

Impacts of 21st century climate change on global air pollution-related premature mortality

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Abstract Climate change modulates surface concentrations of fine particulate matter (PM_{2.5}) and ozone (O₃), indirectly affecting premature mortality attributed to air pollution. We estimate the change in global premature mortality and years of life lost (YLL) associated with changes in surface O₃ and PM_{2.5} over the 21st century as a result of climate change. We use a global coupled chemistry-climate model to simulate current and future climate and the effect of changing climate on air quality. Epidemiological concentration-response relationships are applied to estimate resulting changes in premature mortality and YLL. The effect of climate change on air quality is isolated by holding emissions of air pollutants constant while allowing climate to evolve over the 21st century according to a moderate projection of greenhouse gas emissions (A1B scenario). Resulting changes in 21st century climate alone

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lead to an increase in simulated PM_{2.5} concentrations globally, and to higher (lower) O₃ concentrations over populated (remote) regions. Global annual premature mortality associated with chronic exposure to PM_{2.5} increases by approximately 100 thousand deaths (95 % confidence interval, CI, of 66–130 thousand) with corresponding YLL increasing by nearly 900 thousand (95 % CI, 576–1,128 thousand) years. The annual premature mortality due to respiratory disease associated with chronic O₃ exposure increases by +6,300 deaths (95 % CI, 1,600–10,400). This climate penalty indicates that stronger emission controls will be needed in the future to meet current air quality standards and to avoid higher health risks associated with climate change induced worsening of air quality over populated regions.

Abbreviations

O ₃	Ozone
PM _{2.5}	Fine particulate matter with an aerodynamic diameter less than 2.5 μm
YLL	Years of life lost
GFDL	Geophysical Fluid Dynamics Laboratory
AM3	Atmospheric model version 3
CI	Confidence interval
CCM	Coupled chemistry-climate model
CTM	Chemical transport model
GCM	General circulation model
LCT	Low concentration threshold
GHG	Greenhouse gas

1 Introduction

Climate change is projected to harm human health through adverse changes in food production, heat stress, sea level rise, increased storm intensity, flooding and droughts, increased incidence of vector-borne diseases, etc. (Portier et al. 2010; Tanser et al. 2003; Hales et al. 2002; Rosenzweig et al. 2001; Parry et al. 2007). In addition, climate change also affects surface concentrations of air pollutants, such as fine particulate matter (PM_{2.5}, particulate matter ≤2.5 μm in aerodynamic diameter) and ozone (O₃), which are associated with increases in human morbidity and premature mortality (WHO 2008a).

A number of global chemical-transport models (CTM) driven by off-line general circulation model (GCM) future-climate fields have investigated the effect of 21st century climate change on surface O₃ and PM_{2.5}. They have allowed climate to change while maintaining constant emissions of air pollutants over the 21st century. These studies consistently predict an increase in O₃ concentrations (+1–10 ppbv annually or during summers) over highly polluted northern mid-latitude regions (Jacob and Winner 2009; Weaver et al. 2009; Murazaki and Hess 2006; Liao et al. 2006; Fiore et al. 2012) and changes in global PM_{2.5} (±0.1–1 μg m⁻³ annually or during summer) due to projected 21st century climate change (Jacob and Winner 2009; Fiore et al. 2012). Recent sensitivity studies using newly coupled chemistry-climate models (CCM) provide a more integrated way to examine climate change impacts on surface air quality, as they allow coupling and feedbacks between dynamics, chemistry and physics, all of which directly affect air pollutant levels. CCM studies suggest that PM_{2.5} and aerosol concentrations may increase in a warmer climate because stratiform precipitation (a dominant driver for wet scavenging in the model) decreases when and where soluble pollutants are most abundant (Kloster et al. 2010; Fang et al. 2011).

Elevated concentrations of $\text{PM}_{2.5}$ and O_3 are detrimental to public health. Exposure to $\text{PM}_{2.5}$ is associated with an increased relative risk of lung cancer, cardiopulmonary and all-cause mortalities (Pope et al. 2002; Pope and Dockery 2006) while O_3 exposure is associated with increased incidence of cardiovascular, respiratory and all-cause mortality (Jerrett et al. 2009; Levy et al. 2005; Bell et al. 2004). As climate change leads to changes in surface O_3 and $\text{PM}_{2.5}$, it indirectly affects human health risks associated with these pollutants. Only a few studies have investigated the potential impact of climate change on air-quality relevant health in the United States: Bell et al. (2007) project that total daily mortalities in 50 U.S. cities will increase by 0.11–0.27 % from 2000s to 2050s due to increases in surface O_3 concentrations resulting from climate change; Tagaris et al. (2009) projects that, due to climate driven changes in $\text{PM}_{2.5}$ and O_3 , annual U.S. mortalities will increase by 4,300 deaths from 2001 to 2050. Here we go beyond previous work to use one of the first fully coupled global CCM to examine the effect of climate change induced changes in $\text{PM}_{2.5}$ and O_3 air quality over the 21st century on premature mortalities globally.

2 Methods

We use the Geophysical Fluid Dynamics Laboratory (GFDL) Atmospheric Model version 3 (AM3) global coupled CCM (Donner et al. 2011) to simulate present and future climate and air quality. AM3 builds on the capabilities of AM2 (Atmospheric Model, version 2) (GAMDT 2004), a climate model ranked among the best in the Intergovernmental Panel on Climate Change Fourth Assessment Report (IPCC AR4) (Reichler and Kim 2008) by adding tropospheric gas chemistry from the 3-D global CTM MOZART-2 (Horowitz et al. 2003), aerosol chemistry from the GOCART model (Ginoux et al. 2001), and interactions between clouds and aerosols (Ming et al. 2006).

Using AM3, we design a pair of sensitivity experiments (“present” and “future”) for this study. The “present” simulation is driven by a 1981–2000 climatology of observed monthly mean sea surface temperatures and sea ice. The “future” simulation is driven by this observed climatology plus the 19-model (from IPCC AR4) ensemble mean difference of 20-year average values from 1981–2000 to 2081–2100 under the moderate SRES A1B scenario. The concentrations of the long-lived greenhouse gases (CO_2 , N_2O and CFCs) are set to 1990 values for the present but 2090 (A1B) values for the future simulation. CH_4 is set to the 1990 level for tropospheric chemistry calculations in both simulations, but for radiation it is set to the 1990 and A1B 2090 levels in the current-day and future simulations, respectively. Emissions of short-lived pollutants in both present and future simulations are set to present levels, repeated annually. Each simulation is run for 20 years following a 1-year spin-up. Detailed configuration information can be found in Table S1 in the SI and in Fang et al. (2011, 2013a). With these configurations, changes in the surface concentrations of $\text{PM}_{2.5}$ and O_3 result entirely from changes in climate. Sensitivity experiments have been widely used in atmospheric chemistry and climate communities to isolate the impact of future climate on air quality (ACCMIP 2012; Fiore et al. 2012; Jacob and Winner 2009). Under the A1B scenario, the simulated global surface temperature and precipitation increase by 2.7 °C and 6 % respectively, consistent with the IPCC AR4 model ensemble mean (Meehl et al. 2007). We further evaluate simulated present surface O_3 and $\text{PM}_{2.5}$ (including sulfate, nitrate, fine dust particles, secondary organic aerosols, fine sea salt, black carbon and organic matter) with available observations and find that AM3 generally captures their magnitude and spatial distributions (see Figures S1–S3 in the SI).

The premature mortalities associated with simulated changes in surface O_3 and $PM_{2.5}$ due to the changing climate is calculated using concentration–response relationships that relate changes in pollutant concentrations to changes in mortality. Health impact functions for both O_3 and $PM_{2.5}$ are based on a log-linear relationship between relative risk and concentration. The excess mortalities attributable to air pollution ($\Delta Mort$) are described by:

$$\Delta Mort = POP \times Mort_{base} \times (1 - e^{-\beta \times \Delta X}), \quad (H1)$$

where β is the concentration–response factor (the estimated slope of the log-linear relationship between concentration and mortality), ΔX is the change in pollutant concentration due to climate change, $Mort_{base}$ is the baseline mortality rate and POP is the size of the exposed population.

For $PM_{2.5}$, we use β from Krewski et al. (2009), which indicates a 6 % (95 % CI, 4–8 %) increase in relative risk of all-cause mortality per 10 $\mu g/m^3$ enhancement in annual mean $PM_{2.5}$ for Americans age 30 and above from a reanalysis of the American Cancer Society epidemiological study (Pope et al. 2002). To minimize internal model variability, climate change impacts on air quality (ΔX) are represented as the difference between the 20-year average of $PM_{2.5}$ concentrations in the “future” and “present” simulations.

For surface O_3 , we use β from Jerrett et al. (2009), another reanalysis of the ACS cohort study. They find a 4 % (95 % CI, 1.3–6.7 %) increase in relative risk of death from respiratory disease due to a 10 ppbv increase in average daily maximum O_3 during the 6 months of highest U.S. O_3 (Apr 1st–Sept 30th). Months during which maximum O_3 concentrations occur vary depending on location. To be consistent with Jerrett et al. (2009), for each grid, we use the average of the 6 months with highest monthly average daily 1-h maximum O_3 (health-relevant O_3 , denoted as H- O_3 hereafter) within a year as the indicator of O_3 exposure. This permits us to include maximum O_3 concentrations in regions (e. g. East and South Asia) that have lower O_3 concentrations in summer than in spring and fall (Li et al. 2007; Mauzerall et al. 2000). ΔX of O_3 is then represented as the difference between the 20-year average H- O_3 concentrations in the “future” and “present” simulations. When 6 consecutive months with highest maximum O_3 within a year are used (Anenberg et al. 2010), similar results are obtained.

The exposed population (POP) is calculated by multiplying the year 2000 global population in each model grid (CIESIN 2005) by the percentage of adults age 30 and older (WHO 2004) in each grid. The baseline all-cause mortality rates ($Mort_{base}$) for adults age 30 and older is from (WHO 2004).

Years of life lost (YLL) estimates the number of years of life lost for each premature death relative to life expectancy and it varies among different populations depending on their age structure and health profiles. For its 14 sub-regions, WHO (2008b) provides YLL (calculated by using a discount rate and a non-uniform age weighting, which we define here as YLL_{base}) and mortality rate (defined here as $Mort_{base}$) for various disease categories. We calculate years of life lost (YLL) associated with premature mortalities using the baseline YLL per death ($YLL_{per\ death}$):

$$YLL_{per\ death} = \frac{YLL_{base}}{Mort_{base}}. \quad (H2)$$

Changes in YLL resulting from climate change induced changes in air pollution can therefore be calculated as:

$$\Delta YLL = \Delta Mort \times YLL_{per\ death}. \quad (H3)$$

In this paper, we isolate the effects of climate change on air pollution induced premature mortality by maintaining identical population, age distribution and baseline mortality in our “present” and “future” calculations. This approach has been used in previous studies to isolate the effect of climate change on health (Bell et al. 2007; Tagaris et al. 2009). We choose 10 regions (Figure S4 in the SI) to calculate the regional mean mortality and YLL responses to the impact of climate change on air pollution exposure. The size of the population age 30 and over and their corresponding baseline mortality rate from all-cause and respiratory disease over each region are shown as Table S2 in the SI. We use GIS software to map population and mortality data onto the model grid, and then apply equations (H1) and (H3) in each grid cell and sum globally to estimate the global change in premature mortality and YLL.

Epidemiological literature provides little evidence for any concentration threshold for either O_3 or $PM_{2.5}$ (Jerrett et al. 2009; Krewski et al. 2009; Schwartz et al. 2007; Pope and Dockery 2006). However, there is limited health data available at very low pollutant concentrations and mortality relationships below measured concentrations can only be estimated (i.e., Bell et al. 2006). To account for this uncertainty, previous studies choose to include a low concentration threshold (LCT) to account for natural background concentrations of O_3 and $PM_{2.5}$ (Anenberg et al. 2010, 2009; West et al. 2006a). Following previous literature, we conducted our estimates both with and without a LCT. By including a LCT, we assume O_3 and $PM_{2.5}$ have no effect on mortality below the LCT; by not including LCT, we assume the epidemiological function is valid even when O_3 and $PM_{2.5}$ concentrations are low. We find the application of a LCT of 25 ppbv O_3 and $5.8 \mu\text{g}/\text{m}^3$ $PM_{2.5}$ (Anenberg et al. 2010; West et al. 2006a) changes our results very little.

3 Results

3.1 Impact of 21st century climate change on surface air quality

1) Changes in surface $PM_{2.5}$

Simulated changes in surface $PM_{2.5}$, resulting from 21st century climate change are shown in Fig. 1. $PM_{2.5}$ increases over most regions, with a global mean increase of $0.28 \mu\text{g}/\text{m}^3$ ($0.48 \mu\text{g}/\text{m}^3$ when weighted by population, greater than the global average,

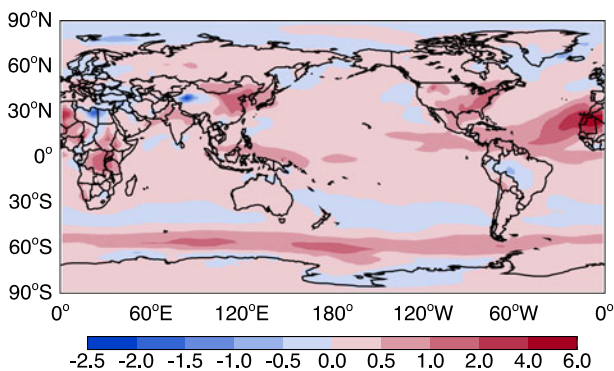


Fig. 1 Climate change induced changes in annual mean surface $PM_{2.5}$ concentrations from late 20th century (1981–2000) to late 21st century (2081–2100) (unit: $\mu\text{g}/\text{m}^3$). See [supplementary material](#) for changes in components of $PM_{2.5}$

indicating a greater increase over more populated areas). Among all $PM_{2.5}$ components, the largest increases are in sulfate, smaller dust particles and organic matter. Changes in major $PM_{2.5}$ components are shown as Figure S5 in the SI. This increase in $PM_{2.5}$ is mainly because stratiform precipitation, which dominates wet deposition in AM3, decreases in the locations where and seasons when soluble pollutants are most abundant, leading to less wet deposition (Fang et al. 2011). The dominance of stratiform precipitation has been confirmed by a more physically-based diagnostic cloud nucleation model (Croft et al. 2010). In addition, increased sulfate concentrations also result from an increase in hydrogen peroxide (H_2O_2 , +12 % globally due to higher moisture and OH concentrations) that leads to increase in-cloud production of sulfate ($SO_2+H_2O_2 \rightarrow H_2SO_4$) (Liao et al. 2006; Racherla and Adams 2006).

As shown in Fig. 1, in the northern hemisphere, surface $PM_{2.5}$ increases substantially near source regions (e.g., over East Asia, eastern United States, northern India, and Africa). These source regions are usually highly populated and hence, increases in $PM_{2.5}$ will adversely impact human health. With climate change, surface $PM_{2.5}$ concentrations decrease over western Brazil, parts of northern Europe, the Middle East and parts of North Africa, suggesting a potential “climate benefit” for air quality there.

2) Changes in surface O_3

Surface O_3 concentrations respond differently than $PM_{2.5}$ to projected 21st century climate change (Fig. 2a). The global annual mean O_3 concentration changes by -1.3 ppbv (-0.2 ppbv when weighted by population) with largest reductions occurring over remote oceanic regions and increases occurring over many polluted continental regions. The decrease in O_3 concentrations over remote oceanic regions and in the global mean is largely driven by an increase in water vapor in the warmer future atmosphere (Fig. 2b), which leads to increases in HO_x ($HO_x = OH + HO_2$) concentrations. Reaction with HO_x is the primary sink of O_3 at low NO_x ($NO_x = NO + NO_2$) concentrations. However, over regions with large NO_x emissions such as south China, north India, northeast United States and central Africa, we project surface O_3 will increase (Liao et al. 2006; Murazaki and Hess 2006). Factors contributing to this increase include: 1) more abundant HO_x due to increased water vapor converting more NO to NO_2 and hence enhancing O_3 production when NO_x concentrations are high; and 2) higher temperatures decreasing the net formation of peroxyacetyl nitrate (PAN, $CH_3C(O)OONO_2$), a reservoir species for NO_x), and hence, more NO_x remaining near its source regions (Fig. 2c), which in turn may enhance local O_3 production. Both higher water vapor concentrations and temperature contribute to higher surface O_3 concentrations where NO_x is abundant and lower concentrations where it is scarce, partially explaining the tendency of surface O_3 to increase over polluted continental regions and decrease over remote oceanic regions. Consistent with earlier studies (Mickley et al. 2004; Wu et al. 2008), we also find weaker cyclones and more stagnation over the northeastern United States under future climate (the root mean square of 2–8 day band-pass filtered 500 hPa geopotential height, as a measure of the climatological cyclone track, decreases by 2–10 %), which likely also contributes to increased surface O_3 over the northeastern US.

Projected changes in H- O_3 have a similar pattern as the changes in annual mean O_3 shown in Fig. 2a. However, H- O_3 generally has a larger increase over highly polluted regions because daily maximum concentrations over these regions are more driven by local daytime photochemical production. As a result, the population-weighted H- O_3 actually increases by 0.3 ppbv, suggesting an overlap of increase in H- O_3 and dense population.

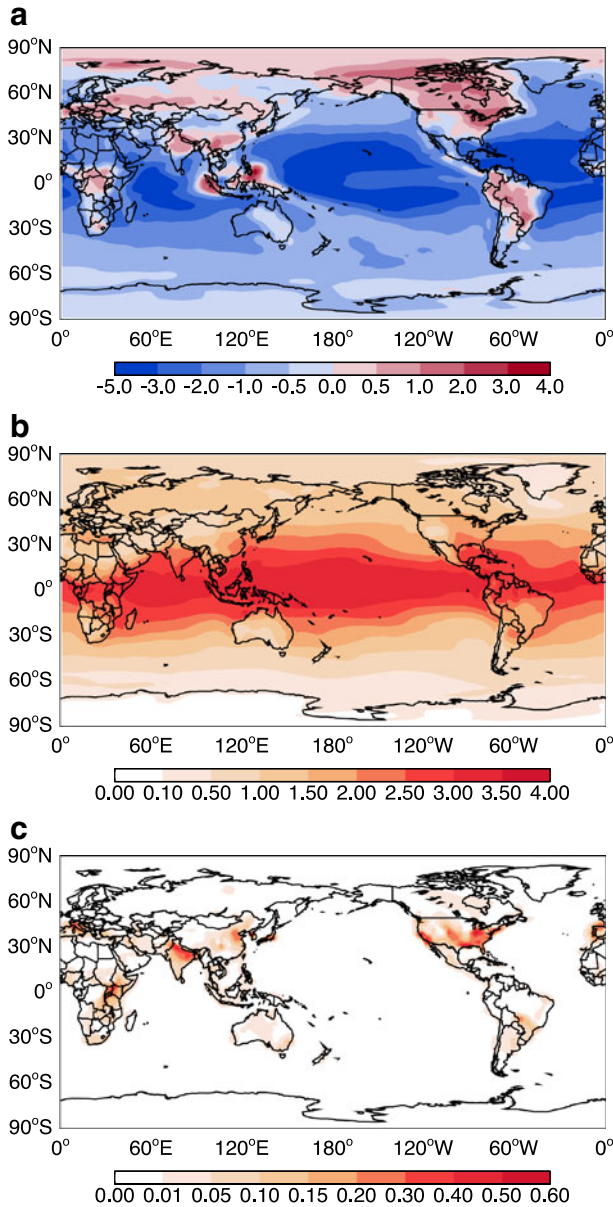


Fig. 2 Late 20th century (“present”) to late 21st century (“future”) climate change induced changes in annual mean surface **a** O₃ concentrations (ppbv), **b** specific humidity (g/kg), and **c** NO_x concentrations (ppbv)

3.2 Climate change induced air quality impact on premature mortality

1) *Effects of PM_{2.5}*

Applying the simulated climate change induced changes in PM_{2.5} concentrations in (H1), we find that global premature mortalities associated with PM_{2.5} increase by approximately 100,000 deaths (95 % CI: 66,000–130,000) without a LCT and 93,000

deaths (95 % CI, 63,000–123,000) with a LCT of $5.8 \mu\text{g}/\text{m}^3$ (Fig. 3). We additionally present percentage changes in mortalities (defined as the ratio between changes in mortality associated with $\text{PM}_{2.5}$ divided by present-day mortalities associated with $\text{PM}_{2.5}$). The present mortality associated with $\text{PM}_{2.5}$ is estimated by using the present-day total $\text{PM}_{2.5}$ concentration in (H1). We continue using the LCT for calculating present mortalities. Regardless whether the LCT is included or not, we find about a 4 % (difference less than 0.1 %) increase in global premature mortalities due to changes in $\text{PM}_{2.5}$ resulting from 21st century climate change. As the difference is small, we only report the relative change without LCT hereafter. As summarized in Table S3 in the SI, the increase in mortality associated with $\text{PM}_{2.5}$ exposure is strongest in East and South Asia where population density is highest; the relative increase, however, is largest over North America (+8 %), consistent with its greater relative change in $\text{PM}_{2.5}$ concentrations under future climate.

2) Effects of Ozone

The simulated change in O_3 concentrations due to 21st century climate change leads to an associated annual global increase in premature mortalities from respiratory disease in persons 30 years and older of 6,300 (95 % CI: 1,600–10,400) deaths (Fig. 3). The changes in mortality associated with O_3 are approximately 5 % of that from chronic exposure to $\text{PM}_{2.5}$. Application of a LCT of 25 ppbv to account for background O_3 concentrations affects our mortality results very little and hence hereafter we report results without applying a LCT. The regional change in premature mortalities associated with chronic O_3 exposure is shown in Table S3 in the SI. The increase in premature mortality from respiratory disease due to chronic O_3 exposure is driven mostly by changes over South Asia (1,400 deaths, 1.0 %), North America (1,000 deaths, 2.2 %) and East Asia (4,300 deaths, 1.5 %).

3) Summary

In North America, Europe, South and East Asia, 21st century climate change induced changes in both $\text{PM}_{2.5}$ and O_3 surface concentrations are projected to increase premature

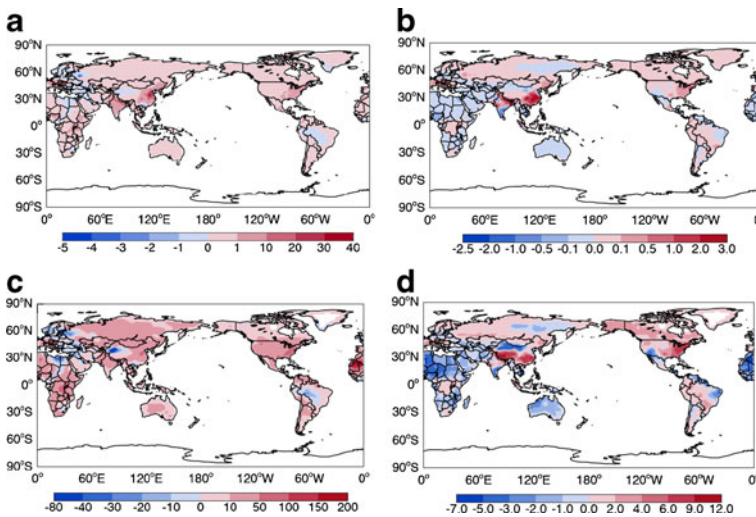


Fig. 3 Estimated change in premature mortalities associated with changes in surface $\text{PM}_{2.5}$ (left column) and O_3 (right column) resulting from 21st century climate change (i.e., “future” minus “present” simulations). **a, c** shows changes due to $\text{PM}_{2.5}$ and **b, d** shows changes due to O_3 . (units: **(a, b)** additional annual premature mortalities per 1,000 km^2 , **(c, d)** additional annual premature mortalities per million population)

mortality under the scenario of a constant population, baseline mortality rate and emissions of short-lived air pollutants. Climate change generates an air quality “climate penalty” by increasing surface concentrations of air pollutants and associated human health risks. As a result, stronger emission controls will be needed in many regions to maintain current air quality and public health. In the Middle East, climate change tends to reduce surface concentrations of $PM_{2.5}$ and O_3 and thus could potentially contribute to a public health “climate benefit”. However, uncertainties associated with this benefit are large. In addition, premature mortality resulting from higher temperatures, insufficient food supply and greater malarial risk will likely counteract this benefit (Husain and Chaudhary 2008).

3.3 Climate change impact on YLL attributed to $PM_{2.5}$ and O_3 exposure

Figure 4a and b show the baseline all-cause and respiratory disease YLL per death ($YLL_{unit\ death}$) for adults aged 30 and older, respectively, calculated using (H2). Figure 4c and e show

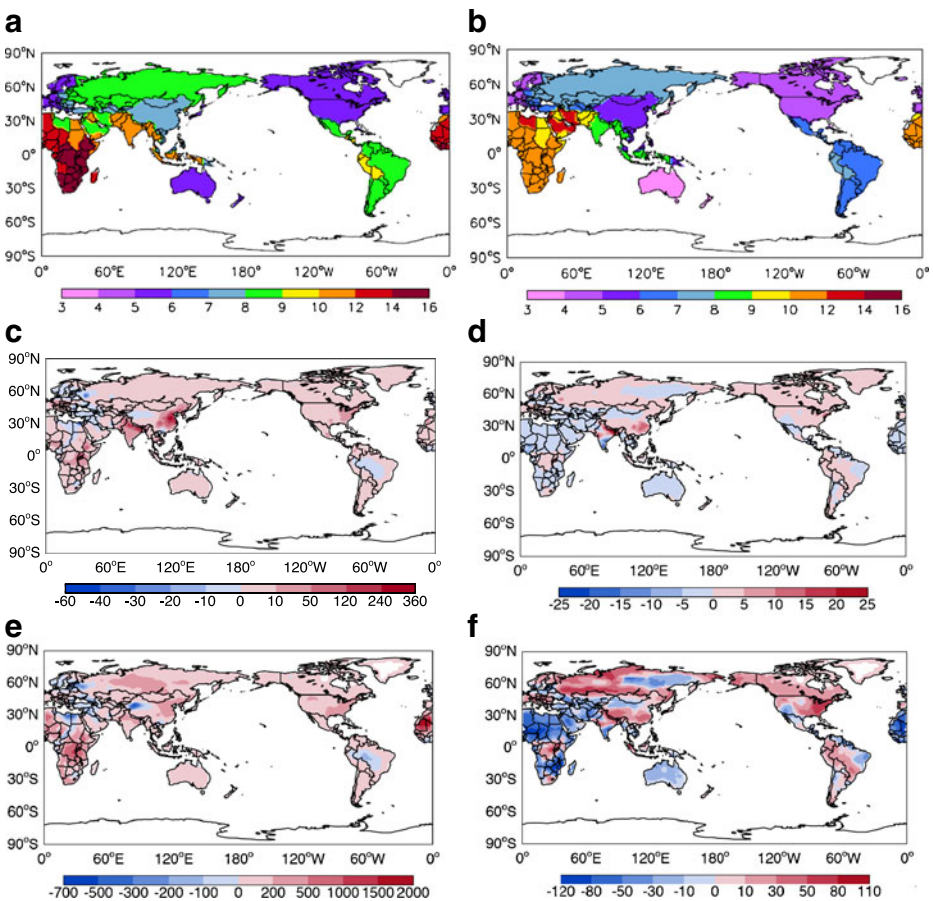


Fig. 4 Baseline YLL per death due to **a** all-cause and **b** respiratory disease mortality for adults aged 30 and older (WHO 2004) (unit: years/death). Changes in the estimated YLL due to changing $PM_{2.5}$ (*left column*) and O_3 (*right column*) in a future climate (unit: (**c**, **d**) years per 1,000 km²; (**e**, **f**) years per 1 million population)

the estimated change in all-cause YLL for adults 30 and older due to 21st century climate change induced changes in surface $PM_{2.5}$ while Figure 4d and f show the estimated change in YLL due to respiratory disease mortality under a future climate due to changes in O_3 exposure (using (H3)).

Globally, the all-cause YLL from changes in $PM_{2.5}$ is projected to increase by about 0.9 million years, a 4.1 % increase relative to the end of the 20th century all-cause YLL (21 million) from $PM_{2.5}$. The change in $PM_{2.5}$ -induced all-cause YLL over specific regions is shown in S4 in the SI. East Asia and South Asia have the most YLL due to the largest increase in premature mortality. Due to the much higher baseline YLL per death in Africa than North America (about 15 vs. 5 YLL per death), Africa has more YLL due to climate change induced changes in $PM_{2.5}$ than North America, despite North America having a larger relative increase in premature mortalities. YLL attributed to climate-induced changes in O_3 exposure is about 38,000 years, a relative increase of 0.5 %, much smaller than that due to changes in $PM_{2.5}$ exposure. The largest increases in YLL from O_3 occur over East Asia, South Asia and North America while YLL due to O_3 decrease over Africa and the Middle East (Table S4 in the SI).

The following is an example, per unit population, of the impact of $PM_{2.5}$ and climate change induced changes in $PM_{2.5}$ on YLL. For adults aged 30 and older, the YLL from all-cause mortality per 1,000 population is about 123 years (WHO 2008b). The additional all-cause YLL attributed to climate change induced increases in $PM_{2.5}$ exposure in the 21st century per 1,000 population aged 30 and older is about 4.5 years. Due to climate change, YLL associated with $PM_{2.5}$ exposure increases approximately 4 % relative to YLL from total $PM_{2.5}$ exposure at the end of the 20th century, resulting in approximately 2 months additional life lost per 1,000 persons globally.

4 Discussion and conclusions

We evaluate projected changes in premature mortality from the end of the 20th century to the end of the 21st century resulting from climate change induced changes in surface O_3 and $PM_{2.5}$. Our study is based on climate change driven by the relatively conservative SRES A1B emission scenario. Using the global coupled CCM (AM3) we first find that climate change leads to increases in global population-weighted annual $PM_{2.5}$ concentrations of $0.5 \mu\text{g}/\text{m}^3$ and global population-weighted H- O_3 concentrations (defined in each grid box as the average of the 6 months with highest monthly average daily 1-h maximum O_3) of 0.3 ppbv. We estimate associated premature mortality using concentration-response relationships obtained from the epidemiological literature (Jerrett et al. 2009; Krewski et al. 2009). Our results indicate that 21st century climate change increases global all-cause premature mortalities associated with $PM_{2.5}$ by approximately 100,000 deaths and respiratory disease mortality associated with O_3 by 6,300 deaths annually.

Premature mortality resulting from exposure to local pollution in high-population urban centers is likely underestimated in our model due to its relatively coarse resolution, a similar limitation as reported by Liu et al. (2009) and Anenberg et al. (2010). To partly offset this effect and indicate the magnitude of the climate effect on mortalities associated with air pollution, we also report the relative change in premature mortality as the percent change between “present” and “future” simulations. 21st century climate change, under the A1B scenario, results in about a 4 % increase in global all-cause mortality associated with $PM_{2.5}$, and less than a 1 % increase in respiratory disease mortality associated with O_3 .

Assuming population, baseline mortality rates and reactive air pollution emissions do not change over the 21st century, our sensitivity study indicates that climate change effects on air quality will increase premature mortality and YLL, suggesting the existence of an air pollution mediated “climate penalty” for health. Other climate-driven factors, not included in our simulations, may exacerbate this penalty. For example, biogenic hydrocarbon emissions increase with temperature and hence will likely increase with climate warming. As they are precursors of O₃ and organic aerosols, their increase will likely enhance O₃ and PM_{2.5} concentrations, particularly in high NO_x regions. In addition, we do not consider the impact on tropospheric chemistry of a likely CH₄ concentration increase of about 200 ppb during the 21st century. Based on previous literature (Fang et al. 2013b; West et al. 2006b; Fiore et al. 2008; Anenberg et al. 2012), this increase in CH₄ will likely raise background O₃ by about 1 ppb. The effect of population growth is also not included in our sensitivity study. If population grow, as indicated in the A1B or A2 scenarios, during the 21st century is considered, premature mortality associated with the climate change effect on PM_{2.5} will increase 4.4 % (A1B) or 11 % (A2) during the 21st century (details shown in the SI).

Significant uncertainties are associated with our sensitivity study. We assume that the relative risks derived from the U.S. ACS epidemiological study (Jerrett et al. 2009; Krewski et al. 2009) are applicable globally, as supported by similar relative risks established in U.S. based time-series studies and in various studies in Europe (Levy et al. 2005; Anderson et al. 2004) and Asia (HEI 2010). However, lifestyles, age distributions, health status, and medical resources vary around the world possibly resulting in varied health responses to the same change in pollution level. Nevertheless, this is unlikely to change the sign of the results associated with this sensitivity study.

Another source of uncertainty is the simulated O₃ and PM_{2.5} concentration changes driven by climate change. As an initial effort, we reviewed recent literature examining climate change effects on these two species. As summarized by Jacob and Winner (2009), there are still large uncertainties regarding model projections of future air quality, especially for PM_{2.5}. However, most studies cited in this review and other more recent papers (Kloster et al. 2010; Fang et al. 2011) show an increase in PM_{2.5} over populated areas due to climate change (See Table S5 in the SI). There is one study by Tagaris et al. (2007) that shows a 10 % decrease in annual mean PM_{2.5} over the United States, however, their companion paper (Tagaris et al. 2009) estimates increased mortality in the United States from PM_{2.5} exposure due to the coincidence of dense population and increases in PM_{2.5}. Therefore, these previous studies generally support our conclusion that climate change imposes a penalty to air quality and associated human mortality risks, especially over populated regions. We further examined the standard deviation for concentration and mortality changes by comparing individual year results in our 20-year simulation to assess uncertainties arising from internal model variability. Using PM_{2.5} as an example, we find global mortality associated with PM_{2.5} is 4.1±1.5 (as mean ± standard deviation) %, indicating that the increase in global mortality is fairly robust to internal model variability. However, some regions (such as the Middle East and Rest of Asia) have a standard deviation greater than the mean, indicating changes that are not significant from internal model variability.

In addition to these initial efforts, a systematic quantification of uncertainties associated with climate models and specific scenarios requires ensemble model studies, in which a variety of chemistry-climate models run the same sensitivity experiments that are then applied to examine the corresponding health effects associated with air pollution. Such ensemble model studies have been applied in Anenberg et al. (2009) to examine the health risks associated with inter-continental air pollution transport. Currently, there are on-going inter-model comparison projects (such as the Atmospheric Chemistry and Climate Model

Intercomparison Project (ACCMIP 2012)) which will provide data for improved estimates of uncertainties.

Our study shows climate change exacerbates air pollution and increases associated health risks globally, especially over polluted regions. This result is consistent with conclusions drawn from studies focused on the United States (Bell et al. 2007; Jacobson 2008; Tagaris et al. 2009). Our study suggests that in the future to improve air quality and protect human health, environmental policies that reduce emissions of greenhouse gases and warming aerosols, as well as traditional air pollutants, will be beneficial. Reducing emissions of black carbon and methane would be particularly effective as mitigation of these pollutants can slow the rate of climate change while reducing concentrations of PM_{2.5} and O₃ and their associated health risks (Shindell et al. 2012; Anenberg et al. 2012; Bond et al. 2013).

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