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2013 Environ. Res. Lett. 8 034005

(http://iopscience.iop.org/1748-9326/8/3/034005)

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Environ. Res. Lett. **8** (2013) 034005 (11pp) doi:10.1088/1748-9326/8/3/034005

Global premature mortality due to anthropogenic outdoor air pollution and the contribution of past climate change

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Received 25 March 2013
Accepted for publication 23 May 2013
Published 11 July 2013
Online at stacks.iop.org/ERL/8/034005

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Abstract

Increased concentrations of ozone and fine particulate matter ($PM_{2.5}$) since preindustrial times reflect increased emissions, but also contributions of past climate change. Here we use modeled concentrations from an ensemble of chemistry–climate models to estimate the global burden of anthropogenic outdoor air pollution on present-day premature human mortality, and the component of that burden attributable to past climate change. Using simulated concentrations for 2000 and 1850 and concentration–response functions (CRFs), we estimate that, at present, 470 000 (95% confidence interval, 140 000 to 900 000) premature respiratory deaths are associated globally and annually with anthropogenic ozone, and 2.1 (1.3 to 3.0) million deaths with anthropogenic $PM_{2.5}$-related cardiopulmonary diseases (93%) and lung cancer (7%). These estimates are smaller than ones from previous studies because we use modeled 1850 air pollution rather than a counterfactual low concentration, and because of different emissions. Uncertainty in CRFs contributes more to overall uncertainty than the spread of model results. Mortality attributed to the effects of past climate change on air quality is considerably smaller than the global burden: 1500 (−20 000 to 27 000) deaths yr^{-1} due to ozone and 2200 (−350 000 to 140 000) due to $PM_{2.5}$. The small multi-model means are coincidental, as there are larger ranges of results for individual models, reflected in the large uncertainties, with some models suggesting that past climate change has reduced air pollution mortality.

Keywords: climate change, air pollution, ozone, particulate matter, human health, premature mortality

S Online supplementary data available from stacks.iop.org/ERL/8/034005/mmedia

1. Introduction

Since the industrial revolution, human activities have significantly increased the concentrations of ozone and fine particulate matter (with aerodynamic diameter less than 2.5 μm, $PM_{2.5}$) in both urban and rural regions (Schulz *et al* 2006, Parrish *et al* 2012). These changes have been driven by direct changes in air pollutant emissions, and, because climate change also influences air quality, a component of the changes in anthropogenic air pollution may result from past climate change. Climate change influences air quality through several mechanisms, including changes in photochemical reaction rates, biogenic emissions, deposition, and atmospheric circulation (Jacob and Winner 2009, Weaver *et al* 2009, Fiore *et al* 2012).

Epidemiological studies have shown that ozone and $PM_{2.5}$ have significant influences on human health, including premature mortality. Evidence for mortality influences comes from a large number of daily time series studies (e.g., Bell *et al* 2004, HEI 2004). There is also evidence for chronic effects on mortality through several large cohort studies for $PM_{2.5}$ (Hoek *et al* 2002, Krewski *et al* 2009, Lepeule *et al* 2012), while evidence for chronic effects of ozone derives mainly from one study (Jerrett *et al* 2009).

Past research to estimate the global burden of disease due to outdoor air pollution has used a variety of methods. Cohen *et al* (2004) estimated 800 000 premature deaths annually attributed to urban $PM_{2.5}$ globally, based on surface measurements. Accounting for both urban and rural regions globally, Anenberg *et al* (2010) used output from a global atmospheric model to estimate 3.7 ± 1.0 million deaths annually due to anthropogenic (present-day relative to preindustrial) changes in $PM_{2.5}$ and 0.7 ± 0.3 million due to ozone. Brauer *et al* (2012) used high-resolution satellite observations of $PM_{2.5}$ together with a global atmospheric model and an extensive compilation of surface measurements to better represent global air pollution exposure. These exposure estimates were then used to estimate 3.2 ± 0.4 million premature deaths due to $PM_{2.5}$ and 150 000 (50 000 to 270 000) due to ozone (Lim *et al* 2012).

Few studies have assessed the effects of climate change on human health via changes in air quality. Of those, the focus has been on the influence of future climate change, including assessments on a metropolitan scale (Knowlton *et al* 2004, Sheffield *et al* 2011), in the US (Bell *et al* 2007, Tagaris *et al* 2009, Post *et al* 2012), and globally (West *et al* 2007, Selin *et al* 2009). Of these studies, only Post *et al* (2012) use a multi-model ensemble, showing significant variability in estimates of ozone-related mortality attributed to climate change depending on the atmospheric model results used. For the effects of past climate change on air quality and human health, Orru *et al* (2013) evaluated regional effects of ozone in Europe for both the recent past and the future. Fang *et al* (2013) conducted a global analysis of past climate change based on simulations from a single atmospheric model (GFDL-AM3); those model simulations are included in the multi-model ensemble used here.

Here we assess the burden of global anthropogenic air pollution on premature human mortality, and the contribution of past changes in climate to the total burden, using simulations from an ensemble of global coupled chemistry–climate models (Lamarque *et al* 2013). Our approach to estimate the global burden of air pollution on mortality expands on that of Anenberg *et al* (2010) by using an ensemble of model estimates of both present-day and preindustrial air pollution. We then use simulations that

combine present-day emissions and preindustrial climate to separate the influences of past climate change on air quality and human health.

2. Methods

2.1. Modeled ozone and $PM_{2.5}$ surface concentrations

The ensemble of global model simulations was conducted under the Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP) (Lamarque *et al* 2013, Fiore *et al* 2012, Stevenson *et al* 2013), including 14 models, 10 of which fully couple meteorological and chemical processes. Here we only analyze historical ACCMIP simulations, and not future simulations under different emissions scenarios. All models in ACCMIP used nearly identical anthropogenic emissions for both the present-day (2000) and preindustrial (1850), but differ in natural emissions (Lamarque *et al* 2010, 2013, Young *et al* 2013). Comparison with observations suggests that the models reproduce aerosol optical depth well, though with a tendency to underestimate particularly in East Asia (Shindell *et al* 2013). For ozone, the models also agree well with satellite and ozonesonde observations, but with a tendency to overestimate in the Northern Hemisphere and underestimate in the Southern Hemisphere (Young *et al* 2013). Differences in natural emissions (biogenic VOCs), model chemical mechanisms, and ozone transport from the stratosphere contribute to the spread of ozone concentrations across models (Young *et al* 2013).

For ozone, we use output from 14 models that report results from both 1850 and 2000 simulations; of these, 9 models also report results from an experiment where 2000 emissions are used together with 1850 climate ('Em2000Cl1850'), to separate the influence of past climate change on air quality. For $PM_{2.5}$, 6 models report results from 1850 and 2000, and 5 of these also report results for Em2000Cl1850.

We refer to the absolute difference in concentrations between 1850 and 2000 as 'anthropogenic' air pollution, although 1850 includes some anthropogenic emissions, such as from biomass burning (Lamarque *et al* 2010), and the simulated past climate change includes some natural forcings as well as anthropogenic forcings. In attributing air pollution changes to past climate change, this approach accounts for effects of climate change on atmospheric processes and natural emissions, but ignores effects on anthropogenic emissions. To reduce the effects of inter-annual variability, models typically report several years of output for each simulation; we use the average of all years reported by most models (varying between 1 and 10 years), and use 10 years for models that reported more than 10 years. In all cases, modeled concentrations from the lowest vertical coordinate are taken to represent surface concentrations.

Modeled concentrations are processed by calculating metrics consistent with the underlying epidemiological studies we use to estimate premature mortality. For $PM_{2.5}$, this is the simple annual average concentration (Krewski *et al* 2009). For ozone, this is the 6-month ozone season average of the 1-h daily maximum ozone concentration (Jerrett *et al* 2009); we estimate the ozone season in each grid cell as the consecutive 6-month period with highest average 1-h daily maximum ozone. Model results for these two metrics are then regridded from each model's native grid resolution (varying from $1.9° \times 1.2°$ to $5° \times 5°$) to a common $0.5° \times 0.5°$ resolution used to estimate mortality. For ozone, 5 of the 14 models report only monthly average ozone concentrations; we calculate the average ratio of the 6-month 1-h maximum ozone to the annual average for the remaining 9 models and apply this ratio to these 5 models. For $PM_{2.5}$, 6 models report results for $PM_{2.5}$ species, and 4 of these models also report a $PM_{2.5}$ metric, estimated by each model as a sum of species using a formula unique to that model. For all 6 models, we estimate total $PM_{2.5}$ as a sum of species using a common formula (see supporting information available at stacks.iop.org/ERL/8/034005/mmedia), and as a sensitivity analysis, we estimate mortality using the $PM_{2.5}$ reported by 4 models.

For the burden of disease results, mortality is estimated for each model based on the change in concentration between the 2000 and 1850 simulations. This approach models anthropogenic air pollution as a result of both anthropogenic air pollutant emissions and past climate change, in contrast to Anenberg *et al* (2010) who did not include past climate change. For mortality due to past climate change, we use the change in concentration between the 2000 and Em2000Cl1850 simulations.

2.2. Health impact assessment

Mortality due to long-term exposure to air pollution is estimated following the methods of Anenberg *et al* (2010), with updated input data. Like Anenberg *et al* (2010), we estimate anthropogenic air pollution as a modeled change in concentration between the present-day and preindustrial, rather than evaluating mortality relative to a counterfactual low concentration (normally a single value representing unpolluted conditions or below which changes in concentration are assumed not to affect mortality, e.g., Cohen *et al* 2004). We use epidemiological concentration–response functions (CRFs, see supporting information available at stacks.iop.org/ERL/8/034005/mmedia) for chronic mortality from the American Cancer Society (ACS) study for $PM_{2.5}$ cardiopulmonary disease (CPD) and lung cancer (LC) mortality (Krewski *et al* 2009), and for ozone respiratory mortality (Jerrett *et al* 2009). We select CRFs from the ACS study because this cohort includes the largest population of the available long-term $PM_{2.5}$ studies (Hoek *et al* 2002, Lepeule *et al* 2012), and it is the only study that reports long-term ozone mortality (Jerrett *et al* 2009). By analyzing $PM_{2.5}$ and ozone mortality based on the same study, we achieve greater consistency and reduce the potential for double-counting of mortality from both pollutants. Relative risks from the ACS study differ from other cohort studies because of differences in population characteristics, pollutant concentrations, and epidemiological methods. CRFs from the US are applied globally, as available studies of the effects of ozone and

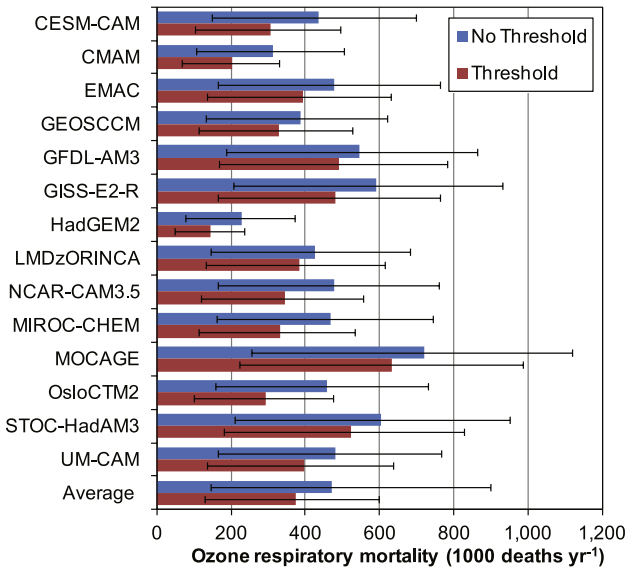

Figure 1. Estimates of the current global burden of anthropogenic ozone (2000–1850) on respiratory mortality from 14 models and the multi-model average, without and with a low-concentration threshold (33.3 ppb). Uncertainty for individual models reflects only the 95% confidence intervals on the CRF (Jerrett *et al* 2009). Uncertainty for the multi-model average is a 95% CI including uncertainty in the CRF and across models. See supporting information (table S1 available at stacks.iop.org/ERL/8/034005/mmedia) for summary information on each model.

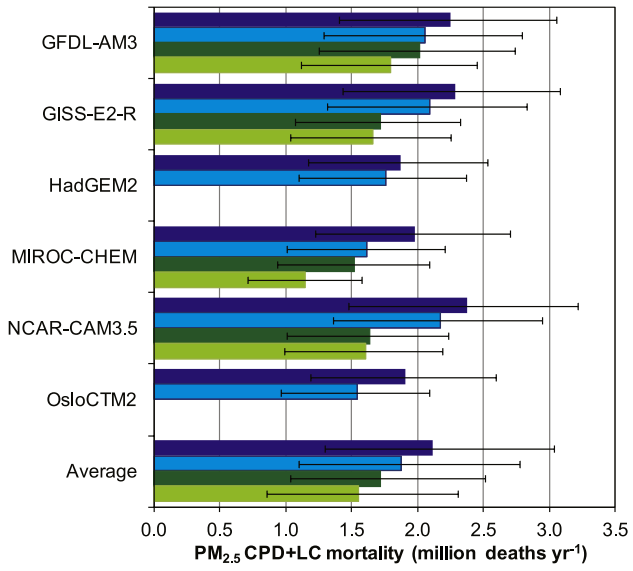

Figure 2. Estimates of the current global burden of anthropogenic $PM_{2.5}$ (2000–1850) on CPD and LC mortality with no low-concentration threshold, for $PM_{2.5}$ calculated as a sum of species for 6 models (dark blue), and for $PM_{2.5}$ as reported by 4 models (dark green). The corresponding estimates with a low-concentration threshold (5.8 μg m^{-3}) are shown for $PM_{2.5}$ calculated as a sum of species (light blue), and for reported $PM_{2.5}$ (light green). Uncertainty for individual models reflects only the 95% confidence intervals on the CRF (Krewski *et al* 2009). Uncertainty for the multi-model average is a 95% CI including uncertainty in the CRF and across models.

$PM_{2.5}$ on mortality outside of the US are broadly consistent (Hoek *et al* 2002, HEI 2004, 2010), and CRFs are not strongly dependent on sex, age, or race (Krewski *et al* 2009, Jerrett *et al* 2009). Nonetheless, differences in population exposure (including pollutant concentrations, the composition of $PM_{2.5}$ and air pollutant mixtures, and activity patterns) and susceptibility (including underlying health status) may cause differences in responses to air pollution internationally.

No low-concentration thresholds are assumed, as there is no clear evidence for the presence of thresholds. We analyze the sensitivity of the results to low-concentration thresholds of 33.3 ppb for ozone and 5.8 μg m^{-3} for $PM_{2.5}$, below which changes in concentration are assumed to have no effect, as these are the lowest measured levels in ACS.

Consistent with ACS, we limit our analysis to adults aged 30 and older (see supporting information, table S10 and figure S6 available at stacks.iop.org/ERL/8/034005/mmedia). Population data comes from LandScan (Dobson *et al* 2000) for the year 2008 at approximately 1 km^2 resolution, which is then regridded to 0.5° × 0.5°. The fraction of the population aged 30 and older in the year 2008 is taken from UN statistics for individual countries. Note that present-day population is used in all cases, so that we evaluate the effect of 2000 air pollution relative to 1850 or relative to the Em2000Cl1850 simulations, for present-day mortality. Baseline mortality rates (also for 30 and older) are from the WHO for individual countries, using the most recent data available for each country between 2000 and 2008, and when unavailable, reported regional rates are used (see supporting information). Baseline mortality rates for individual countries were gridded to the 0.5° grid using ArcGIS10 geoprocessing tools.

3. Global mortality burden of anthropogenic air pollution

Figures 1 and 2 show estimates of premature mortality due to anthropogenic ozone and $PM_{2.5}$ for each model, and changes in concentration underlying these estimates are presented in the supporting information (available at stacks.iop.org/ERL/8/034005/mmedia). The average estimate across the 14 models suggests that 470 000 premature respiratory deaths occur globally and annually due to anthropogenic increases in ozone, with no low-concentration threshold. Accounting for both the 95% confidence interval (CI) on the CRF, reported by Jerrett *et al* (2009), and the distribution of results from the 14 models, using Monte Carlo sampling, yields a 95% CI of 140 000 to 900 000 (uncertainty ranges reported hereafter follow the same methods). Global ozone mortality is about 20% lower when a low-concentration threshold is used. In figure 3 and table 1, ozone-related mortality is widespread globally, as ozone has increased essentially everywhere from human activities, but is greatest in highly populated and highly polluted areas of India and East Asia, which account for 68% of the global total.

For $PM_{2.5}$ estimated as a sum of species, the 6-model average indicates that 2.1 (1.3 to 3.0) million premature CPD and LC deaths occur globally and annually due to anthropogenic increases, with no low-concentration threshold.

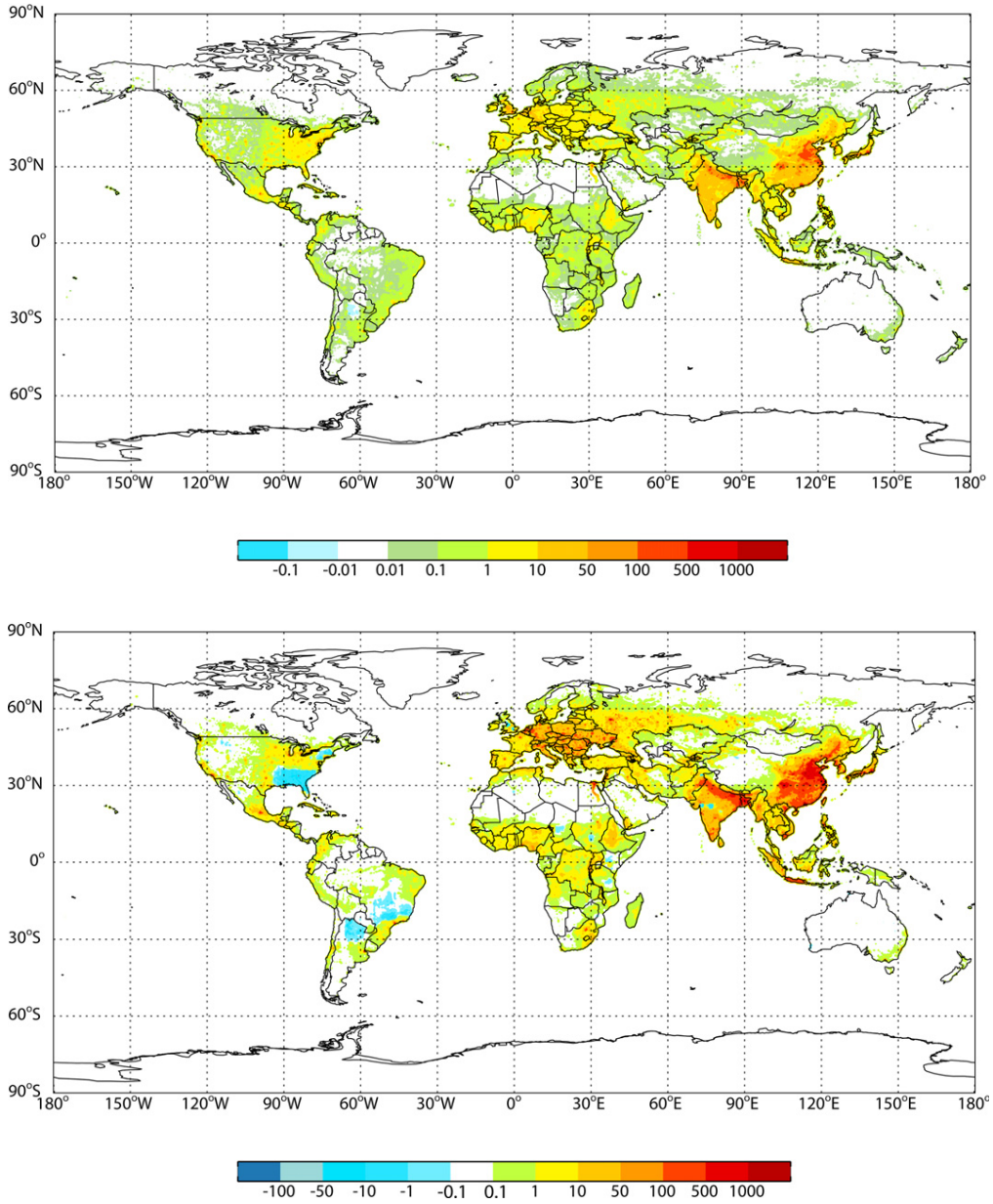

Figure 3. Current premature mortality due to anthropogenic air pollution (2000–1850), in deaths yr^{-1} $(1000\ km^2)^{-1}$, for the multi-model mean in each grid cell, for (top) ozone (respiratory mortality) for 14 models and (bottom) $PM_{2.5}$ (CPD + LC) for the sum of species for 6 models.

Of these deaths, 93% are related to CPD and 7% to LC. Relative to ozone, there is less scatter among the models, with a coefficient of variation (σ/μ) among models of 0.10 for $PM_{2.5}$, compared to 0.26 for ozone. For both $PM_{2.5}$ and ozone, the uncertainty in the CRF is greater than the uncertainty over the range of models. Global $PM_{2.5}$ mortality is 11% lower for the multi-model average when using a low-concentration threshold of 5.8 $\mu g\ m^{-3}$, and is 19% lower when using $PM_{2.5}$ as reported by 4 models. While the formulas for estimating $PM_{2.5}$ differ between models, the larger change in concentrations when adding species is mainly due to the omission of nitrate in the $PM_{2.5}$ reported by the models. Large differences may also result from differences in how dust and sea salt are added to $PM_{2.5}$, as models that calculate $PM_{2.5}$ use size-resolved information and so are likely more accurate than the common formula used here. $PM_{2.5}$-related mortality is widespread in populated regions, principally in East Asia and India, but also in Southeast Asia, Europe, and the Former Soviet Union. However, some locations are modeled as having a decrease in $PM_{2.5}$ relative to 1850, including the southeast US and parts of Latin America, and small regions elsewhere. In the southeast US, this decrease is caused by reductions in biomass burning relative to 1850, as changes in primary organic carbon are primarily responsible for the decrease, which also is apparent in the radiative forcing due to biomass burning aerosols (Shindell *et al* 2013). Local decreases in India and Africa likely relate to past climate change (see supporting information available at stacks.iop.org/ERL/8/034005/mmedia).

Table 1. Regional premature annual deaths from anthropogenic outdoor air pollution (2000–1850), for ozone (respiratory) and $PM_{2.5}$ calculated as a sum of species (CPD + LC), shown for the mean and full range across 14 models for ozone and 6 models for $PM_{2.5}$ (3 significant figures shown). Also shown are deaths per million people (present-day exposed population aged 30 and over) in each region, in parenthesis.

	Ozone			$PM_{2.5}$		
Region	Mean	Low	High	Mean	Low	High
North America	34 400 (121)	12 300 (44)	52 200 (184)	43 000 (152)	12 200 (43)	77 000 (272)
Europe	32 800 (96)	13 700 (40)	46 200 (135)	154 000 (448)	105 000 (306)	193 000 (562)
Former Soviet Union	10 600 (66)	5180 (32)	14 600 (91)	128 000 (793)	91 000 (568)	168 000 (1044)
Middle East	16 200 (68)	10 300 (43)	22 100 (93)	88 700 (371)	80 900 (339)	95 100 (398)
India	118 000 (212)	76 800 (138)	208 000 (376)	397 000 (715)	205 000 (370)	549 000 (989)
East Asia	203 000 (230)	62 900 (71)	311 000 (353)	1049 000 (1191)	908 000 (1031)	1240 000 (1406)
Southeast Asia	33 300 (119)	20 900 (75)	49 300 (176)	158 000 (564)	118 000 (422)	187 000 (669)
South America	6970 (38)	5180 (28)	8950 (49)	16 800 (92)	11 900 (65)	24 900 (137)
Africa	17 300 (73)	14 400 (61)	19 900 (84)	77 500 (327)	65 400 (276)	91 100 (385)
Australia	469 (29)	273 (17)	698 (44)	1250 (78)	911 (57)	2350 (147)
Global	472 000 (149)	229 000 (72)	720 000 (227)	2110 000 (665)	1880 000 (590)	2380 000 (748)

These estimates of the global burden are smaller than those reported by Anenberg *et al* (2010). Since Anenberg *et al* (2010) used the same CRFs and only small differences in global population and baseline mortality rates, the lesser estimated mortality is mainly due to differences in modeled concentrations. While the model used in that study differs from the ensemble used here, the greater difference is likely to be the different emissions used for both the present-day and preindustrial simulations (Lamarque *et al* 2010, Fang *et al* 2013).

These global burden estimates are also greater for ozone but less for $PM_{2.5}$ than were estimated in the most recent Global Burden of Disease study (Lim *et al* 2012). For ozone, these differences are likely explained by the fact that modeled 1850 ozone (table S5 available at stacks.iop.org/ERL/8/034005/mmedia) is lower than the assumed counterfactual low concentration of Lim *et al* (2012) of 37.6 ppb. For $PM_{2.5}$, the modeled 1850 concentrations are close to the counterfactual concentrations used by Lim *et al* (2012) of 7.3 μg m^{-3}; the smaller estimate here may be due to differences in CRFs.

Our estimates using results from the GFDL-AM3 simulations of Fang *et al* (2013) are 45% higher for ozone mortality and 24% higher for $PM_{2.5}$ mortality than those reported by Fang *et al* (2013); this difference is accounted for mainly by the smaller global population aged 30 years and older for the year 2000 used in that study and, to a lesser extent, differences in baseline mortality rates.

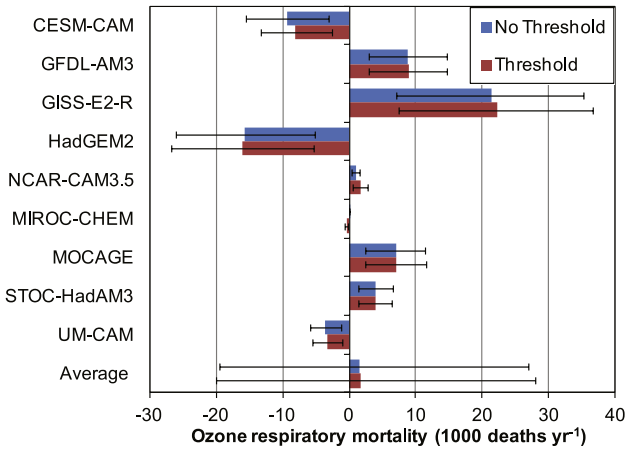

Figure 4. Estimates of the current global ozone respiratory mortality attributed to past climate change (2000–Em2000Cl1850), for 9 models and the multi-model average, with and without a low-concentration threshold (33.3 ppb). Uncertainty for individual models reflects only the 95% confidence intervals on the CRF (Jerrett *et al* 2009). Uncertainty for the multi-model average is a 95% CI including uncertainty in the CRF and across models.

4. Air pollution mortality attributed to past climate change

The 9-model average estimate of the effect of past climate change on ozone respiratory mortality is 1500 (−20 000 to 27 000) deaths annually with no threshold (figure 4). There is large variability among models, with six of nine models

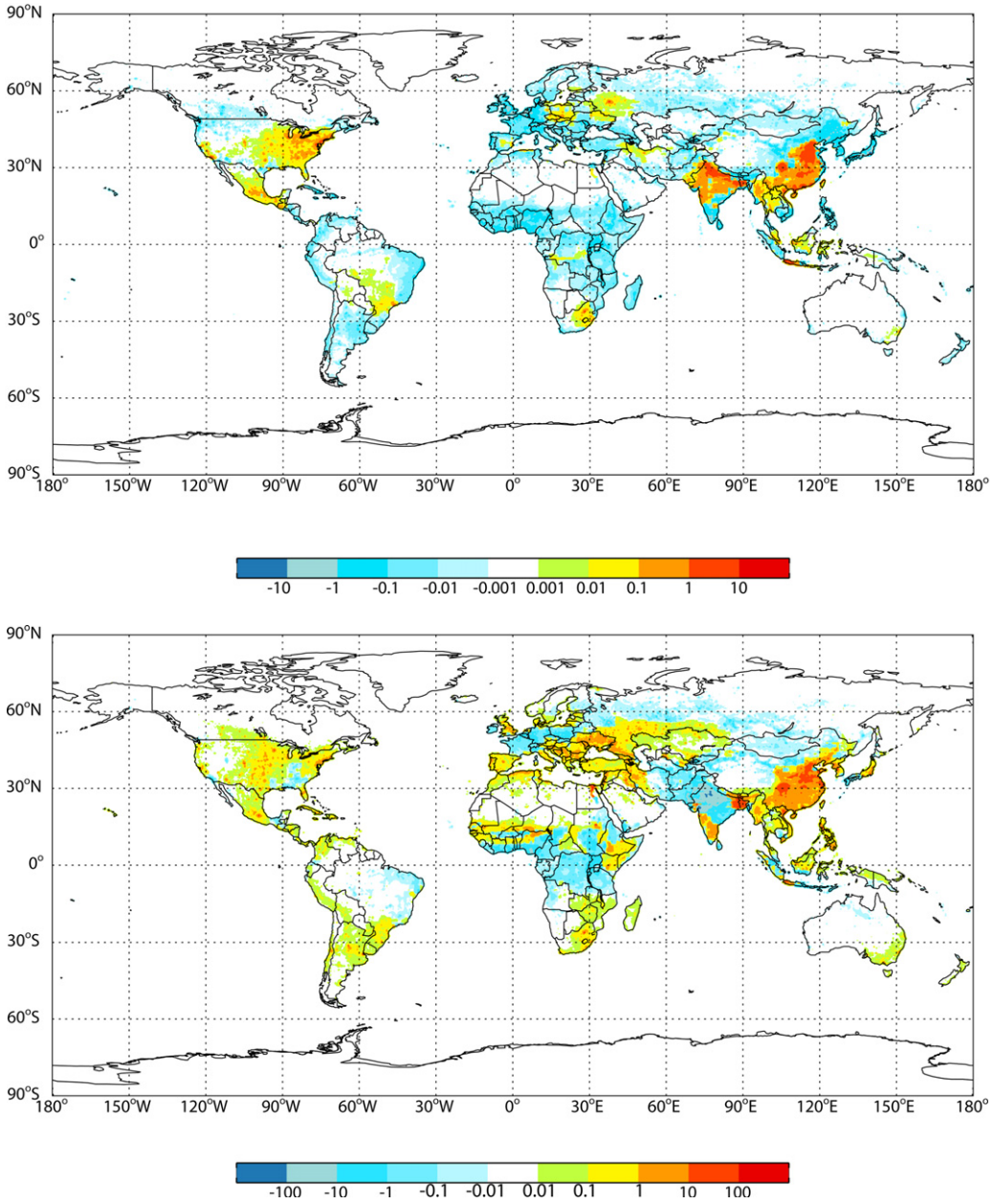

Figure 5. Premature mortality attributable to past climate change (2000–Em2000Cl1850), in deaths $yr^{-1}(1000\ km^2)^{-1}$, for the multi-model mean in each grid cell, for (top) ozone (respiratory mortality) for 9 models and (bottom) $PM_{2.5}$ (CPD + LC mortality) for the sum of species for 5 models.

suggesting that past climate change caused ozone mortality to increase. In figure 5 and table 2, deaths are greatest in East Asia for the multi-model average, but also positive in North America and parts of India. In figure 6, most models predict ozone decreases due to climate change in tropical regions and over oceans. This likely results from increases in water vapor, which causes greater production of HO_x radicals and greater destruction of ozone. Over polluted regions, however, ozone increases from faster reaction rates and meteorological changes (Jacob and Winner 2009). Because most models reported several years of simulations, the variability between models is not likely a result of inter-annual meteorological variability.

For $PM_{2.5}$, the 5-model average mortality (CPD + LC) attributed to past climate change is 2200 (−350 000 to 140 000) deaths annually, with no threshold and estimating $PM_{2.5}$ as a sum of species (figure 7). Four of the five models estimate an increase in deaths, but the average is decreased by one model (HadGEM2) that estimates −283 000 deaths from $PM_{2.5}$ decreases due to climate change. The multi-model median mortality is 61 000 deaths annually, and, if HadGEM2 is excluded, the multi-model average is 74 000 (30 000 to 140 000) deaths yr^{-1}. Average mortality is higher when using $PM_{2.5}$ from the four models that reported it, and without the large negative uncertainty, as HadGEM2 did not report $PM_{2.5}$. In figure 5 and table 2, the 5-model average shows that past climate change caused the largest increases in

Table 2. Regional premature annual deaths attributable to past climate change (2000–Em2000Cl1850), for ozone (respiratory) and $PM_{2.5}$ calculated as a sum of species (CPD + LC), shown for the mean and full range across 9 models for ozone and 5 models for $PM_{2.5}$ (3 significant figures shown). Also shown are deaths per million people (present-day exposed population aged 30 and over) in each region, in parenthesis.

	Ozone			$PM_{2.5}$		
Region	Mean	Low	High	Mean	Low	High
North America	621 (2)	−1110 (−4)	2360 (8)	3700 (13)	−6560 (−23)	18 800 (67)
Europe	−541 (−2)	−1520 (−4)	774 (2)	583 (2)	−27 100 (−79)	10 700 (31)
Former Soviet Union	−74 (0)	−674 (−4)	489 (3)	2090 (13)	−16 500 (−102)	9570 (59)
Middle East	−90 (0)	−851 (−4)	377 (2)	136 (1)	−6410 (−27)	12 300 (51)
India	871 (2)	−10 700 (−19)	11 000 (20)	−27 700 (−50)	−248 000 (−447)	59 400 (107)
East Asia	1490 (2)	−5720 (−6)	11 500 (13)	23 700 (27)	−32 500 (−37)	112 000 (128)
Southeast Asia	290 (1)	−852 (−3)	1730 (6)	3300 (12)	−7620 (−27)	8330 (30)
South America	−215 (−1)	−694 (−4)	260 (1)	1000 (6)	495 (3)	2390 (13)
Africa	−794 (−3)	−2930 (−12)	301 (1)	−4790 (−20)	−43 000 (−181)	16 200 (68)
Australia	−15 (−1)	−78 (−5)	25 (2)	193 (12)	39 (2)	520 (32)
Global	1540 (0)	−15 700 (−5)	21 400 (7)	2200 (1)	−283 000 (89)	111 000 (35)

$PM_{2.5}$ premature mortality in East Asia, and notable increases elsewhere including North America, but decreases in India and parts of Africa, the Former Soviet Union and Europe. The strong negative mortality estimate from HadGEM2 is the result of $PM_{2.5}$ decreases over India, driven by changes in dust, as India has a large exposed population. Figure 6 shows that most models predict an increase in $PM_{2.5}$ over India due to climate change, with only HadGEM2 being a strong exception. Most models predict increases in $PM_{2.5}$ over land, but it is difficult to explain the different regional patterns of concentration changes due to past climate change across the different models. These inter-model differences for both ozone and $PM_{2.5}$ are likely driven by the processes included in the different models, such as whether and how emissions from dust, vegetation, and lightning are modified as a result of climate change, and differences in how the models represent past climate change and its influences on photochemistry and pollutant transport and removal.

5. Conclusions

We estimate that in the present-day, anthropogenic changes to air pollutant concentrations since the preindustrial are associated annually with 470 000 (95% CI, 140 000 to 900 000) premature respiratory deaths related to ozone, and 2.1 (1.3 to 3.0) million CPD and LC deaths related to $PM_{2.5}$. Our estimates differ from those of Lim *et al* (2012) in that we estimate mortality for changes in air pollution relative to the modeled preindustrial conditions, rather than using a counterfactual low concentration. Relative to Anenberg *et al* (2010), our results also differ mainly because of the different emissions used in the models for preindustrial and present-day conditions, and by using modeled concentrations from an ensemble of models rather than a single model.

There is significant variability in mortality estimates driven by different atmospheric models, even though these models used very similar anthropogenic emissions, highlighting the uncertainty in basing results on a single model. Variability among models is higher for ozone than for $PM_{2.5}$, but for both pollutants, it contributes less to overall uncertainty than the uncertainty in CRFs. The uncertainty in CRFs is understated because it does not account for the full range over the literature—e.g., use of the CRF for $PM_{2.5}$ from Lepeule *et al* (2012) would lead to higher mortality estimates. The relative magnitude of results using different CRFs and with low-concentration thresholds, analyzed by Anenberg *et al* (2010), would also apply here. As for previous studies that estimate the mortality burden of outdoor air pollution, our methods likely underestimate the true burden because we have limited the evaluation to adults aged 30 and older, and base the analysis on coarse-resolution models that likely underestimate exposure, particularly for $PM_{2.5}$ in urban areas (Punger and West 2013). On the other hand, recent studies suggest that the relationship between $PM_{2.5}$ and mortality may flatten at high concentrations (Pope *et al* 2011), suggesting that we may overestimate $PM_{2.5}$ mortality in regions with

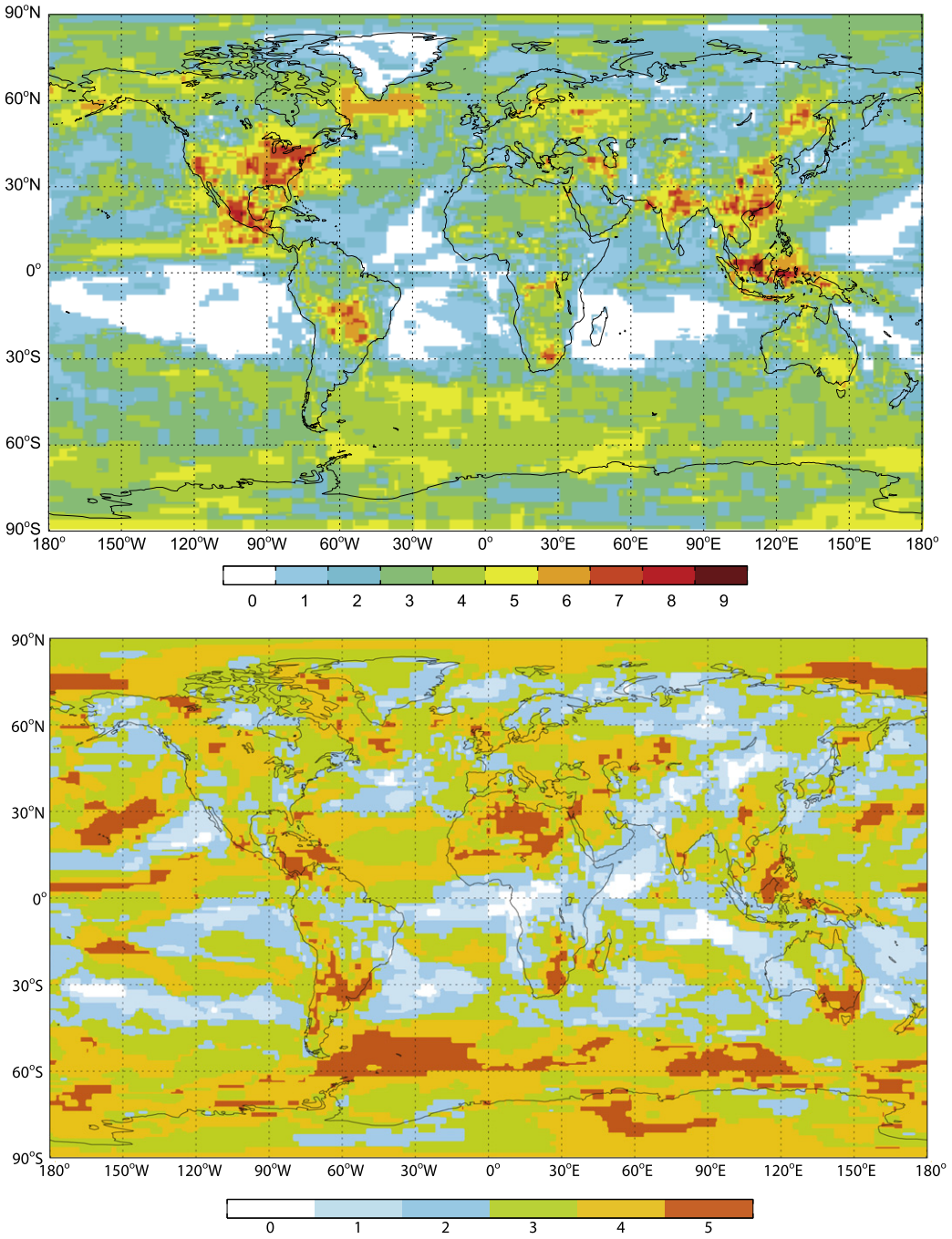

Figure 6. The number of models that show a positive change in concentration in each grid cell due to past climate change (2000–Em2000Cl1850), for (top) the 6-month ozone season average of 1-h daily maximum ozone (of 9 models total), and (bottom) annual average $PM_{2.5}$ calculated as a sum of species (5 models).

very high concentrations. We also caution that there are large uncertainties in applying CRFs from the US globally.

Air pollution-related mortality due to past climate change is shown to be significantly smaller than the total anthropogenic burden—i.e., anthropogenic increases in emissions likely have had a much greater influence on air pollutant concentrations than past climate change. We estimate here that 1500 (−20 000 to 27 000) premature respiratory deaths related to ozone and 2200 (−350 000 to 140 000) CPD and LC deaths related to $PM_{2.5}$ occur each year due to past climate change. The large uncertainties reflect significant variability among different atmospheric models, with some models estimating an overall decrease in mortality from past climate change. The multi-model averages for both ozone and $PM_{2.5}$ mortality are very small by coincidence, as the results for individual models show a large range of positive and negative values.

Consequently, it cannot be clearly concluded that past climate change has increased air pollution mortality. This large variability among models suggests that using a single

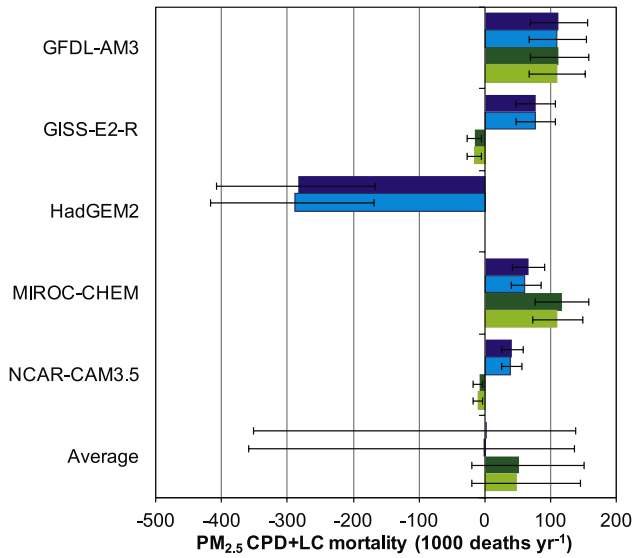

Figure 7. Estimates of the global $PM_{2.5}$ CPD and LC mortality attributed to past climate change (2000–Em2000Cl1850), for $PM_{2.5}$ calculated as a sum of species for 5 models (dark blue), and for $PM_{2.5}$ as reported by 4 models (dark green). Light-colored bars show the corresponding estimates with a low-concentration threshold (5.8 μg m^{-3}). Uncertainty for individual models reflects only the 95% confidence intervals on the CRF (Krewski *et al* 2009). Uncertainty for the multi-model average is a 95% CI including uncertainty in the CRF and across models.

model to represent past climate change may have significant uncertainties. This conclusion agrees with that of Post *et al* (2012) who analyzed the effects of future climate change on air pollution mortality in the US from an ensemble of atmospheric models. As models continue to develop and more comprehensively represent the mechanisms by which climate change might influence air quality, we should expect that large differences between estimates based on different models will likely persist.

Acknowledgments

This work was supported by fellowship SFRH/BD/62759/2009 (to RAS) from the Portuguese Foundation for Science and Technology (FCT), and by the International Council for Clean Transportation. Acknowledgments of funding for the modeling groups participating in ACCMIP can be found in previous publications (e.g., Lamarque *et al* 2013).

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