Air pollution and associated human mortality: the role of air pollutant emissions, climate change and methane concentration increases from the preindustrial period to present

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Title:**Air pollution and associated human mortality: the role of air pollutant emissions, climate change and methane concentration increases from the preindustrial period to present**

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Abstract. Increases in surface ozone (O_3) and fine particulate matter (≤ 2.5 µm aerodynamic diameter, $PM_{2.5}$) are associated with excess premature human mortalities. We estimate changes in surface O_3 and $PM_{2.5}$ from pre-industrial (1860) to present (2000) and the global present-day (2000) premature human mortalities associated with these changes. We extend previous work to differentiate the contribution of changes in three factors: emissions of short-lived air pollutants, climate change, and increased methane (CH_4) concentrations, to air pollution levels and associated premature mortalities. We use a coupled chemistry-climate model in conjunction with global population distributions in 2000 to estimate exposure attributable to concentration changes since 1860 from each factor. Attributable mortalities are estimated using health impact functions of long-term relative risk estimates for O_3 and $PM_{2.5}$ from the epidemiology literature. We find global mean surface $PM_{2.5}$ and health-relevant O_3 (defined as the maximum 6-month mean of 1-h daily maximum O_3 in a year) have increased by $8 \pm 0.16\,\mu g\,m^{-3}$ and 30 ± 0.16 ppbv (results reported as annual average $\pm$standard deviation of 10-yr model simulations), respectively, over this industrial period as a result of combined changes in emissions of air pollutants (EMIS), climate (CLIM) and CH_4 concentrations (TCH4). EMIS, CLIM and TCH_4 cause global population-weighted average $PM_{2.5}$ (O_3) to change by $+7.5 \pm 0.19\,\mu g\,m^{-3}$ ($+25 \pm 0.30$ ppbv), $+0.4 \pm 0.17\,\mu g\,m^{-3}$ ($+0.5 \pm 0.28$ ppbv), and $0.04 \pm 0.24\,\mu g\,m^{-3}$ ($+4.3 \pm 0.33$ ppbv), respectively. Total global changes in $PM_{2.5}$ are associated with 1.5 (95 % confidence interval, CI, 1.2–1.8) million cardiopulmonary mortalities and 95 (95 % CI, 44–144) thousand lung cancer mortalities annually and changes in O_3 are associated with 375 (95 % CI, 129–592) thousand respiratory mortalities annually. Most air pollution mortality is driven by changes in emissions of short-lived air pollutants and their precursors (95 % and 85 % of mortalities from $PM_{2.5}$ and O_3 respectively). However, changing climate and increasing CH_4 concentrations also contribute to premature mortality associated with air pollution globally (by up to 5 % and 15 %, respectively). In some regions, the contribution of climate change and increased CH_4 together are responsible for more than 20 % of the respiratory mortality associated with O_3 exposure. We find the interaction between climate change and atmospheric chemistry has influenced atmospheric composition and human mortality associated with industrial air pollution. Our study highlights the benefits to air quality and human health of CH_4 mitigation as a component of future air pollution control policy.

1 Introduction

Human activities since preindustrial time have resulted in large increases in air pollution (IPCC, 2001). Measurements at various sites in the Northern Hemisphere indicate an increase from the 1860s to 2000s in surface ozone (O_3) of approximately a factor of 4 (from about 10 to 50 ppbv) (Gros, 2006; Marenco et al., 1994). Sulfate aerosol concentrations in Greenland ice cores suggest a factor of 3–4 increase from the mid-1860s to the present (Döscher et al., 1995; Fischer et al., 1998). Over the same period, European high-alpine glaciers indicate an increase in carbonaceous aerosols of a factor of 3 (Lavanchy et al., 1999), while Greenland ice-cores show little change (Lamarque et al., 2010). Sulfate and carbonaceous aerosols are key components of fine particulate matter (≤ 2.5 μm aerodynamic diameter, $PM_{2.5}$), which, along with O_3, are pollutants that adversely impact human health (Bell et al., 2004; Jerrett et al., 2009; Krewski et al., 2009; Levy et al., 2005; Pope et al., 2002; Pope and Dockery, 2006). Here, we apply simulations of a global atmospheric chemistry-climate coupled model to investigate changes in O_3 and $PM_{2.5}$ from the preindustrial era to the present and their associated effects on premature mortality.

O_3 is a secondary air pollutant that is formed in the troposphere by catalytic photochemical reactions of nitrogen oxides ($NO_x = NO + NO_2$) with carbon monoxide (CO), methane (CH_4) and other volatile organic compounds (VOCs). $PM_{2.5}$, including sulfate, nitrate, organic carbon (OC), black carbon (BC), secondary organic aerosol (SOA), fine dust and sea salt, is either directly emitted from various sources or produced via chemical reactions between directly-emitted gas-phase precursors (including SO_2, NO_x, NH_3, biogenic VOCs etc.) and atmospheric oxidants (i.e., OH, H_2O_2, O_3). Changes in O_3 and $PM_{2.5}$ concentrations from the preindustrial period to the present (1860–2000) are difficult to quantify because of sparse and uncertain preindustrial measurements, spatial heterogeneity of these species, uncertainties in estimating preindustrial emissions, and the nonlinear dependence of O_3 and $PM_{2.5}$ on their precursor emissions (Horowitz, 2006).

Changes in surface O_3 and $PM_{2.5}$ concentrations are largely controlled by changes in emissions of their precursors. Consequently, many recent studies have applied chemical transport models (CTMs) to estimate changes in tropospheric O_3 and aerosol concentrations from the preindustrial era to the present (Grenfell et al., 2001; Horowitz, 2006; Lamarque et al., 2005; Mickley et al., 2001; Tsigaridis et al., 2006; Wang and Jacob, 1998). Anenberg et al. (2010) used preindustrial and present simulations from one of these CTM modeling studies (Horowitz, 2006) to estimate the effect of anthropogenic O_3 and $PM_{2.5}$ on present premature human mortality. However, these studies, which usually apply different emissions of short-lived species but use the same meteorological driver for preindustrial and present day simulations, do not take into account the interaction between climate and air pollution (Jacob and Winner, 2009; Isaksen et al., 2009; Fiore et al., 2012). Some short-lived species are radiatively active; therefore, they perturb climate and meteorology from regional to global scales (Naik et al., 2013; Levy et al., 2008; Shindell et al., 2008). As a result, quantifying the impact of their emission changes on air quality using CTM simulations driven by the same meteorology neglects the feedbacks between short-lived species and climate. Conversely, studies have shown that climate change can affect surface O_3 and $PM_{2.5}$ concentrations and thus indirectly affect human mortality (Bell et al., 2007; Fang et al., 2013; Tagaris et al., 2009). Additionally, CH_4 concentration changes (from 800 ppbv in 1860 to 1750 ppbv in 2000) not only give a direct radiative forcing of $+0.42\ W\ m^{-2}$ (calculated as in Ramaswamy et al., 2001), but also contribute to increasing O_3 concentrations which indirectly changes climate (Shindell et al., 2009). To understand changes in surface O_3 and $PM_{2.5}$ over the industrial period (defined here as 1860–2000), we need to consider the effects of changing emissions of short-lived species, climate and CH_4 concentrations on surface air quality and allow feedbacks between chemistry and climate to take place.

In this paper, we utilize the Geophysical Fluid Dynamics Laboratory (GFDL) Atmospheric Model, version 3 (AM3), a newly developed global 3-D model that fully couples atmospheric chemistry and climate. Our goal is to understand changes in O_3 and $PM_{2.5}$ from the preindustrial era to the present ("industrial" or "historic" period) and their associated effects on premature mortality. We further attribute the changing $PM_{2.5}$ and O_3 concentrations over this period to three factors: (1) changes in direct emissions of their constituents and precursors; (2) climate change induced changes in surface concentrations, and (3) the influence of increasing CH_4 concentrations on tropospheric chemistry. For each factor, we estimate the associated impact on human health. The GFDL AM3 model and our simulations are described in Sect. 2. We evaluate simulated surface O_3 and $PM_{2.5}$ concentrations in Sect. 3. Changes in surface air quality are attributed to specific factors in Sect. 4. In Sect. 5, we calculate the changes in premature mortality associated with the simulated changes in air quality. Findings and conclusions are presented in Sect. 6.

2 Methods

2.1 Model description

The AM3 model (Donner et al., 2011) is the atmospheric component of the GFDL atmosphere-ocean coupled climate model CM3. AM3 is designed to address key emerging issues in climate science, including aerosol-cloud interactions and chemistry-climate feedbacks. It is GFDL's first global atmospheric model to include the indirect effects of cloud-aerosol interactions (with 16 interactive aerosol species) and

Table 1. Model simulation configurations. All simulations are run for 11 yr with the first year used for spin-up (SST: sea surface temperature; SIC: sea ice; WMGG: well-mixed greenhouse gases; ODS: ozone-depleting substances).

Simulations		SST and SIC	WMGG	ODS[a]	CH_4 (tropospheric chemistry)	Anthropogenic and biomass burning emissions
1	2000[b]	2000	2000	2000	2000	2000
2	1860[b]	1860	1860	1860	1860	1860
3	2000CL1860EM	2000	2000	2000	2000	1860
4	1860CL2000EM[b]	1860	1860	1860	2000	2000
5	1860ALL2000EM	1860	1860	1860	1860	2000

[a] The concentrations of ODS in the 1860 simulation are set to pre-1950 levels.
[b] Simulations run for the Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP).

of tropospheric and stratospheric chemistry (with 81 gas species) coupled with climate. Detailed chemistry, emissions, and deposition processes in AM3 are described in Naik et al. (2013) with additional details described below, and transport (advection, vertical diffusion and convection) along with physics are described in Donner et al. (2011). The model uses a finite-volume dynamical core with a $6 \times 48 \times 48$ cubed-sphere horizontal grid with the grid size varying from 163 km (at the corners of each face) to 231 km (near the center of each face). Vertically, the model extends from the surface up to 0.01 hPa (86 km) with 48 vertical hybrid sigma pressure levels.

2.2 Adverse health impacts

We analyze the effect of changes in air pollution concentrations during the industrial period ($\sim$1860 to $\sim$2000) on premature mortality using health impact functions that relate changes in air pollutant concentrations to changes in mortality. We further evaluate the relative importance of changes in emissions of air pollutants, climate change and increased CH_4 concentrations on air pollution concentrations and the associated incidence of premature mortalities.

To obtain estimates of the excess mortalities (ΔMort) attributable to air pollution changes during the industrial period, we use health impact functions for O_3 and $PM_{2.5}$. These functions are based on log-linear relationships between relative risk and concentration derived from the American Cancer Society (ACS) cohort studies for adults aged 30 and older (Jerrett et al., 2009; Krewski et al., 2009; Pope et al., 2002). We apply

$$\Delta\text{Mort} = \text{POP} \times \text{Frac} \times \text{Mort}_{\text{base}} \times (1 - e^{-\beta \Delta C}) \quad (1)$$

in each of the AM3 surface grid cells and separately calculate changes in mortality associated with changes in $PM_{2.5}$

3. Impact of increased CH_4 concentration

a primary sink of atmospheric OH. Higher CH_4 concentrations in 2000 than 1860 result in an OH decrease of $0.24 \pm 0.01 \times 10^6$ molec cm^{-3} and an increase of $0.35 \pm 0.03 \times 10^{10}$ molec cm^{-3} in H_2O_2 (Table 3). As OH and H_2O_2 are associated with the gas-phase and in-cloud production of sulfate, changing CH_4 thus indirectly influences $PM_{2.5}$. Compensating changes in OH and H_2O_2 lead to a small and insignificant global change in $PM_{2.5}$ (global population-weighted $PM_{2.5}$ decreases by $0.04 \pm 0.24\,\mu g\,m^{-3}$, Table 3). The spatial pattern of $PM_{2.5}$ changes driven by the impact of increased CH_4 concentration is also not correlated with its total change during the industrial period.

CH_4 increases (from 800 ppbv in 1860 to 1750 ppbv in 2000) result in an increase in the global population-weighted H-O_3 concentration of 4.3 ± 0.33 ppbv (Table 3, TCH4), accounting for almost 15 % of the total H-O_3 produced during the industrial period. The fraction that CH_4 contributes to total H-O_3 produced during the industrial period is much higher (22 % over land) if it is weighted by area. The distribution of surface O_3 enhancement driven by increased CH_4 is significant everywhere in the world and is approximately 5–10 ppbv in the Northern Hemisphere and 2–5 ppbv in the Southern Hemisphere (Fig. 3j). A spatial correlation of 0.7 between changes in surface H-O_3 driven by increased CH_4 and that driven by all factors supports total O_3 changes being partly driven by CH_4. Although the impact of CH_4 on O_3 has been discussed in previous literature (Dentener et al., 2005; Fiore et al., 2002, 2008; West et al., 2006), most of these studies focus on the potential benefit of future CH_4 mitigation while our study examines the total change in O_3 resulting from historic increases in CH_4. However, the magnitude of CH_4 impact on O_3 in those studies is consistent with ours: for example, Fiore et al. (2008) estimate that anthropogenic CH_4 emissions contribute 5 ppbv to global mean surface O_3; West et al. (2006) and Anenberg et al. (2009) find that a 20 % reduction in anthropogenic CH_4 and a 20 % reduction in global CH_4 mixing ratio lead to approximately a 1 ppbv decrease in global mean surface O_3.

year (Table 4); while industrial O_3 is associated with 0.37 (95 % CI, 0.13–0.59) million respiratory mortalities per year (Table 4). Our estimates suggest that about 1.5 million cardiopulmonary and 95 thousand lung cancer mortalities associated with $PM_{2.5}$ exposure, and 0.37 million respiratory mortalities associated with O_3 exposure would have been avoided in 2000 if surface $PM_{2.5}$ and O_3 had remained at 1860 levels (i.e., anthropogenic and biomass burning emissions of air pollutants, CH_4 and climate had all remained the same in 2000 as they were in 1860). Our estimated mortalities associated with industrial $PM_{2.5}$ and O_3 are considerably lower than those in Anenberg et al. (2010), which is consistent with differences between our emission scenarios and calculated surface concentrations and theirs, as discussed in Sect. 4.1. If we apply a low concentration threshold (LCT) of $5.8\,\mu g\,m^{-3}$ $PM_{2.5}$ and 33.3 ppbv O_3 (the lowest values in the ACS studies), premature mortalities associated with industrial $PM_{2.5}$ and O_3 are 15 % and 11 % lower, respectively. These relative differences are smaller here than in Anenberg et al. (2010) (33 % and 28 %, respectively for mortalities associated with $PM_{2.5}$ and O_3) because our preindustrial emissions and hence simulated preindustrial O_3 and $PM_{2.5}$ are higher than theirs (see Sect. 4.1 and Table S1). As differences with and without use of the LCT are relatively small in our study, we hereafter only report mortalities without the LCT.

We separate the world into 10 regions as in Liu et al. (2009b) and Fang et al. (2013) to estimate the regional mortalities associated with industrial air pollution. The regional distribution of premature mortality associated with industrial $PM_{2.5}$ and O_3 is shown in Fig. 5. Eastern China and northern India are hotspots for air pollution mortalities, driven by their large increases in surface $PM_{2.5}$ and O_3 concentrations and their large populations. East Asia accounts for 43 % (56 %) of the global cardiopulmonary (lung cancer) mortalities associated with industrial $PM_{2.5}$ and 50 % of the global respiratory mortalities associated with industrial O_3. South Asia is second, accounting for 27 % (16 %) of the global cardiopulmonary (lung cancer) mortalities associated with industrial $PM_{2.5}$ and 19 % of the global respiratory mortalities associated with industrial O_3. None of the other regions contribute over 15 % to the global mortalities associ-

Table 4. Premature mortalities in 2000 associated with industrial air pollution. Values are calculated as in Eq. (1), using ACS health impact functions, concentration difference in annual $PM_{2.5}$ and H-O_3 between "1860" and "2000" simulations, WHO baseline mortality rate and population in the year 2000. The 95 % confidence intervals are shown in brackets.

Regions	Change in Premature mortalities (1000s deaths)		
	$PM_{2.5}$ mortality (Chronic, cardiopulmonary)	$PM_{2.5}$ mortality (Chronic, lung cancer)	O_3 mortality (Chronic, respiratory)
World	1532 (1214, 1832)	95 (44, 144)	375 (129, 592)
North America	38 (30, 46)	4.4 (2.0, 6.9)	26 (9, 41)
South America	15 (12, 18)	0.8 (0.4, 1.3)	5 (2, 8)
Europe	125 (99, 152)	8.2 (3.7, 12.7)	31 (11, 49)
Africa	77 (61, 93)	2.1 (0.9, 3.2)	19 (6, 30)
South Asia	417 (331, 499)	15 (7, 23)	70 (24, 111)
Southeast Asia	108 (86, 130)	6.9 (3.2, 10.5)	27 (9, 43)
East Asia	661 (527, 788)	53 (25, 79)	183 (64, 287)
Middle East	52 (41, 63)	2.6 (1.2, 4.0)	7.5 (2.6, 12.0)
Rest of Asia	29 (23, 35)	1.4 (0.6, 2.3)	5.5 (2.6, 8.8)
Australia	0.7 (0.5, 0.8)	0.1 (0.05, 0.2)	0.3 (0.1, 0.4)

each factor with that associated with the three factors together for each region and globally (the number of mortalities in each region associated with each factor is summarized in Table S3 of the Supplement). To provide a quantitative assessment of their relative importance, for each region (i), a normalized mortality contribution (NMC) is defined in the following way:

$$\mathrm{NMC}_i = \frac{\text{Mortality response driven by one factor}}{\text{Mortality response driven by all factors}} \quad (2)$$

and analyzed for each factor in the following discussion.

We first estimate the global mortality response associated with industrial $PM_{2.5}$ and O_3 pollution resulting from changes in air pollutant emissions only (2000 – 2000CL1860EM, EMIS). We find that if air pollutant emissions in year 2000 had remained at 1860 levels, 1.49 (95 % CI, 1.18–1.79) million cardiopulmonary mortalities, 92 (95 % CI, 43–140) thousand lung cancer mortalities associated with $PM_{2.5}$ exposure and 0.33 (95 % CI, 0.11–0.52) million respiratory mortalities associated with O_3 exposure could have been avoided.

We next estimate the global mortality response associated with industrial $PM_{2.5}$ and O_3 pollution resulting from climate change (and stratospheric O_3 depletion) (2000 – 1860CL2000EM). We find that if climate and stratospheric O_3 in 2000 were the same as in 1860, about 91 (95 % CI, 71–110) thousand cardiopulmonary mortalities, 5 (95 % CI, 2–8) thousand lung cancer mortalities associated with $PM_{2.5}$ exposure and 7 (95 % CI, 2–12) thousand respiratory mortalities associated with O_3 exposure could have been avoided.

Then, we evaluate the effects of CH_4 concentration increases on global premature mortalities associated with air pollution. To be entirely consistent with the previous comparisons, we would compare $PM_{2.5}$ and O_3 in simulation 2000 with that in a simulation identical to simulation 2000 except with the lower boundary condition of global CH_4 concentration specified at 1860 instead of 2000 levels in its tropospheric chemistry calculation. Unfortunately, such a simulation is not available. However, two of our available simulations (1860CL2000EM and 1860ALL2000EM), although simulating 1860 climate, differ only in their treatments of CH_4 in tropospheric chemistry. This allows us to estimate the effect of increased CH_4 on premature mortality due to O_3 and $PM_{2.5}$ exposure. We assume the bias due to the difference between 1860 and 2000 climates is small. Our results suggest that if CH_4 had remained at 1860 levels (about 950 ppbv less than the year 2000 level), about 50 (95 % CI, 17–82) thousand respiratory mortalities would have been avoided due to lower O_3 concentrations resulting from less CH_4. Our estimated O_3 mortalities associated with industrial CH_4 increase is comparable with previous estimates in the literature that focused on the health benefits of CH_4 mitigation. For example, both West et al. (2006) and Anenberg et al. (2009) find a 20 % reduction in anthropogenic CH_4 emissions or a 20 % reduction in global CH_4 concentrations (both equivalent to about 300 ppbv CH_4 concentration reduction) would reduce cardiopulmonary mortalities by 16–17 thousand.

We finally assess the relative importance of each factor to air pollution mortality regionally and globally. Figure 6 shows NMC for each region. Due to the non-linearity in the health impact function, chemistry, and chemistry-climate system, the value of each bar in Fig. 6 is close to, but not exactly, 1.

Global premature mortality associated with industrial $PM_{2.5}$, is dominated by increased emissions of reactive air pollutants (~ 95 %), however, climate change is influential with a global NMC of ~ 5 %. Regionally, contributions of climate change to cardiopulmonary and lung cancer mortality associated with industrial $PM_{2.5}$ can be as high as 14 % with the highest values over Europe and Australia. The

4. Discussion and conclusions

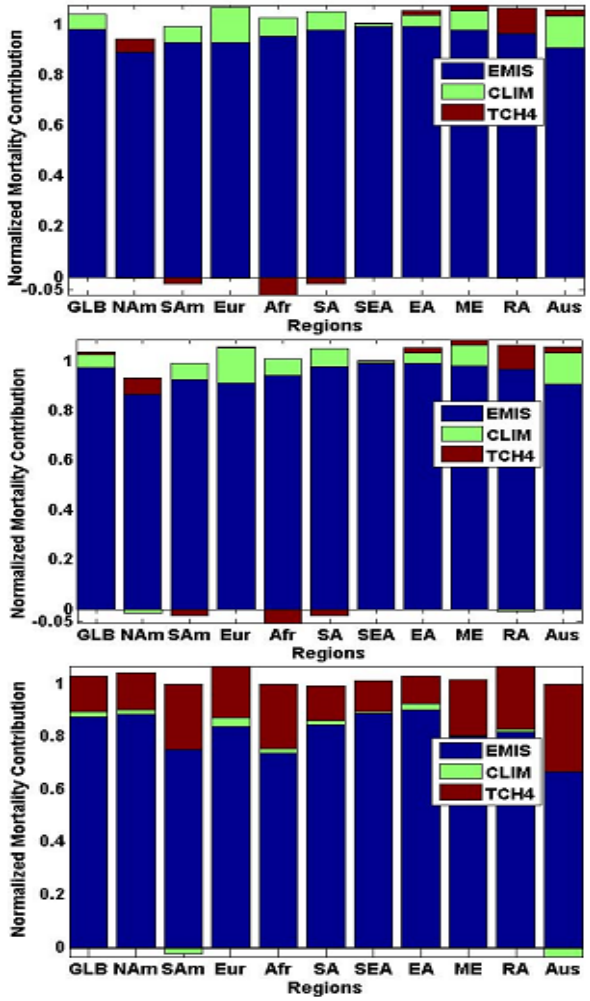

Fig. 6. Normalized Mortality Contribution of **(a)** cardiopulmonary and **(b)** lung cancer associated with surface $PM_{2.5}$ and of **(c)** respiratory disease associated with O_3 over each region. Blue, green and dark red represent the impact of changes in emissions of short-lived species, climate change and the impact of CH_4 change on tropospheric chemistry, respectively. Labels on the x-axis represent the following regions: Global, North America, South America, Europe, Africa, South Asia, Southeast Asia, East Asia, Middle East, Rest of Asia, and Australia, as in Fang et al. (2013).

India and China suffer most from industrial air pollution mortality as they have experienced strong increases in air pollution levels and have large exposed populations.

We further evaluate the relative importance of changes in emissions of short-lived species, climate, and CH_4 concentrations in driving changes in $PM_{2.5}$, O_3 and associated premature mortalities.

We find that increases in short-lived air pollutant emissions from 1860 to 2000 lead to $7.5 \pm 0.19\,\mu g\,m^{-3}$ and 25 ± 0.30 ppbv increases in $PM_{2.5}$ and H-O_3 concentrations respectively, accounting for a majority (94 % and 83 %) of total increases in these two species over this period. Changes in emissions of short-lived pollutants account for over 95 % of cardiopulmonary or lung cancer mortalities associated with industrial $PM_{2.5}$ and over 85 % of respiratory mortalities associated with industrial O_3.

CH_4 concentration increase is the second most important driver of H-O_3 increases, causing an increase of 4.3 ± 0.33 ppbv and accounting for almost 15 % of the total increase of H-O_3 from 1860 to 2000. CH_4 contributes nearly 15 % of the total respiratory mortalities associated with industrial O_3. CH_4 has negligible effects on $PM_{2.5}$ and has an insignificant effect on lung cancer or cardiopulmonary mortalities associated with $PM_{2.5}$.

Changing climate has a small role in driving O_3 change during the industrial period, causing an increase of 0.5 ± 0.28 ppbv in global H-O_3 (2 % of the total increase) from 1860 to 2000. Climate change is the second most important driver of changes in $PM_{2.5}$ during the industrial period, causing an increase of $0.4 \pm 0.17\,\mu g\,m^{-3}$ (5 % of its total increase) from 1860 to 2000. The effect of climate change on industrial air pollution mortalities is small but non-negligible for both $PM_{2.5}$ and O_3, accounting for < 5 % and ~ 2 % of changes in mortality associated with these species, respectively.

The contribution of climate change and CH_4 concentration increases together to excess mortalities over various regions ranges from 1 % to 14 % for cardiopulmonary and lung cancer mortality associated with industrial $PM_{2.5}$ and from 8 % to 33 % for respiratory mortality associated with industrial O_3. Increased CH_4 concentrations alone contribute more than 20 % to respiratory mortalities associated with industrial O_3 exposure over South America, Europe, Africa, Middle East and Rest of Asia. Recent projections indicate that over Europe and the United States local O_3 precursor emissions are likely to continue to decrease after 2000 (Richter et al., 2005; van der A et al., 2008). In the meantime, CH_4 is projected to increase in almost all SRES (Nakicenovic et al., 2000) and RCP (Meinshausen et al., 2011) emission scenarios (except RCP2.6 and SRES B2). As a result, the relative contribution of increased CH_4 to O_3 mortality will likely continue to rise, increasing the relative health benefits of CH_4 mitigation.

As the benefit of CH_4 reduction does not depend on its location, for cleaner regions, such as Europe, South America and Australia (where we find mortality burdens are more sensitive to CH_4 concentrations than other regions), identifying low-cost CH_4 mitigation options internationally may be an

effective method of reducing local premature mortalities associated with O_3 exposure. Our study highlights the benefits of controlling CH_4 emissions as part of air quality policy.

Many opportunities to mitigate CH_4 are available (UNEP, 2011). Anenberg et al. (2012) examined potential measures and showed that, relative to the 2030 reference scenario and population, implementing available CH_4 measures would avoid around 70 thousand deaths due to respiratory disease associated with O_3 exposure. We estimate industrial O_3 respiratory mortalities to be approximately 375 thousand in the year 2000 (Table 3), This suggests that, with currently available CH_4 mitigation measures, respiratory mortalities associated with industrial O_3 pollution could be reduced by nearly 20 %.

Modeling estimates of industrial air pollution and associated excess mortalities strongly depend on emission changes applied during this period, as reflected by differences between this study and Anenberg et al. (2010). They also depend on simulated physical, dynamical and chemical processes in the atmosphere. To evaluate the robustness of our results, similar studies using different chemistry-climate models could be conducted. Many of the simulations applied in this study were conducted under the ACCMIP Project. Multiple modeling groups have participated in this project, all of which use the same emission inventories and run simulations for preindustrial (1860) and present (2000) (http://www.giss.nasa.gov/projects/accmip/specifications.html). Further analysis of the ACCMIP simulations (http://www.atmos-chem-phys-discuss.net/special_issue176.html) could reduce uncertainties in modeling estimates of industrial air pollution and associated mortalities.