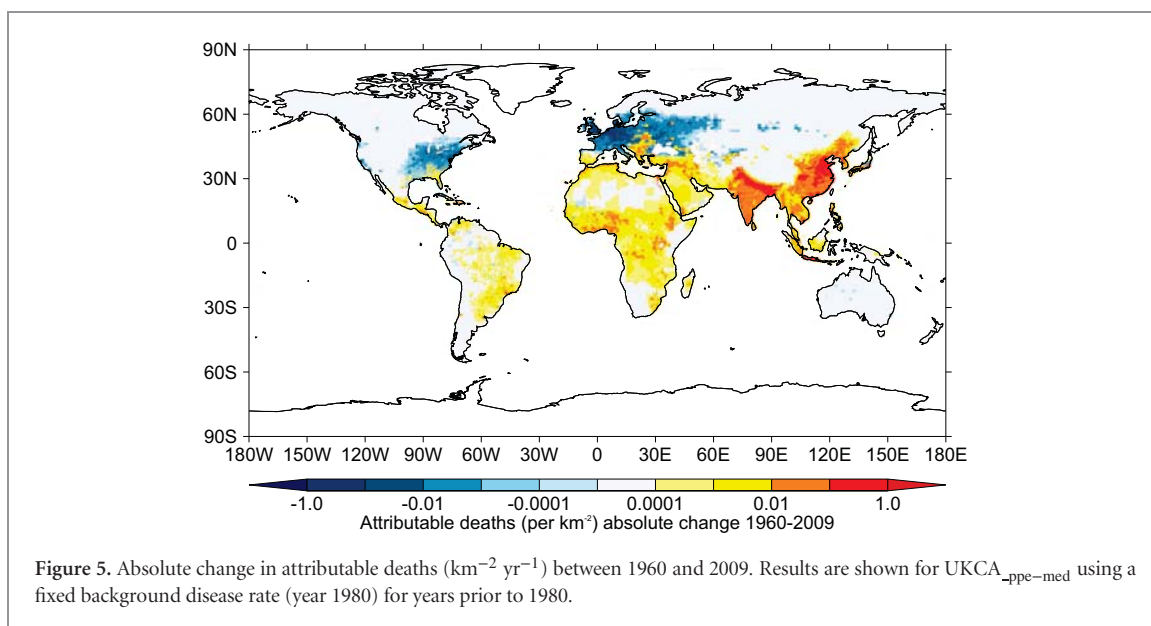
regional scale, UKCA_{ppe-med} simulated broadly similar fractional changes to GBD-PM in the EU and US, but smaller changes in China and India. During the period 1960 to 2009, global population-weighted PM_{2.5} simulated by UKCA increased by 37.5%, dominated by large increases in China and India of 52.7% and 69.8%, respectively. In contrast, population-weighted PM_{2.5} reduced in the EU and US by -55.3% and -38%, respectively. Because of the positive correlation between the spatial distribution of PM_{2.5} concentrations and population, population-weighted PM_{2.5} concentrations are higher than the regional average (see figure S8). We find that the ratio of population-weighted to regional average PM_{2.5} in the EU and US has decreased over the period 1960 to 2009 (1.3 to 1.0 and 2.0 to 1.6, respectively), whereas the ratio has increased in both China and India (1.4 to 1.6 and 1.1 to 1.3, respectively). These changes match those reported previously (Wang *et al* 2017) and are driven by anthropogenic emission changes (figure 1) and changes in population (figure S5).

Figure 4 shows the estimated attributable deaths over the period 1960 to 2009 (see SI data 2 for all data values). Using PM_{2.5} concentrations from GBD-PM_{high}, we estimate 2.6 million global attributable deaths in the year 2009, with a lower and upper uncertainty interval of 1.87 to 3.57 million. Estimated attributable deaths from UKCA_{ppe-med} are 22.5% lower at 2.0 (1.4 to 2.9) million for the same year, due to lower estimated PM_{2.5} concentrations. When GBD-PM_{high} is averaged to the same resolution as UKCA (GBD-PM_{low}), global attributable deaths are reduced by less than 3%, again demonstrating that the coarse resolution of UKCA is not the dominant reason for the lower global mortality estimate in UKCA_{ppe-med}. Larger regional differences occur in

regions with low PM_{2.5} concentrations such as the US where estimated attributable deaths from GBD-PM_{low} are ~10% lower than GBD-PM_{high}. This greater sensitivity occurs because the IER relationship is non-linear and particularly sensitive to changes in PM_{2.5} just above the theoretical minimum risk exposure level (TMREL) (~6 $\mu\text{g m}^{-3}$). This sensitivity also explains the large difference in deaths estimated in UKCA_{ppe-med} compared to GBD-PM in the EU and US. Estimated attributable deaths from UKCA and GBD-PM are in better agreement over China and India, where higher PM_{2.5} concentrations are associated with reduced sensitivity in the IER. Attributable deaths estimated using the upper and lower bound of the PPE bracket GBD-PM, showing the contribution of uncertainty in model processes to estimated mortality.

During the period 1990 to 2009, UKCA_{ppe-med} estimated global deaths increased by 15.6%, similar to the 22% change in GBD-PM. At the regional scale, UKCA_{ppe-med} simulates broadly similar fractional changes to GBD-PM in both China and India, but only simulates half the fractional change in the EU and US. During the period 1960 to 2009, global attributable deaths increased by an average of 124.4%. If we assume background disease rates prior to 1980 vary, this increase is reduced to 88.5% (see figure S9) because background diseases are comparatively higher in 1960. The percentage increase in attributable deaths is substantially greater than increases in population-weighted PM_{2.5} concentrations over the same period, owing to the non-linear IER and to increases in population. Our results imply that global attributable deaths are now larger in the present day than at any other point since 1960.

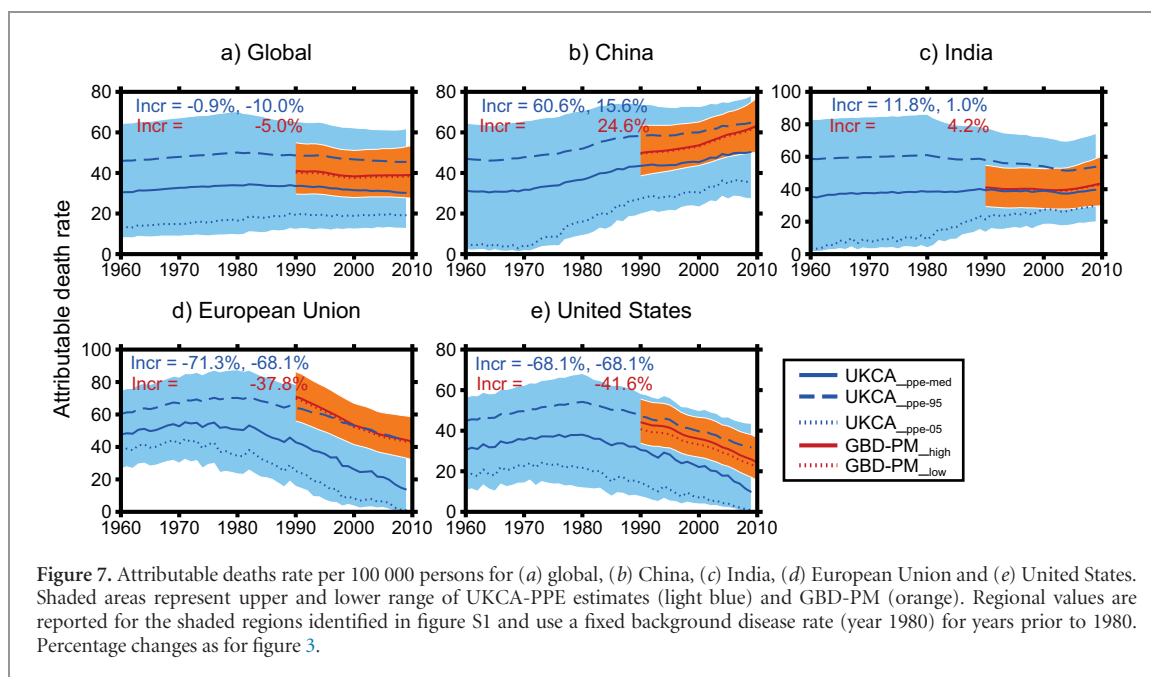
Global increases in attributable deaths were dominated by large increases in China (238%) and India



(194%). China and India accounted for 39% of global deaths attributable to $\text{PM}_{2.5}$ in 1960 growing to 55% in 2009. In contrast, attributable deaths reduced in the EU (−65.7%) and US (−47.9%) over this period. The US and EU accounted for 27% of global attributable deaths in 1960 falling to ~1% in 2009. If we assume that background disease rates prior to 1980 vary, attributable deaths in the EU and US peak in early-1970s following that of population-weighted $\text{PM}_{2.5}$ concentrations rather than peaking in early-1980s (see figure S9).

Figure 5 shows the spatial pattern of change in attributable deaths between 1960 and 2009. Large increases in deaths attributable to $\text{PM}_{2.5}$ are simulated in China and India as well as parts of Africa, the Middle East, and Central and South America. In contrast, reductions are simulated across much of Western Europe and North America.

Attributable deaths from cardiovascular disease contribute most to total global and regional attributable deaths (see figure S10). Figure 6 explores the relative contribution to changes in attributable deaths for the period 1980 to 2009 (see SI 1.6). Population growth and ageing act to increase attributable deaths, whereas declining background disease acts to reduce attributable deaths. In China and India, population growth and ageing and to a lesser extent increasing $\text{PM}_{2.5}$ concentrations act to increase mortality offset by reductions in background disease rates. In contrast, in the US and EU, reductions in background cardiovascular disease and $\text{PM}_{2.5}$ concentrations offset the contribution from population growth and ageing. Our results imply that air quality regulation and emission controls in Europe and North America are acting to reduce attributable burdens as observed in the US (Correia *et al* 2013, Pope *et al* 2009b).



Since the end of our simulation period (year 2009) regional changes in $PM_{2.5}$ concentrations and population demographics may have occurred. For example in China, population-weighted $PM_{2.5}$ concentrations stabilised between 2010 and 2015 (Brauer *et al* 2015, Cohen *et al* 2017), but attributable deaths continued to increase (Cohen *et al* 2017, Forouzanfar *et al* 2016). Our findings suggest that while primary and precursor emissions in China (and other parts of Asia) are likely to decrease over the next few decades (Zhao *et al* 2013, Zhao *et al* 2014), attributable deaths are likely to increase in the near future because of projected population growth and ageing (UN 2015). This highlights the need of strict control of $PM_{2.5}$ in the face of changing demographics.

Figure 7 shows the attributable death rate per 10⁵ of population for the period 1960 to 2009, which removes the influence of population growth. China had the highest attributable death rate in 2009, comparable to the EU in 1960. In contrast to the growth in total global attributable deaths, global attributable death rates reduced (−0.9%) over the period 1960 to 2009, a result of overall decreasing background disease rates and health benefits of cleaner air quality in North America and Western Europe. Decreasing background disease rates played an important role in influencing changes in attributable years of life lost (YLLs). For example, YLLs in India were markedly reduced between 1990 and 2010 (see figure S11) because of declines in infant (<5 yr) attributable mortality from LRI (see figure S10), a result of reduced disease rates (see figure S7), in part due to improved vaccination efforts, poverty alleviation and access to health care (Naghavi *et al* 2015).

Calculating the uncertainty in our attributable burdens is challenging because there are multiple sources of uncertainty. We have quantified uncertainty using

the lower and upper uncertainty bounds in the IER and background disease rates. Applying an exposure-response relationship (IER) based on epidemiological data from North America and Europe to the rest of the world, where lifestyles, age-structures, healthcare systems and $PM_{2.5}$ composition differ, is a critical source of uncertainty.

The IER neglects $PM_{2.5}$ particle composition and toxicity, which may be important (Lelieveld *et al* 2015, Thurston *et al* 2016, Tuomisto *et al* 2008). Further research is needed to establish the health implications of particle toxicity and source which may differ for each region.

The shape of the IER remains uncertain, particularly in very clean and polluted regions. We follow the GBD2013 and use a TMREL ($\sim 6 \mu g m^{-3}$) below which we assume zero risk. However, there is limited evidence for such a threshold. Additional research to constrain relative risks in very clean regions (Crouse *et al* 2012, Shi *et al* 2016, Tomczak *et al* 2016), where there is a lack of epidemiological data, is needed. Similarly, because of a lack of data, relative risks in polluted regions are based on extrapolations from active and passive smoking cohort studies (Pope *et al* 2009a, Pope *et al* 2011), leading to uncertainty in the IER at high exposure levels. This is important as the predicted shape of the IER is highly non-linear in polluted settings (figure S4). This implies that polluted regions will display the smallest reductions in relative risk from incremental pollution reduction. Our results suggest that current $PM_{2.5}$ concentrations in China and India are higher than those experienced in the EU and US during the 1960s and 1970s (figure 3). This suggests that stringent emission controls will be required to reduce population-weighted $PM_{2.5}$ concentrations and attributable health burdens.

Simulated $PM_{2.5}$ concentrations are uncertain due to uncertainties in emissions, meteorological input and

model processes. We have evaluated our estimated $PM_{2.5}$ concentrations using available long-term observations in North America and Europe. In regions where long-term observations are not available, we compare against satellite derived $PM_{2.5}$ data. There is an urgent need for more $PM_{2.5}$ observations, particularly in polluted and data sparse regions. We used the range of $PM_{2.5}$ concentrations from the PPE as an indication of the contribution of uncertain model processes, which indicated large uncertainties associated with dry deposition of accumulation mode particles in all regions, and mass flux of small scale residential combustion carbonaceous emissions in Asia (see SI 1.3 and figure S2). This analysis confirms a large contribution of residential emissions to $PM_{2.5}$ over Asia that has been shown previously (Butt *et al* 2016, Lelieveld *et al* 2015). Future research should prioritise constraining these large model uncertainties. UKCA does not include nitrate or anthropogenic secondary organic aerosol formation which may contribute to the underestimation of $PM_{2.5}$ concentrations. Multi-decadal global simulations of $PM_{2.5}$ are currently restricted to relatively coarse resolution, as used here. Differences in model spatial resolutions have been found to affect estimated attributable burdens (Ford and Heald 2015, Pungler and West 2013, Thompson *et al* 2014). Although we find small differences between estimates at $0.1^\circ \times 0.1^\circ$ versus the resolution of UKCA ($1.875^\circ \times 1.25^\circ$), further research using higher resolution estimates below $0.1^\circ \times 0.1^\circ$ like those used in Jerrett *et al* (2016) may provide more realistic personal exposures and thus attributable burdens.

Our estimates are subject to increased uncertainty prior to 1980 where we do not have data on background diseases. We find that varying assumptions about trends in background disease prior to 1980 increases global attributable deaths in 1960 by 16%. Information on historical background diseases trends would improve our attributable burden estimates prior to 1980. Background disease data is also provided at the national level, which does not account for any subnational variability, which may be important (Apte *et al* 2015, Chowdhury and Dey 2016, Cossman *et al* 2010). Similarly, we use national level data for different age groups, which is also unrealistic. Future research using subnational background disease and age group distribution data would improve future attributable burden estimates.

4. Conclusions

We used the HadGEM3-UKCA global coupled chemistry–climate model to investigate changes in ambient $PM_{2.5}$ concentrations and attributable burdens over the period 1960 to 2009. We found that the uncertainty in the model, estimated using a perturbed parameter ensemble of 235 simulations across 26 aerosol parameters, brackets long-term $PM_{2.5}$ measurements and satellite derived $PM_{2.5}$

concentrations used in the Global Burden of Disease (GBD) 2013.

We estimate that global population-weighted $PM_{2.5}$ concentrations increased by 37.5% over the period 1960 to 2009, dominated by increases in China and India, a result of economic expansion and growth in emissions. In contrast, air quality regulation and emission controls in the European Union (EU) and United States (US) has reduced population-weighted $PM_{2.5}$ concentrations over the same period.

We found that global attributable deaths increased by 89% to 124% over the period 1960 to 2009, much larger than the changes in $PM_{2.5}$ over the same period. Global changes were dominated by large increases in China and India. In contrast, attributable deaths decreased in the EU and US.

Increases in attributable deaths in China and India were dominated by population growth and ageing, and to a lesser extent increasing $PM_{2.5}$ concentrations. Reduced attributable deaths in the EU and US were driven by reductions in background disease rate and $PM_{2.5}$ concentrations. Our results suggest that projected changes in demography in China and India will pose challenges as policy makers attempt to reduce attributable deaths in the near future. Our results provide the first estimate of how $PM_{2.5}$ concentrations and associated health burden has changed over the 1960 to 2009 period. Understanding the reasons for these changes is required to help policy makers craft sound policies to reduce future health impacts.

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