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Future global mortality from changes in air pollution attributable to climate change

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Ground-level ozone and fine particulate matter ($PM_{2.5}$) are associated with premature human mortality[1-4]; their future concentrations depend on changes in emissions, which dominate the near-term[5], and on climate change[6,7]. Previous global studies of the air-quality-related health effects of future climate change[8,9] used single atmospheric models. However, in related studies, mortality results differ among models[10-12]. Here we use an ensemble of global chemistry–climate models[13] to show that premature mortality from changes in air pollution attributable to climate change, under the high greenhouse
Q.2 gas scenario RCP8.5 (ref. 14), is likely positive. We estimate 3,340 (−30,300 to 47,100) ozone-related deaths in 2030, relative to 2000 climate, and 43,600 (−195,000 to 237,000) in 2100 (14% of the increase in global ozone-related mortality). For $PM_{2.5}$, we estimate 55,600 (−34,300 to 164,000) deaths in 2030 and 215,000 (−76,100 to 595,000) in 2100 (countering by 16% the global decrease in $PM_{2.5}$-related mortality). Premature mortality attributable to climate change is estimated to be positive in all regions except Africa, and is greatest in India and East Asia. Most individual models yield increased mortality from climate change, but some yield decreases, suggesting caution in interpreting results from a single model. Climate change mitigation will likely reduce air-pollution-related mortality.

Climate change can affect air quality through several pathways, including changes in the ventilation and dilution of air pollutants, photochemical reaction rates, removal processes, stratosphere–troposphere exchange of ozone, wildfires, and natural biogenic and lightning emissions[6,7]. Overall, changes in these processes are expected to increase ozone in polluted regions during the warm season, especially in urban areas and during pollution episodes, but decrease ozone in remote regions due to greater water vapour concentrations leading to greater ozone destruction. These effects are exacerbated by the greater decomposition of reservoir species such as PAN[7]. $PM_{2.5}$ will also be affected by climate change, but Q.4
impacts vary in sign among models and show regional variation related to differences in precipitation, wildfires, biogenic emissions, $PM_{2.5}$ composition and other factors.

Previous studies have examined the impact of future climate change on human health via air quality globally[8,9,15] in the US[10,16–20] and in Europe[21]. However, only two studies have previously used an ensemble of models to assess air-pollution-related mortality attributable to climate change: one for the US[10], and our previous global work with the same ensemble used here, but evaluating the effects of historical climate change prior to 2000[11]. Both studies found a large spread of mortality outcomes depending on the atmospheric model used. Silva *et al.*[11] found that the multi-model Q.5
average suggested a small detrimental effect of climate change on global present-day air pollution-related mortality, but individual models yielded estimates of opposing sign.

The Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP) ensemble (Supplementary Table 1) simulated air quality in 2000, and in 2030, 2050 and 2100 for the four global Representative Concentration Pathway scenarios (RCPs)[22]. We previously estimated future air pollution premature mortality under all four RCP scenarios, estimating the net effect of both emissions changes and climate change[12]. Under RCP8.5, ozone concentrations increase in most locations in 2100 relative to 2000, due to increases in methane emissions and the effect of climate change[7,23], but $PM_{2.5}$ decreases in 2100 due to a projected decrease in particulate and precursor emissions[24]. These changes in pollutant concentrations lead to 316,000 (95% CI: −187,000 to 1.38 million) ozone-related excess deaths yr^{-1} and −1.31 (−2.04 to −0.17) million $PM_{2.5}$-related (avoided) deaths yr^{-1} in 2100[12]. Here we present results from additional ACCMIP simulations that were designed to isolate the influences of future climate change under RCP8.5, by simulating the projected climates of 2030 and 2100 (imposed by prescribing sea surface temperatures, sea ice

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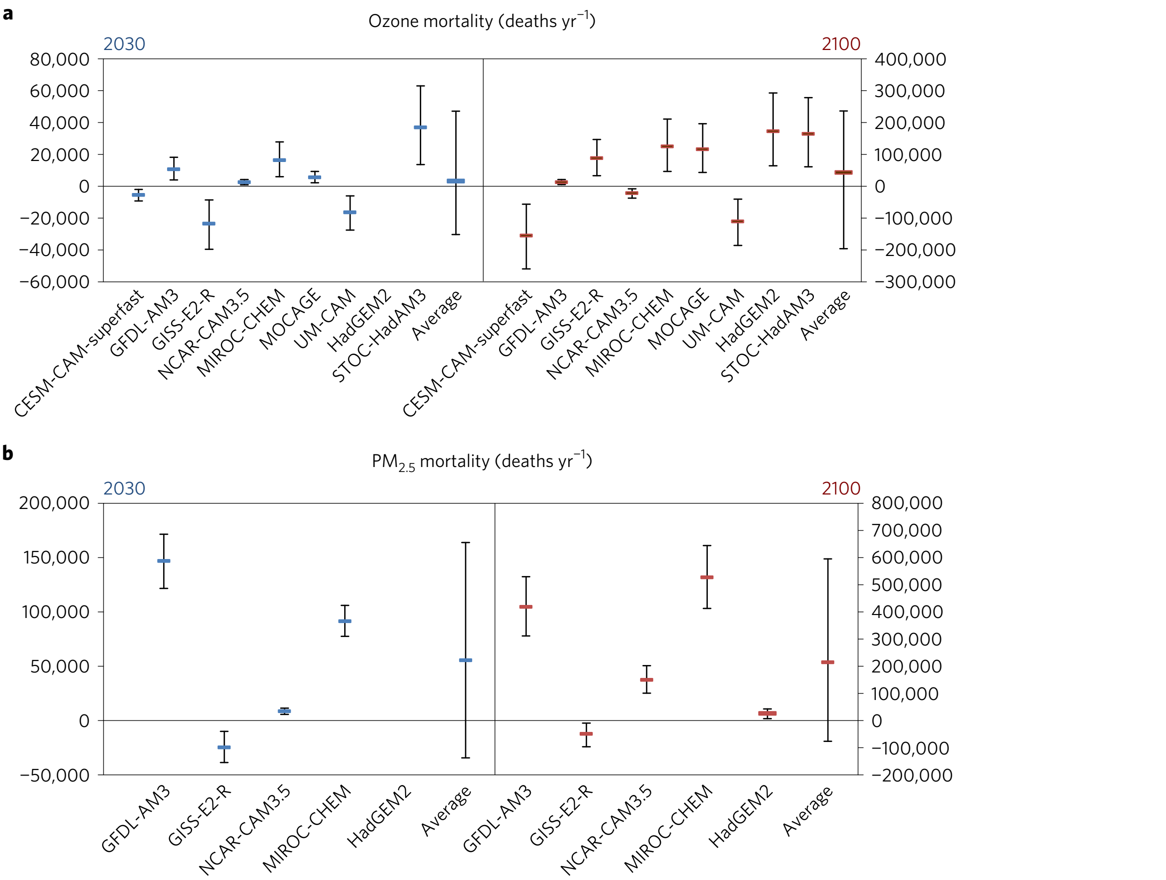

Figure 1 | Impact of RCP8.5 climate change on global mortality for individual models and the multi-model average. a,**b**, Estimates are for 2030 and 2100 for ozone respiratory mortality (9 models) (**a**) and PM2.5 IHD + STROKE + COPD + LC mortality (5 models) (**b**). PM2.5 is calculated as a sum of species. Uncertainty for each model is the 95% CI taking into account uncertainty in RR. Uncertainty for the multi-model average is the 95% CI including uncertainty in RR and across models.

cover, and greenhouse gas (GHG) concentrations for radiation) together with air pollutant emissions from 2000. The effects of climate change are then isolated by a difference with historical 2000 simulations. Premature mortality attributable to RCP8.5 climate change is estimated following the methods of Silva *et al.*[12], including projected population and baseline mortality rates (see Methods), such that mortality estimates here can be compared directly with overall changes in air-pollution-related mortality in RCP8.5.

We estimate that global ozone mortality attributable to RCP8.5 climate change will be 3,340 (−30,300 to 47,100) deaths yr^{-1} in 2030 and 43,600 (−195,000 to 237,000) deaths yr^{-1} in 2100 (Figs 1a and 2a). In 2100, ozone mortality increases in most regions, especially in highly populated and highly polluted areas, with marked spatial differences within regions that include both positive and negative mortality changes (Fig. 3a and Supplementary Table 2 and Supplementary Figs 1 and 2a). The effect on ozone mortality in 2100 is greatest in East Asia (45,600 deaths yr^{-1}, 41 deaths yr^{-1} per million people), India (16,000 deaths yr^{-1}, 8 deaths yr^{-1} per million people) and North America (9,830 deaths yr^{-1}, 13 deaths yr^{-1} per million people), but some areas within these and other regions show decreases in mortality. East Asia has high mortality effects per person in part because of its higher projected mortality rate from respiratory diseases. Climate change contributes 14% of the overall increase in ozone mortality estimated for RCP8.5 in 2100 relative to 2000[12]. However, three of eight models in 2030 and three of nine in 2100 show global decreases in ozone mortality due to climate change. For each model, the uncertainty range does not include zero; only the spread of models causes the overall uncertainty to span zero. Uncertainty in modelled ozone concentrations contributes over 97% to the overall uncertainty in both 2030 and 2100, with the remainder from uncertainties in relative risk (RR). Results from a sensitivity analysis using present-day population and baseline mortality rates (Table 1) show 32% and 67% lower mortality estimates in 2030 and 2100, respectively, largely because the projected baseline mortality rates of chronic respiratory diseases increase through 2100. The models agree that ozone will increase due to climate change in some polluted regions, notably the northeast US as found in other studies[6] and decrease in the tropics over the oceans (Supplementary Figs 3 and 4a). These changes are consistent with those analysed by Schnell *et al.*[25] for 2100, using four of these same models, and were attributed to a greater efficiency of precursor emissions to generate surface ozone in polluted regions, along with reductions in the export of precursors to downwind regions.

The impact of climate change on $PM_{2.5}$ mortality is estimated to result in 55,600 (−34,300 to 164,000) deaths yr^{-1} in 2030 and 215,000 (−76,100 to 595,000) deaths yr^{-1} in 2100 (Figs 1b and 2b). Mean estimates of $PM_{2.5}$ mortality increase in 2100 in all regions except Africa (−25,200 deaths yr^{-1}) (Fig. 3b and Supplementary Table 3 and Supplementary Fig. 2b). The greatest increases in mortality in 2100 occur in India (80,200 deaths yr^{-1}, 40 deaths yr^{-1} per million people), Middle East (50,400 deaths yr^{-1}, 45 deaths yr^{-1} per million people) and East Asia (47,200 deaths yr^{-1}, 43 deaths yr^{-1} per million people), although the Former Soviet Union shows greater mortality per million people in 2100 (11,800 deaths yr^{-1}, 57 deaths yr^{-1} per million people). Similar to ozone mortality, there are substantial spatial differences within each region, including both increases and decreases in mortality. For $PM_{2.5}$, a large decrease in mortality is projected in RCP8.5

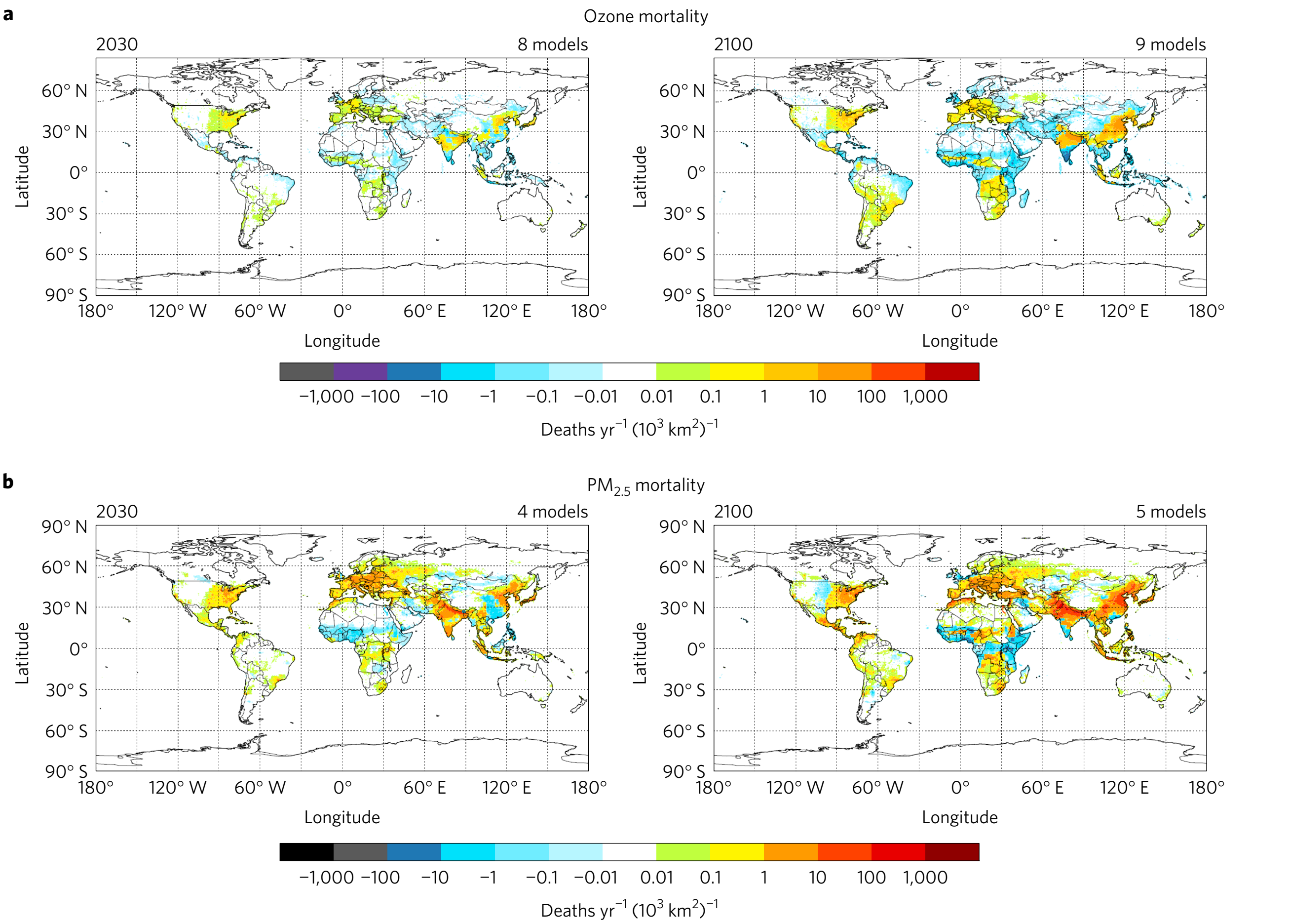

Figure 2 | Geographical impact of climate change on mortality. a,**b**, Estimates are for 2030 and 2100 for ozone respiratory mortality (**a**) and PM2.5 IHD + STROKE + COPD + LC mortality (**b**), showing the multi-model average in each 0.5° × 0.5° grid cell. PM2.5 is calculated as a sum of species.

relative to 2000 (when accounting for changes in both emissions and climate)[12], but climate change alone increases mortality, partially counteracting the decrease associated with declining emissions in RCP8.5. Without climate change, the decrease in $PM_{2.5}$-related mortality would be roughly 16% greater in 2100 relative to 2000. Propagating uncertainty in RR to the mortality estimates leads to coefficients of variation of 8–31% (2030) and 11–46% (2100) for the different models, but the spread of model results increases overall coefficients of variation to 123% in 2030 and 106% in 2100. In both years, one model (GISS-E2-R) yields a decrease in global mortality from climate change while the other three (2030) or four (2100) show an increase. Uncertainty in modelled $PM_{2.5}$ concentrations in 2000 makes a similar contribution to the overall uncertainty (50% in 2030 and 52% in 2100) compared with uncertainty in modelled $PM_{2.5}$ concentrations in future years (50% in 2030, 48% in 2100). Uncertainty in RR makes a negligible contribution in both periods (<1%), as the multi-model mean is small and different models disagree on the sign of the influence. Considering present-day population and baseline mortality rates (Table 1), we estimate 23% and 33% lower mortality in 2030 and 2100, respectively, mostly associated with the increase in projected baseline mortality rates through 2100.

$PM_{2.5}$-related mortality was estimated above for the sum of $PM_{2.5}$ species reported by five models, using a common formula (see Methods), to increase the number of models considered and to increase consistency among PM2.5 estimates. Additionally, we present a sensitivity analysis considering the PM2.5 concentrations reported by four models using their own PM2.5 formulae, for which multi-model average mortality results are modestly higher: 15% greater in 2030 and 12% in 2100 (Supplementary Fig. 5). The degree of agreement between the two estimates varies among the four models, and for one model (GISS-E2-R) the two sources of $PM_{2.5}$ estimates yield impacts of different sign in 2030.

There is considerable agreement among models regarding the increase in $PM_{2.5}$ concentrations in many locations in 2100, including most polluted regions, due to RCP8.5 climate change (Supplementary Fig. 4b). Allen *et al.*[26] analysed four of these same models in 2100 and found that global average surface $PM_{2.5}$ concentrations increased due to climate change, reflecting increases in nearly all relevant species for each model. They attributed this increase in $PM_{2.5}$ mainly to a decrease in wet deposition associated with less large-scale precipitation over land. Our multi-model mean estimates of global population-weighted changes for $PM_{2.5}$ and individual species (Supplementary Table 4 and Supplementary Fig. 6) are similar to those of Allen and colleagues[26]. Unlike Allen *et al.*[26], however, GISS-E2-R shows a net decrease in global population-weighted concentrations of total $PM_{2.5}$ and of each $PM_{2.5}$ species except sea salt, in 2100, likely due to projected concentration decreases over densely populated eastern China. Models also differ strongly in the sign and magnitude of changes in dust, particularly over North Africa and the Middle East; HadGEM2 projects increases in $PM_{2.5}$ for all species except dust, but a strong decrease in dust over the Middle East and South Asia. In Africa, the decrease in $PM_{2.5}$ near the Equator is likely caused by increased precipitation, whereas $PM_{2.5}$ increases are associated with (Q.7) precipitation decreases in southern Africa[26]. Differences in $PM_{2.5}$ (and ozone) responses to climate change among models likely result from differences in large-scale meteorological changes, and different

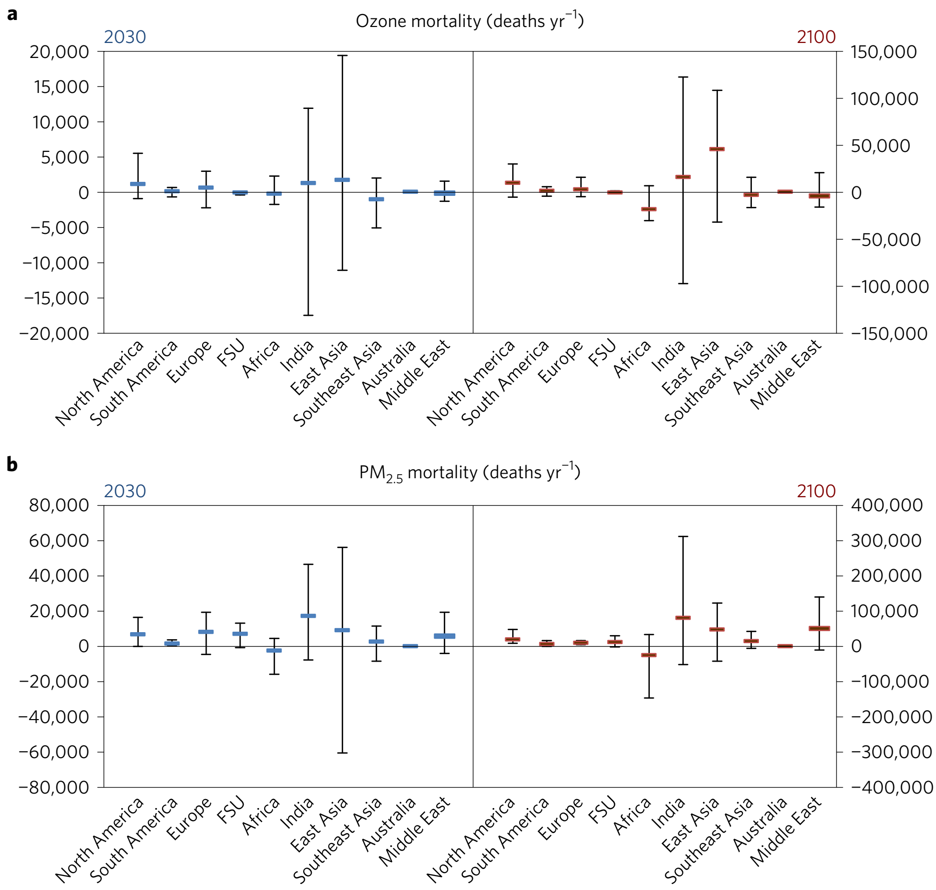

Figure 3 | Projected mortality for ten world regions. a,**b**, Estimates are for 2030 and 2100 for ozone respiratory mortality (**a**) and PM2.5 IHD + STROKE + COPD + LC mortality (**b**), showing the multi-model regional average. PM2.5 is calculated as a sum of species. Uncertainty for the multi-model regional average is the 95% CI including uncertainty in RR and across models. World regions are shown in Supplementary Fig. 1.

treatments of atmospheric chemistry and feedback processes among the models (such as the response of dust to climate change).

In the US, our multi-model mean mortality estimates for the impact of RCP8.5 climate change for ozone (1,130 deaths yr^{-1} in 2030; 8,810 deaths yr^{-1} in 2100) compare well to those of Fann *et al.*[20], who report 420 to 1,900 ozone-related deaths yr^{-1} for RCP8.5 climate change in 2030, despite differences in concentration–response functions and population and baseline mortality projections. These results for ozone and those for $PM_{2.5}$ (6,900 deaths yr^{-1} in 2030; 19,400 deaths yr^{-1}in 2100) are also consistent with the increases in mortality and spatial heterogeneity attributed to climate change in 2050 by Bell *et al.*[16] for ozone and Tagaris *et al.*[17] for ozone and $PM_{2.5}$, although these studies used different climate change scenarios besides other methodological differences. Across models, our estimates for ozone mortality in the US vary between −435 and 4,750 deaths yr^{-1} in 2030 and between −1,820 and 27,012 deaths yr^{-1} in 2100. This spread of model results, with a few models suggesting avoided mortality due to climate change, is similar to that of Post *et al.*[10] (−600 to 2,500 deaths yr^{-1} in 2050) using Special Q.8 Report on Emissions Scenarios (SRES) of GHG emissions. Similarly, results show spatial heterogeneity within several regions (Fig. 2) that is similar to Post *et al.*[10] for the US and Orru *et al.*[21] for Europe.

The spread of results among models highlights the uncertainty in the effect of climate change on air quality. Further improvements in chemistry–climate models are needed to better model the interaction and feedbacks between climate and air quality, including the sensitivity of biogenic emissions to climate change, the effects of meteorological changes on air quality (for example, aerosol–cloud interactions, secondary aerosol formation, wet deposition and gas–aerosol partitioning), and the impact of climate change on wildfires. Stratosphere–troposphere exchange of ozone is also important, as is the impact of land use changes on regional climate and air pollution. Our results are specific to climate change as projected under RCP8.5 and would differ for other scenarios. We estimate the effect of climate change as the difference between simulations with future climate and year 2000 climate, both with

Table 1 | Sensitivity analysis for changes in global air-pollution-related mortality attributable to climate change.

	$PM_{2.5}$-related mortality		Ozone-related mortality	
	2030	**2100**	**2030**	**2100**
Base results	56,300	218,000	10,700	128,000
$PM_{2.5}$ using Krewski *et al.*[2]	66,200	318,000	–	–
Present-day (2011) population	35,500	93,800	2,970	59,400
Present-day (2010) baseline mortality rates	69,600	510,000	2,790	13,300
Present-day population and baseline mortality rates	43,300	144,000	2,300	14,500

Estimates are for multi-model averages (deaths yr^{-1}) for the deterministic results.

year 2000 emissions, although global emissions of $PM_{2.5}$ and its main precursors decrease under RCP8.5. Had we instead modelled future emissions with present versus future climate, we would likely have attributed smaller changes in air pollution and mortality to climate change, given the projected emission reductions. Whereas the net effect of missing and uncertain processes does not clearly indicate an under- or overestimate for the effect of climate change on air quality, we likely underestimate the magnitude of the health impact by omitting mortality for people under 25, and morbidity effects. We also neglect possible synergistic effects of a warmer climate to modify air pollution–mortality relationships. Although a few studies have suggested stronger relationships between ozone[27] and $PM_{2.5}$ (ref. 28) and health at higher temperatures, there is insufficient evidence to include those effects here.

Despite these uncertainties, this study is the first to use a multi-model ensemble to show that global air-pollution-related mortality attributable to climate change is likely positive. The spread of results among models within the ensemble, including differences in the sign of global and regional mortality estimates, suggests that results from studies using a single model and a small number of model years should be interpreted cautiously. Actions to mitigate climate change, such as reductions in long-lived GHG emissions, will likely benefit human health by reducing the effect of climate change on air quality in many locations. These health benefits are likely to be smaller than those from reducing co-emitted air pollutants[29], but both types of health benefit via changes in air quality would add to reductions in many other influences of climate change on human health[30].

Methods

Methods, including statements of data availability and any associated accession codes and references, are available in the online version of this paper.

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Author contributions

J.J.W., J.-F.L., D.T.S. and R.A.S. conceived the study. All other co-authors conducted the model simulations. R.A.S. processed model output and estimated human mortality. R.A.S. and J.J.W. analysed results. R.A.S. and J.J.W. prepared the manuscript and all co-authors commented on it.

Additional information

Supplementary information is available in the online version of the paper. Reprints and permissions information is available online at www.nature.com/reprints. Publisher's note: Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations. Correspondence and requests for materials should be addressed to J.J.W.

Competing financial interests

The authors declare no competing financial interests.

Methods

The Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP)[13] included contributions from 14 modelling groups, of which 9 completed simulations that are used here (Supplementary Table 1). ACCMIP models incorporate chemistry–climate interactions, including mechanisms by which climate change affects ozone and $PM_{2.5}$, although models do not all include the same interactions, and do not always agree on their net effects[7]. Of these nine, three models are not truly coupled chemistry–climate models: MOCAGE is a chemical transport model driven by external meteorology, and UM-CAM and STOC-HadAM3 do not model the feedback of chemistry on climate[13]. As a result, these models do not fully capture the effects of changes in air pollutant concentrations on processes that affect meteorology, such as through radiative transfer and clouds. Prescribed anthropogenic and biomass burning emissions were very similar for the different models, but they used different natural emissions (for example, biogenic volatile organic compounds, ocean emissions, soil and lightning NO_x)[14,23]. Modelled 2000 concentrations show good agreement with observations for ozone[23] and $PM_{2.5}$ (ref. 24), although models tend to overestimate ozone in the Northern Hemisphere and underestimate it in the Southern Hemisphere, and to underestimate $PM_{2.5}$, particularly in East Asia.

We isolate the effect of climate change on air quality as the difference in concentrations between ACCMIP simulations using year 2000 emissions together with future year climate, imposed by prescribing RCP8.5 (ref. 31) sea-surface temperatures, sea ice cover and GHGs (for radiation) for 2030 and 2100 (referred to as 'Em2000Cl2030' and 'Em2000Cl2100'), and simulations with 2000 emissions and climate ('acchist2000')[13]. We analyse results from the nine models reporting ozone from the Em2000Cl2030/2100 simulations, and the five reporting $PM_{2.5}$ (Supplementary Table 1). Ozone and $PM_{2.5}$ species surface concentrations from each model are calculated in each grid cell, after regridding output from the native horizontal resolutions of each model ($1.9° \times 1.2°$ to $5° \times 5°$) to a common $0.5° \times 0.5°$ resolution. To be consistent with the epidemiological studies considered[1,4], we use the seasonal average of daily 1-h maximum ozone concentrations for the six consecutive months with the highest concentrations in each grid cell, and annual average $PM_{2.5}$ concentration.

Seven of the nine models with Em2000Cl2030/2100 simulations reported both hourly and monthly ozone concentrations, while two reported only monthly values. We calculate the ratio of the 6-month average of daily 1-h maximum concentrations to the annual average concentrations, for each grid cell and each year, for those models that reported both hourly and monthly concentrations; then, we apply that ratio to the annual average ozone concentrations for the other two models, following Silva *et al.*[11,12].

We calculate $PM_{2.5}$ concentration using the sum of $PM_{2.5}$ species mass mixing ratios reported by five models and a common formula:

$$PM2.5 = BC + OA + SO4 + SOA + NH4 + 0.25 * SS + 0.1 * Dust$$

where BC is black carbon, OA is (primary) organic aerosol corrected to include species other than carbon, NH4 is NH_4 in ammonium sulfate, SOA is secondary organic aerosol and SS is sea salt, as had been done previously by Fiore *et al.*[32] and Silva *et al.*[11,12]. The factors 0.25 and 0.1 are intended to approximate the fractions of sea salt and dust that are in the $PM_{2.5}$ size range. Nitrate was reported by three models, but we chose to omit nitrate from our $PM_{2.5}$ formula to avoid imposing changes inconsistent with the effect of climate change for other models, following Silva *et al.*[11], although nitrate was included in estimates of total $PM_{2.5}$ by Silva *et al.*[12]. Four of these models also reported their own estimate of $PM_{2.5}$ (Supplementary Table 1).

The impacts of climate change on global population-weighted differences (Em2000Cl2030/2100 minus acchist2000) in $PM_{2.5}$ and ozone concentrations for the different models are shown in Supplementary Tables 4 and 5, respectively, while regional multi-model average differences are shown in Supplementary Figs 7 and 8.

We estimate premature mortality by calculating the fraction of cause-specific mortality attributable to long-term changes in pollutant concentrations, using methods that are identical to those of Silva *et al.*[12], so that mortality attributable to climate change can be compared simply with changes in mortality under the RCP scenarios. We use relative risks (RRs) from Jerrett *et al.*[1] for ozone and respiratory diseases and Burnett *et al.*[4] for $PM_{2.5}$ and cardiopulmonary diseases and lung cancer. Then, we apply that attributable fraction in each grid cell to future adult population (age 25 and older) and baseline mortality rates based on projections from the International Futures (IFs) integrated modelling system[33]. Using country-level projections per age group, we mapped and gridded to the $0.5° \times 0.5°$ grid assuming that the present-day spatial distribution of total population within each country is unchanged in the future, as well as the present-day ratio of baseline mortality for the specific causes included in the epidemiological studies and for three disease groups projected in IFs (chronic respiratory diseases, cardiovascular diseases and malignant neoplasms). We select population projections from IFs instead of those underlying RCP8.5 to ensure consistency between projections of population and baseline mortality, since the latter are not available for RCP8.5, and for consistency with Silva and colleagues[12]. IFs projections of future total population are lower than those of RCP8.5 (−5% in 2030 and −27% in 2100) (Supplementary Fig. 9). Had we used projections of population underlying RCP8.5, we would have likely estimated greater changes in premature mortality relative to 2000. IFs projections of baseline mortality rates reflect an ageing population and regional demographic changes, showing a steep rise in chronic respiratory diseases (roughly tripling globally by 2100), particularly in East Asia and India, some regional increases in cardiovascular diseases (for example, Middle East and Africa), and global decreases in lung cancer.

Overall uncertainty in mortality estimates includes uncertainty from the RRs and from air pollutant concentrations. First, we conduct 1,000 Monte Carlo simulations separately for each model-year to propagate uncertainty from the RRs to mortality estimates. For ozone, we use the 95% confidence intervals (CIs) for RR reported by Jerrett *et al.*[1] and assume a normal distribution, while for $PM_{2.5}$ we use the parameter values of Burnett *et al.*[4] for 1,000 Monte Carlo simulations. Then, we calculate the average and 95% CI for the pooled results of the 1,000 Monte Carlo simulations for each model to quantify the spread of model results. We do not include uncertainties associated with population and baseline mortality rates, since these are not reported. As ACCMIP models used the same anthropogenic and biomass burning emissions, we do not consider uncertainty in emissions inventories; however, we acknowledge that this is an important source of uncertainty, especially in particular regions[34–37]. Our mortality estimates are affected by our choices of and underlying assumptions regarding concentration–response functions, population, and baseline mortality rates. Although a number of factors, such as vulnerability of the exposed population and $PM_{2.5}$ composition, vary spatially and possibly temporally, we assume that the RRs estimated for the present day apply on a global scale and in future time periods. Also, our assumption that the spatial distribution of population within each country is constant in the future likely understates the effects of rural-to-urban migration, which is currently underway and expected to continue. However, the effects of climate change on air pollutant concentrations may be spatially Q.12 uniform (as opposed to changes in emissions), and the coarse grid resolution of global models would not resolve air pollutant concentrations well in urban areas.

Data availability. Data used in this project are archived as follows:
Air pollutant concentrations. Atmospheric Chemistry & Climate Model Intercomparison Project (ACCMIP) data sets, http://catalogue.ceda.ac.uk/uuid/b46c58786d3e5a3f985043166aeb862d. Data retrieved from August 2012 to December 2013.
Present-day population. Oak Ridge National Laboratory (ONRL) - LandScan 2011 Global Population Dataset, http://spruce.lib.unc.edu.libproxy.lib.unc.edu/content/gis/LandScan. Data retrieved on 12/05/2012. Q.13
Present-day baseline mortality. Institute for Health Metrics and Evaluation (IHME): Global Burden of Disease Study 2010 (GBD 2010) Results by Cause 1990–2010 - Country Level, Seattle, United States, 2013, https://cloud.ihme.washington.edu/index.php/s/d559026958b38c3f4d12029b36d783da?path=%2F2010. Data retrieved from December 2013 to March 2014.
Future population and baseline mortality. Web-Based IFs - The International Futures (IFs) modeling system, version 6.54, www.ifs.du.edu. Data retrieved in July 2012.
IER model. Global Burden of Disease Study 2010. Global Burden of Disease Study 2010 (GBD 2010)—Ambient Air Pollution Risk Model 1990–2010. Seattle, United States: Institute for Health Metrics and Evaluation (IHME), 2013, http://ghdx.healthdata.org/record/global-burden-disease-study-2010-gbd-2010-ambient-air-pollution-risk-model-1990-2010. Data retrieved on 11/08/2013.

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