Response to Referees' Comments:

We thank three Referees for their helpful and constructive comments. We have made substantial improvements to the paper in responding to each comment. The reviewer comments are shown below in italics, followed by our responses to each point in blue.

***Anonymous Referee #1***

*This is a valuable analysis on an important topic that takes advantage of a detailed model ensemble to address air pollution mortalities. However, the work has a few major deficiencies which ought to be addressed in a revision, as well as several minor comments.*

*1) The paper falls short in putting the work in the context of previous efforts. It cites only two previous papers assessing global health-related impacts of future air pollutants, while there is in fact a larger literature, including both global and regional effects. By not putting their work in the context of the previous work, the authors overstate the novelty of their contribution, and are not able to discuss their work in the context of what is known or unknown in this field.*

We have expanded the number of studies we reference to include more global studies and now include regional studies. While there have been many studies to assess air pollution health effects, the number of global studies that explore future scenarios remains limited. The revised text (p. 2, line 27, to p. 3 line 2) is:

"Previous studies have estimated the present-day global burden of disease due to exposure to ambient ozone and/or PM2.5 (e.g., Apte et al., 2015; Evans et al., 2013; Forouzanfar et al., 2015), with several studies estimating this burden using only output of global atmospheric models (Anenberg et al., 2010; Fang et al., 2013a; Lelieveld et al., 2013; Rao et al., 2012; Silva et al., 2013). However, few studies have evaluated how the global burden might change in future scenarios (Lelieveld et al., 2015; Likhvar et al., 2015; West et al., 2007). Other global studies have estimated future air pollution-related mortality as a by-product of analyses of other future changes, such as the effects of climate change or of climate change mitigation (e.g., Fang et al., 2013b; Selin et al., 2009; West et al., 2013), but do not focus on the range of plausible future mortality as their main purpose. Similarly, studies at local and regional scales have evaluated the mortality impact of changes in air quality due to future climate change (Bell et al., 2007; Chang et al., 2010; Fann et al., 2015; Heal et al., 2012; Jackson et al., 2010; Knowlton et al., 2004, 2008; Orru et al., 2013; Post et al., 2012; Sheffield et al., 2011; Tagaris et al., 2009) but few such studies have evaluated changes beyond 2050."

*2) The paper does not fully take advantage of the potential utility of using a multi-model ensemble in the analysis. As is, it presents straightforward calculations of resulting mortalities, which (for the most part) are very predictable based on the air quality results presented in previous work. The authors do have some very interesting results about the relative contributions of different assumptions of air pollution concentrations, exposure-response functions, population, etc. While some of these results are noted, in my opinion, they are the most interesting implications of this analysis and could be*

*highlighted. However, the authors fall short in this area by not correctly characterizing the uncertainties and variabilities captured by their use of the RCP scenarios (a significant limitation which could be more thoroughly discussed and caveated) and the ACCMIP effort. These ought to be discussed more carefully.*

Taking into account the referee's comments, we have revised the Abstract, Introduction and Discussion sections:

Abstract, (p. 2, line 17): "Mortality estimates differ among chemistry-climate models due to differences in simulated pollutant concentrations, which is the greatest contributor to overall mortality uncertainty for most cases assessed here, supporting the use of model ensembles to characterize uncertainty. Increases in exposed population and baseline mortality rates of respiratory diseases magnify the impact on premature mortality of changes in future air pollutant concentrations and explain why the future global mortality burden of air pollution can exceed the current burden, even where air pollutant concentrations decrease."

Introduction (p. 3, lines 33-37): "All RCPs assume increasingly stringent air pollution controls as countries develop economically, leading to decreases in air pollutant emissions that reflect the different methods of the different RCP groups (e.g., Smith et al., 2011). But as assumptions are similar among the RCPs, the four scenarios do not span the range of possible futures published in the literature for short-term species. For example, other studies have simulated scenarios in which air pollution controls are kept at current levels while underlying trends (e.g., energy use) increase overall emissions (Lelieveld et al. 2015; Likhvar et al. 2015)."

Discussion (p. 12, lines 7-17): "The importance of conducting health impact assessments with air pollutant concentrations from model ensembles, instead of from single models, is highlighted by the differences in sign of the change in mortality among models, and by the marked impact of the spread of model results on overall uncertainty in our mortality estimates. In most cases assessed here (ozone mortality in 2030 relative to 2000, PM2.5 mortality in 2030, 2050 and 2100 relative to 2000), uncertainty in modeled air pollutant concentrations is the greatest contributor to uncertainty in mortality estimates. The differences in air pollutant concentrations reported by the ACCMIP models reflect different treatments of atmospheric dynamics and chemistry, chemistry-climate interactions, and natural emissions in each model (Young et al., 2013). Although there is likely a bias in estimating health effects using air pollutant concentrations from coarse resolution models (Li et al., 2015; Punger and West, 2013), particularly for PM2.5, we do not expect resolution to be an important factor for the differences in simulated concentrations across these coarse resolution global models."

Discussion (p. 13, lines 1-11): "Our results are limited by the range of air pollutant emissions projected by the RCPs, which assume that economic growth strengthens efforts to reduce air pollutant emissions. All RCPs project reductions in anthropogenic precursor emissions associated with more extensive air quality legislation as incomes rise, except for methane in RCP8.5 and for ammonia in all scenarios. These scenarios together do not encompass the range of plausible air pollution futures for the 21st century, as the RCPs were not designed for this purpose (van Vuuren et al., 2011a). Other plausible scenarios have been considered, such as the Current Legislation Emissions and Maximum Feasible Reductions scenarios used by Likhvar et al. (2015) and the Business-As-Usual scenario of Lelieveld et al (2015). As noted above, our global burden estimates for 2050 are considerably lower than the Business-As-Usual scenario of Lelieveld et al. (2015). If economic growth does not lead to stricter air pollution

control, emissions and health effects may rise considerably, particularly for scenarios of high population growth in developing countries (Amman et al., 2013)."

*3) The mortality numbers, while interesting, are not put in proper context such that the reader can understand what they mean. To address this, some comparison with existing literature could be very useful.*

To provide context for our estimates of the future global burden of air pollution on mortality, we estimated the present-day burden and compared with existing literature (Forouzanfar et al. 2015, Silva et al. 2013). Please see Results section, p. 10, lines 28-37):

"For context, we estimate the present-day global burden, using 2000 concentrations, population from Landscan 2011 Population Dataset, and baseline mortality rates from GBD2010, to be 382,000 (121,000 to 728,000) ozone deaths/year and 1.70 (1.30 to 2.10) million PM2.5 deaths/year. These estimates are 18.7% lower for ozone-related mortality and 19.1% lower for PM2.5 mortality than those obtained in our previous study (Silva et al., 2013), reflecting: a) more restrictive mortality outcomes (chronic respiratory diseases rather than all respiratory diseases, and IHD+STROKE+COPD rather than all cardiopulmonary diseases); b) updated population and baseline mortality rates; c) the use of the recent IER model (Burnett et al., 2014) for PM2.5 (instead of Krewski et al., 2009). Compared with the GBD 2013 (Forouzanfar et al. 2015), our estimates are 76% higher for ozone-related mortality and 42% lower for PM2.5-related mortality, likely due to the fact that we estimate the global mortality burden using 1850 concentrations as baseline, while Forouzanfar et al. (2015) consider counterfactual concentrations (theoretical minimum-risk exposure) that are mostly higher for ozone (uniform distribution between 33.3 and 41.9 ppb) and lower for PM2.5 (uniform distribution between 5.9 and 8.7 μg/m3) than 1850 concentrations. In addition, we consider ozone mortality from all chronic respiratory diseases while Forouzanfar et al. (2015) only account for COPD, and we restrict our mortality estimates to adult population while Forouzanfar et al. (2015) include PM2.5 mortality from lower respiratory tract infections in children under 5 years old."

Additionally, we compare our estimates of the global burden of PM2.5 mortality in 2050 with those reported by Lelieveld et al. 2015. While other studies of future air pollution mortality exist, large differences in scenarios and methods make a comprehensive comparison difficult. Please see Results section, p. 11, lines 21-25:

"Our estimates for the global burden of PM2.5 mortality in 2050 (between 1.82 and 3.50 million deaths/year for the four RCPs) are considerably lower than those of Lelieveld et al. (2015) (5.87 million deaths / year for IHD+STROKE+COPD+LC), likely due to the assumption in the RCP scenarios of further regulations on air pollutants, while the Business-As-Usual scenario of Lelieveld et al. (2015) does not assume regulations beyond those currently defined."

*In addition to these major comments, there are several areas in which analyses are not fully described, and/or relevant methods-related information is missing. These are noted below.*

*Minor comments follow:*
*p3, line 30-31: "few studies have evaluated how the global burden might change in future scenarios" and this seems like a small slice of the literature. There are other*

*papers that could be cited here.*

As mentioned above, we have revised the paragraph to include other literature (p. 2, line 27, to p. 3, line 2):
"Previous studies have estimated the present-day global burden of disease due to exposure to ambient ozone and/or PM2.5 (e.g., Apte et al., 2015; Evans et al., 2013; Forouzanfar et al., 2015), with several studies estimating this burden using only output of global atmospheric models (Anenberg et al., 2010; Fang et al., 2013a; Lelieveld et al., 2013; Rao et al., 2012; Silva et al., 2013). However, few studies have evaluated how the global burden might change in future scenarios (Lelieveld et al., 2015; Likhvar et al., 2015; West et al., 2007). Other global studies have estimated future air pollution-related mortality as a by-product of analyses of other future changes, such as the effects of climate change or climate change mitigation (e.g., Fang et al., 2013b; Selin et al., 2009; West et al., 2013), but do not focus on the range of plausible future mortality as their main purpose. Similarly, studies at local and regional scales have evaluated the mortality impact of changes in air quality due to future climate change (Bell et al., 2007; Chang et al., 2010; Fann et al., 2015; Heal et al., 2012; Jackson et al., 2010; Knowlton et al., 2004, 2008; Orru et al., 2013; Post et al., 2012; Sheffield et al., 2011; Tagaris et al., 2009) but few such studies have evaluated changes beyond 2050."

*p3, line 26-27: "RCPs… do not span the range of possible futures published in the literature for short-term species." This is a key point and it could be highlighted.*

We have strengthened the discussion of this point in responding to major comment #2 above.

"All RCPs assume increasingly stringent air pollution controls as countries develop economically, leading to decreases in air pollutant emissions that reflect the different methods of the different RCP groups (e.g., Smith et al., 2011). But as assumptions are similar among the RCPs, the four scenarios do not span the range of possible futures published in the literature for short-term species. For example, some other studies have simulated scenarios in which air pollution controls are kept at current levels while underlying trends (e.g., energy use) increase overall emissions (Lelieveld et al. 2015; Likhvar et al. 2015)."

*p4, line 4-6: but the ACCMIP is coarser. The mortality estimates thus should be justified. Also, line 33-34 on same page: this regridding to a scale finer than that modeled should be better described and justified.*

Here we regrid each model to a much finer resolution (0.5°x0.5°). We select a resolution that is much finer than any model to limit errors associated with regridding. For each individual model, the fact that the results were regridded to a finer resolution should not influence the results. For the multi-model ensemble, however, our regridding takes maximum advantage of how the different model grids line up or overlay one another. This is preferable to regridding to a common coarse resolution grid, as some of the information of how grids overlay on one another would be lost. We have used these methods previously for the same reason (Anenberg et al., 2009, 2014; Silva et al., 2013).

Our 0.5°x0.5° gridded estimates do not truly represent the fine-scale structure of air pollutant concentrations as a model simulation at this resolution might be able to achieve, since no model

was run at this fine resolution. As a result, we clearly indicate in the paper that the resolution is insufficient to capture local or urban scale effects (p.4, lines 16-18):

“Mortality estimates are obtained at a sufficiently fine horizontal resolution (0.5°x0.5°) to capture both global and regional effects and inform regional and national air quality and climate change policy, but are not expected to capture local scale (e.g., urban) air pollution effects.”

Also, we have revised the text as follows (p. 5, lines 8-11)

“The native grid resolutions of the 14 models varied from 1.9°x1.2° to 5°x5°; we regrid ozone and PM2.5 species surface concentrations from each model to a common 0.5°x0.5° horizontal grid to take maximum advantage of how the grids of different models overlap, following Anenberg et al. (2009, 2014) and Silva et al. (2013).”

And we have added discussion of the uncertainty in results brought about by the coarse resolution of global models (p. 12, lines 6-10):

“The differences in air pollutant concentrations reported by the ACCMIP models reflect different treatments of atmospheric dynamics and chemistry, chemistry-climate interactions, and natural emissions in each model (e.g., Young et al., 2013). Although there is likely a bias in estimating health effects using air pollutant concentrations from coarse resolution models (Li et al., 2015; Punger and West, 2013), particularly for PM2.5, we do not expect resolution to be an important factor for the differences in simulated concentrations across coarse resolution models.”

*p5, line 19-20: “similar to Silva et al 2013...except for...” Does this mean exactly the same as the Silva et al 2013 paper except for those two differences? The description is unclear, and the language here could be more precise.*

The methods are identical except for those two differences. As we detail later in the Methods section, “we apply the IER model instead of RRs from Krewski et al. (2009), used by Silva et al. (2013), as the newer model should better represent the risk of exposure to PM2.5, particularly at locations with high ambient concentrations”, and we use projections of population and baseline mortality rates to estimate the effect of future air pollution “considering the population that will potentially be exposed to those effects.”

We have revised the initial sentence of the Methods section to make it more precise (p. 5, lines 26-30):

“We estimate future air pollution-related cause-specific premature mortality using generally the same methods as those used by Silva et al. (2013) to obtain present-day estimates, but with two important differences: (1) we use the recently published Integrated Exposure-Response (IER) model for PM2.5 (Burnett et al., 2014), and (2) we use projections of population and baseline mortality rates from the International Futures (IFs) integrated modeling system (Hughes et al., 2011).”

*p6, line 9-10: using a common projection of population across the RCPs introduces both consistency in this analysis, but inconsistency relative to underlying social drivers. The implications of this choice should be discussed further, with quantifications of the*

*magnitude as well as the direction.*

Taking into account the referee's comments, we have revised the text (p. 6, lines 32-33):

"Population projections from IFs differ from those underlying each RCP, but lie within the range of the RCPs (Figure S4). In 2030, global total population in IFs is within 0.08% of that reported for RCP2.6, RCP4.5 and RCP6.0 and 5% lower than for RCP8.5; however, in 2100 IFs projects larger global populations than RCP2.6 (+7%), RCP4.5 (+13%) and RCP6.0 (+2%) and considerably lower than RCP8.5 (-27%). IFs projects rising baseline mortality rates for cardiovascular diseases (CVD) and RESP, globally and in most regions (particularly in East Asia and India), reflecting an aging population. By using projections from IFs, we have a single source of population and baseline mortality rates, assuring their consistency and enabling us to isolate the effect of changes in air pollutant concentrations across the RCPs. Had we used the population projections from each scenario, the magnitude of the changes (increases or decreases in premature mortality relative to 2000) would likely increase in RCP8.5, but decrease in RCP2.6, RCP4.5 and RCP6.0."

*p6, line 27-28: I can guess what the authors are referring to here, but the language could be easily misinterpreted (as the authors do actually look at the influence of climate on air pollutants themselves, just not modifications in ER factors). Rephrase?*

We have revised the sentence, as suggested (p.7, lines 9-12):

"Our results do not reflect the potential synergistic effect of a warmer climate on air pollution-related mortality, i.e., we do not account for potential changes in the exposure-response relationships at higher temperatures (Pattenden et al. 2010; Wilson et al., 2014 and references therein)."

p7, line 1-5: are potential correlations between different RRs accounted for in the Monte Carlo sampling? If so, how is that done? If not, the spread could be artificially narrowed. Please discuss.

It is not clear that there would be correlations between the RRs for different causes of death resulting from PM2.5, or if there are, it is not clear how they would be modeled. We have evaluated uncertainty for each cause of death separately and then added these results together. For ozone, there is only one cause of death and this is not an issue. For PM2.5, there are four causes of death. The Referee is correct that if there are correlations between these RRs, our methods would underestimate the overall uncertainty for PM2.5.

We have added a sentence to acknowledge this limitation (p. 7, lines 29-31):

"Uncertainty from the RRs is propagated separately for each model-scenario-year to mortality estimates in each grid cell, through 1000 Monte Carlo (MC) simulations, i.e. we repeat the calculations in each grid cell 1000 times using random sampling of the RR variable. For ozone, we use the reported 95% Confidence Intervals (CIs) for RR (Jerrett et al., 2009) and assume a normal distribution, while for PM2.5 we use the parameter values reported by Burnett et al. (2014) for 1000 MC simulations (GHDx 2013). Then for each of the 1000 simulations, we add mortality over many grid cells to obtain regional and global mortality and estimate the empirical

mean and 95% CI of the regional and global mortality results. We assume no correlation between the RRs for the four causes of death; thus we may underestimate the overall uncertainty for PM2.5 mortality estimates."

*p7, line 10: for the ACP audience, please describe 'tornado analysis' more thoroughly and quantitatively. Also, it is not addressed again, and there is no associated figure that corresponds to a traditional tornado-type plot.*

We have revised the sentence in Methods to include a description of the tornado analysis (p. 7, line 34, to p. 8 line 2):

"We also estimate the contribution of uncertainties in RR and in air pollutant concentrations to the overall uncertainty in mortality estimates using a tornado analysis; we obtained global mortality estimates treating each variable as uncertain individually (year 2000 concentrations, future year concentrations, RR for ozone, and the four parameters in the IER model for PM2.5) and used central estimates for all other variables, and then calculated the contribution of each variable to the overall uncertainty (when all variables are treated as uncertain simultaneously)."

The quantitative results from the tornado analysis are included in the following sentences:

(p. 9, lines 10-13) [ozone] "While uncertainty in RR and in modeled ozone concentrations have similar contributions to overall uncertainty in mortality results in 2050 (51% and 49%, respectively), in 2030 modeled ozone concentrations are the greatest contributor (81%), and in 2100 uncertainty in RR contributes the most to overall uncertainty (88%)."

(p. 10, lines 12-16) [$PM_{2.5}$] "Uncertainty in modeled PM2.5 concentrations in 2000 is the greatest contributor to overall uncertainty (59% in 2030, 45% in 2050, and 49% in 2100), followed by uncertainty in modeled PM2.5 in future years (40% in 2030, 26% in 2050 and 32% in 2100). Uncertainty in RR has a negligible contribution to overall uncertainty in 2030 (<1%), as the multi-model mean mortality change happens to be near zero (one model projects a large increase while the other five models project decreases), but contributes 29% in 2050 and 20% in 2100."

We do not show a traditional tornado plot since there are few variables treated as uncertain, and we combine related uncertainties together (the IER parameters). The full results of such a plot are communicated in the above sentences.

*p7. line 7: While the authors do have a certain spread of air pollutant concentrations, this should not be taken as a measure of 'uncertainty'. It is decidedly not a quantitative uncertainty analysis, as there are many other factors affecting 'uncertainty' in air pollutant concentrations that are not captured by the ACCMIP ensemble. This should be noted and discussed, and language carefully examined throughout the paper.*

We consider that the spread of air pollutant concentrations across models is a measure of uncertainty in air pollutant concentrations, although it does not account for uncertainty in emission inventories or for potential bias in modeled air pollutant concentrations. We have revised the Methods and the Discussion sections to address the referee's comments:

(p. 7, lines 31-34)
"Uncertainty in air pollutant concentrations is based on the spread of model results by calculating the average and 95% CI for the pooled results of the 1000 MC simulations for each model. This estimate of uncertainty in concentrations does not account for uncertainty in emissions inventories (as the ensemble used identical emissions) or for potential bias in modeled air pollutant concentrations."

(p. 12, lines 29-37)
"The spread of model results does not account for uncertainty in emissions inventories, as all ACCMIP models used the same projections of anthropogenic emissions. Moreover, climate and air quality interactions and feedbacks are sufficiently understood to be fully reflected in modeled air pollutant concentrations, and global models simplify atmospheric physics and chemical processes. This is particularly important when modeling air quality given scenarios of future emissions and climate change. For example, most global models do not fully address climate sensitivity to biogenic emissions (e.g. isoprene, soil NOx and methane) and stratosphere-troposphere interactions (e.g. stratospheric influx of ozone). A better understanding of aerosol-cloud interactions, of the impact of climate change on wildfires, and of the impact of land use changes on regional climate and air pollution is also crucial."

*p 7, line 19: "In some cases…" This sentence is confusing. Rephrase?*

We have revised the sentence as suggested:

(p. 8, lines 10-13) "In some cases, the changes in future mortality due to changes in future concentrations relative to 2000 show a different trend than the global mortality burden; this difference reflects the combined effects of future changes in concentrations relative to 1850, exposed population and baseline mortality rates."

*p 9, lines 21-22: I'm not clear what was done here. This should be addressed in detail in methods.*

These results reflect the following text in Methods (p. 5, lines 6-8):

"We use our PM2.5 estimates to obtain all mortality results, and perform a sensitivity analysis using the PM2.5 concentrations reported by four models using their own PM2.5 formulas, which differed among models, as reported in Silva et al. (2013)."

We revised the text in Results to expand the explanation (p. 10, lines 17-18):

"We compared mortality results using our estimates of PM2.5 from the sum of reported species with results using PM2.5 reported by four models applying their own formula to estimate PM2.5 (Figure 7)."

*p 10, line 31-32: This difference is noted. However, anyone familiar with the ACCMIP effort could have gleaned this simply from the previous reported results. What is new here? Why is this particularly significant in terms of mortality?*

We agree that there is a spread of results among the ACCMIP models. But it is not entirely obvious how this would influence the spread of mortality results, since one would have to account for the uneven distribution of population around the world, as we have done here. We highlight here the results specifically for estimates of human mortality, and show in the next sentence how the uncertainty contributed by the spread of model results compares with the uncertainty in the concentration response function itself (p. 12 , lines 8-13):

"The importance of conducting health impact assessments with air pollutant concentrations from model ensembles, instead of from single models, is highlighted by the differences in sign of the change in mortality among models, and by the marked impact of the spread of model results on overall uncertainty in our mortality estimates. In most cases assessed here (ozone mortality in 2030 relative to 2000, PM2.5 mortality in 2030, 2050 and 2100 relative to 2000), uncertainty in modeled air pollutant concentrations is the greatest contributor to uncertainty in mortality estimates."

*p 11, line 16+ This could be discussed in more depth, including more quantitatively, as it's a key limitation of the authors' analysis.*

We have revised the text to account for this comment, as well as in responding to major comment #2 above. We highlight the comparison with results from other studies using different scenarios:

Discussion (p. 13, line 1-11): "Our results are limited by the range of air pollutant emissions projected by the RCPs, which assume that economic growth strengthens efforts to reduce air pollutant emissions. All RCPs consider reductions in anthropogenic precursor emissions associated with more extensive air quality legislation as incomes rise, except for methane in RCP8.5 and for ammonia in all scenarios. These scenarios together do not encompass the range of plausible air pollution futures for the 21st century, as the RCPs were not designed for this purpose (van Vuuren et al., 2011a). Other plausible scenarios have been considered, such as the Current Legislation Emissions and Maximum Feasible Reductions scenarios used by Likhvar et al. (2015) and the Business-As-Usual scenario of Lelieveld et al (2015). As noted above, our global burden estimates for 2050 are considerably lower than the Business-As-Usual scenario of Lelieveld et al. (2015). If economic growth does not lead to stricter air pollution control, emissions and health effects may rise considerably, particularly for scenarios of high population growth in developing countries."

***Anonymous Referee #2***

*This manuscript uses the RCPs to project estimated air pollutant levels and health impacts globally for 10-year intervals between 2000 and 2100. It advances previous publications through the use of projected baseline mortality and population size along with projected air pollutant concentrations and therefore one can isolate the impacts of projected emissions from those of demographic changes in estimating future health impacts from air pollution. Further, the use of ensemble forecasts allows for the evaluation of the role of model variability in future estimates. Interestingly, while mortality impacts related to PM2.5 levels are projected to decrease under all scenarios, mortality from ozone exposure is projected to increase in all scenarios due to changes in population demographics, the absence of widespread decreases in ozone*

*concentrations, increases in methane and climate warming. There are two main analyses in the manuscript:1) the impact of concentration changes relative to those in the year 2000 which is focused on the effects of future emissions and the variability between the different models, and 2)the assessment of the overall burden of disease attributable to air pollution in future years relative to pre-industrial (1850) concentrations where the relative impacts of emissions, and population projections are compared (cases A and B).*

*General comments*
*Overall the manuscript provides unique new information to assess both potential future health impacts under well-defined scenarios and the role of model variability, uncertainty in concentration-response functions, uncertainty in emissions and the role of demographic changes in the estimation of future impacts. While the absolute numbers from the simulations are interesting, arguably more important is the assessment of uncertainty and the relative roles of different factors (demographics, emissions) in future estimates. For this component of the manuscript, decreasing the emphasis on the absolute numbers while providing more relative comparisons would help the reader sort through all of the results. Further the manuscript would benefit from some clear take-home messages on the relative impacts of future emissions and demographic changes and on the largest contributors to overall uncertainty. This information is in the manuscript but is hard to find and needs to be brought forward (even if it means decreasing emphasis on the absolute numbers).*

We thank Referee #2 for these encouraging and helpful comments. We have made changes throughout the manuscript, particularly in the Discussion and Conclusions sections to decrease emphasis on particular numerical results, and to strengthen our communication of key messages, and have responded to the specific comments below.

*The estimates for 2000 are low compared with other similar estimates and the authors attribute this to the choice of counterfactual. Given that the counterfactual is a choice, it would seem useful to isolate the impact of the choice of counterfactuals if the absolute number is being emphasized – some simple sensitivity analyses in which, for example, the Global Burden of Disease counterfactuals were applied, would be useful.*

Following the referee's suggestion, we included a simple sensitivity analysis considering the global burden for 2000 using the GBD counterfactuals. We have improved the comparison with GBD 2013 and added the comparison with estimates using the GBD counterfactuals to the Results section (p. 10, line 34, to p. 11, line 10):

"Compared with the GBD 2013 (Forouzanfar et al. 2015), our estimates are 76% higher for ozone-related mortality and 42% lower for PM2.5-related mortality, likely due to the fact that we estimate the global mortality burden using 1850 concentrations as baseline, while Forouzanfar et al. (2015) consider counterfactual concentrations (theoretical minimum-risk exposure) that are mostly higher for ozone (uniform distribution between 33.3 and 41.9 ppb) and lower for PM2.5 (uniform distribution between 5.9 and 8.7 μg/m3) than 1850 concentrations. In addition, we consider ozone mortality from all chronic respiratory diseases while Forouzanfar et al. (2015) only account for COPD, and we restrict our mortality estimates to adult population while Forouzanfar et al. (2015) include PM2.5 mortality from lower respiratory tract infections in young children. As a sensitivity analysis, when we apply a counterfactual of 33.3 ppb (instead of using 1850 concentrations), our ozone-related mortality estimates are 23% higher for the multi-model mean, varying between +10% and +52% among models. Similarly, using the IER model

counterfactual, our PM2.5-related mortality estimates are 22% lower for the multi-model mean, varying between -8% and -44% among models."

*Future ozone and PM2.5 attributable mortality is clearly driven by China and India; given this it might be useful to present (or at least comment on) the model variability in these regions as what appears to be overall agreement across most of the models may be a result of smoothing due to other regions which have relatively minor impacts on future trends.*

We have added to the Supplemental Material maps of the coefficient of variation (Figures S8 and S9) to show the spatial distribution of model variability for all RCPs and all future years. In most cases, PM2.5 concentrations show lower variability in India and China than in other regions across RCPs and future years. In most cases, variability of ozone concentrations across models is much greater in 2030 than in 2050 and 2100, including in China and India. We have added text to the Results section to address this point (p. 9, line 36 to p. 10, line 2):

"East and South Asia are the regions with the greatest projected mortality burdens, and the variability in PM2.5 among models is typically less in these regions than in several other regions globally, depending upon the scenario and year (Figure S9)."

*Specific comments Abstract should be more consistent in presenting uncertainty in estimates and should include some quantification of uncertainty. Abstract should also provide more emphasis on uncertainty and relative impacts of different sources for the burden of disease estimates*

We have revised the Abstract taking into account the reviewer's comment (p. 2, lines 8, 12-13 and 17):

"However, the global mortality burden of ozone markedly increases from 382,000 (121,000 to 728,000) deaths/year in 2000 to between 1.09 and 2.36 million deaths/year in 2100, across RCPs, mostly due to the effect of increases in population and baseline mortality rates. PM2.5 concentrations decrease relative to 2000 in all scenarios, due to projected reductions in emissions, and are associated with avoided premature mortality, particularly in 2100: between -2.39 and -1.31 million deaths/year for the four RCPs. The global mortality burden of PM2.5 is estimated to decrease from 1.70 (1.30 to 2.10) million deaths/year in 2000 to between 0.95 and 1.55 million deaths/year in 2100 for the four RCPs, due to the combined effect of decreases in PM2.5 concentrations and changes in population and baseline mortality rates. Trends in future air pollution-related mortality vary regionally across scenarios, reflecting assumptions for economic growth and air pollution control specific to each RCP and region. Mortality estimates differ among chemistry-climate models due to differences in simulated pollutant concentrations, which is the greatest contributor to overall mortality uncertainty for most cases assessed here, supporting the use of model ensembles to characterize uncertainty. Increases in exposed population and baseline mortality rates of respiratory diseases magnify the impact on premature mortality of changes in future air pollutant concentrations and explain why the future global mortality burden of air pollution can exceed the current burden, even where air pollutant concentrations decrease."

As we did not estimate uncertainty for future scenarios (except for RCP8.5), we do not report uncertainty ranges for future results in the abstract.

*L89 -Lim et al should be updated with Forouzanfar et al., 2015*

We have updated the reference as suggested (p. 2, line 29; p. 10, line 34)

*L102 - suggest that in future ozone concentrations will decrease with climate change; can this be reconciled with observations on global increases during recent periods? (Emissions vs warming?)*

As we state in the paper, concentrations of ozone are expected to increase in polluted regions in the warm season, as a result of future climate change. Ozone is likely to decrease in remote regions as a result of climate change. Our previous work analyzed the effects of past climate change on air pollution-related mortality, finding a small influence (Silva et al., 2013). This finding is consistent with the current literature, which reports that the effect of past emissions changes far outweighs the effect of climate change at present. As we do not focus on climate change in this paper, we have not changed the text to address this point.

*L239 How do IF projections compare with current numbers, i.e. from the Global Burden of Disease ( ~~for~~ 2010)?*

IF projections for 2010 and GBD 2010 estimates of age-standardized mortality rates (deaths per 100,000 people) are:

| Diseases | IF | GBD 2010 |
|---|---|---|
| Cardiovascular | 234.9 | 234.8 |
| Chronic Respiratory | 58.4 | 57.0 |
| Neoplasms | 106.9 | 121.4 |

We have added this table to the Supplemental Material (Table S4).

Data in Figure S5 are for adult population only. We added this text to the paper (p. 7, lines 19-20):

"IFs projections for 2010 are comparable to GBD 2010 estimates (Lozano et al., 2012) for CVD (+0.04%), RESP (+2.5%) and neoplasms (-12%)."

*L283 –Should mention in limitation/discussion that the absence of uncertainty in the IF projections may be as important as other sources of uncertainty and that this uncertainty would increase over time (i.e. 2100 vs 2030)*

We have revised the text in the Discussion section taking into account this comment (p. 12, lines 26-28):

"Uncertainty is evaluated for a single future population projection, not accounting for the wide range of projections in the literature, and does not reflect uncertainty in baseline mortality rates, as these are not reported; uncertainties in both population and baseline mortality rates would be expected to increase with time into the future."

*L299-310 –There would appear to be ~20x variability estimates for the different RCP scenarios - this is very large and clearly makes the case that emissions DO matter - it seems that this point should also be brought out a bit more.*

This is a good point. We have revised the conclusions section to make this point in a more prominent place in the manuscript (p. 13, lines 19-22):

"These reductions in ambient air pollution-related mortality reflect the decline in pollutant emissions projected in the RCPs, but the large range of results from the four RCPs highlights the importance of future air pollutant emissions for ambient air quality and global health."

The theme of the importance of emissions is again reiterated in the last paragraph of the paper.

*L404 what are the 1850 concentrations that are used as the counterfactual? These should be provided in the text.*

We do not use a single value for 1850 concentrations that applies globally. Rather, a different value is present in each grid cell as a result of the model simulations. We have added maps of 1850 concentrations for ozone and PM2.5 to the supporting information (Figures S4 and S5).

*Apte JS et al., ES&T 2015 also estimates future mortality assuming only changes on population – it would be useful to cite this paper and make some rough comparisons*

We have included Apte et al. 2015 in the literature cited in the Introduction (p. 2, lines 28)

"Previous studies have estimated the present-day global burden of disease due to exposure to ambient ozone and/or PM2.5 (e.g., Apte et al., 2015; Evans et al., 2013; Forouzanfar et al. 2015Lim et al., 2012), with several studies estimating this burden using only output of global atmospheric models (Anenberg et al., 2010; Fang et al., 2013a; Lelieveld et al., 2013; Rao et al., 2012, ; Silva et al., 2013). However, few studies have evaluated how the global burden might change in future scenarios (e.g. Lelieveld et al., 2015; Likhvar et al., 2015; West et al., 2007)."

and in the comparisons included in the Results (p. 11, lines 17-24):

"For PM2.5, the increase in exposed population and the decline in concentrations have a much greater effect than changes in baseline mortality rates (Figure 9). These results are similar to those of Apte et al. (2015) who report a stronger effect of projected demographic trends in India and China in 2030 than of changes in baseline mortality rates. Our estimates for the global burden of PM2.5 mortality in 2050 (between 1.82 and 3.50 million deaths/year for the four RCPs) are considerably lower than those of Lelieveld et al. (2015) (5.87 million deaths / year for IHD+STROKE+COPD+LC), likely due to the assumption in the RCP scenarios of further regulations on air pollutants, while the Business-As-Usual scenario of Lelieveld et al. (2015) does not assume regulations beyond those currently defined."

L472 "preature" typo

Corrected.

***Anonymous Referee #3***

*General comments: This study performs a global health impact assessment from ambient air pollution, using chemical transport or chemistry-climate models, for a set of RCP scenarios, for the years 2000, 2030, 2050 and 2100. Similar studies have been published before (properly acknowledged by the authors). The novelty of this study lies in the use of an ensemble of models, allowing for an evaluation of the contribution of model-calculated population exposure to pollution in the total uncertainty on the health impact. However a comparison of the outcome with previous studies, both for present day and future projections, is not obvious because of differences in methodology.*

We thank Referee #3 for the constructive comments and have addressed the specific comments below. We have improved our presentation of previous studies in the Introduction, Although we compare our results for the present-day GBD with several studies, we agree that it is difficult to do a systematic comparison of future mortality with the few comparable studies available, and only compare quantitatively with the results of Lelieveld et al. (2015).

*Specific comments: In the paper two ways are used to evaluate the impact of emission scenarios for the future on human health: 1) By using future demographics and health statistics, and combining these with exposure to year 2000 pollutant levels and to pollutant levels corresponding to projected emissions for the specific year respectively and making the difference 2) by calculating the absolute number of mortalities for each considered year and making the difference with mortalities for 1850 ('mortality burden'). It took me a while to understand that reported 'avoided' and 'excess' mortalities refer to method 1). It should be better explained in the methodology section. Usually, avoided or excess mortalities for a given scenario are calculated versus a reference scenario for the same year (e.g. a stringent policy versus a business-as-usual as reference case). It's not clear here what the year 2000 pollution transposed to 2030 and 2050 actually represents as a reference. The avoided or excess mortalities can not be directly linked to specific policies (which pathway would have led to the year 2000 levels in 2030 - 2050 - 2100?). Wouldn't it make more sense to use e.g. RCP 8.5 as a reference, and evaluate the benefits of the 2.6 and 4.5 pathways? Using year 2000 pollution levels as a reference for future years also introduces an issue with exposure; concentration field spatial distribution is linked to population spatial patterns – in particular for PM. Does is make sense to overlay year 2000 pollution spatial patterns with year xxxx population spatial distribution?*

We use year 2000 pollution levels as a common counterfactual against which future pollutant concentrations are evaluated. In doing so, we evaluate future concentrations as they affect future population, relative to the year 2000 concentrations. That is, we evaluate air pollution mortality in future years relative to the case where that future population breathes air from the year 2000. This approach is analogous to using 1850 concentrations as a counterfactual, and we choose to use 2000 rather than 1850 as the main results that we present for the reasons we state in the paper – particularly because it does not require assumptions about the shape of the

concentration-response function at the very low concentrations present in 1850. We also prefer comparing with 2000 concentrations because that is the state of air pollution with which we are all familiar.

We have revised the text in the Methods section to improve discussion of these points, and to clarify the use of 'avoided/excess' mortality (p. 5, line 35 to p. 6, line 1):

"We calculate changes in premature mortality by applying the change in pollutant concentrations in each future year (2030, 2050, 2100) relative to year 2000 concentrations - the present-day state of air pollution - to the future population. We therefore estimate 'avoided' or 'excess' premature mortality due to decreases or increases in air pollutant concentrations in the future years relative to 2000 concentrations. This approach differs from a calculation of the global burden of air pollution-related mortality since we use 2000 rather than 1850 concentrations as baseline. We estimate mortality changes due to future concentration changes, relative to the present, to avoid applying the health impact function at very low concentrations where there is less confidence in the exposure-response relationship. For example, the simulated 1850 air pollutant concentrations are often below the lowest measured value of the American Cancer Society study (Jerrett et al., 2009; Krewski et al., 2009). For illustration, we also estimate mortality relative to 1850 concentrations, which could be regarded as global burden of disease calculations, following Silva et al. (2013)."

*Mortalities are estimated at 0.5x0.5 deg resolution: is this just a regridding of the native model resolution or was any downscaling done to better estimate the exposure indensely populated areas? Apparently the concentrations are just regridded; this cannot be considered as a proper population-weighted exposure estimate at the coarse resolution of the models, as all population within a single grid will be exposed to the same level.*

We have regridded the concentration fields for each model individually, without doing any downscaling, as the Referee suggests. We regrid to a finer resolution to improve our estimates for the multi-model average, retaining information on how the grids for different models line up. We have improved the text to clarify our purpose in regridding (p. 5, lines 8-11):

"The native grid resolutions of the 14 models varied from 1.9°x1.2° to 5°x5°; we regrid ozone and PM2.5 species surface concentrations from each model to a common 0.5°x0.5° horizontal grid to take maximum advantage of how the grids of different models overlap, following Anenberg et al. (2009, 2014) and Silva et al. (2013)."

*Regarding the use of Burnett's IER functions: specify whether age-specific functions have been used or all-ages. From what is written in the first par. of page 15, I understood that the Burnett functions have been applied without the counterfactual value? In fact it is not well explained how teh difference with 1850 was made: by first subtracting 1850 concentrations and then applying the exposure-response functions, or by applying exposure-response functions to both years and then subtracting mortalities. And how was it done for calculating the excess/avoided mortalities relative to year 2000?*

When applying the IER model, we used age-specific functions when reported by Burnett et al. 2014 (IHD and Stroke). We have revised the text to mention this (p. 6, lines 16-17):

"We also estimate PM2.5-related mortality due to ischemic heart disease (IHD), cerebrovascular disease (STROKE), chronic obstructive pulmonary disease (COPD) and lung cancer (LC), using RRs from the IER model (Burnett et al., 2014). We use RR per age group for IHD and STROKE and RR for all-ages for COPD and LC."

We used the RRs (central estimate) from the IER model reported by Burnett et al. 2014 for $PM_{2.5}$ concentrations up to 300 $\mu g/m^3$ (GHDx 2013) to obtain the deterministic mortality estimates. For the uncertainty analysis, we use the values for parameters alpha, gamma, delta and $z_{cf}$ (counterfactual) reported by Burnett et al. (2014) for 1000 simulations (GHDx 2013).

We have revised the text to make this more clear (p. 7, lines 25-27):

"For ozone, we use the reported 95% Confidence Intervals (CIs) for RR (Jerrett et al., 2009) and assume a normal distribution, while for PM2.5 we use the values for the parameters alpha, gamma, delta and zcf (counterfactual) reported by Burnett et al. (2014) for 1000 MC simulations (GHDx 2013)."

We applied the exposure-response function to both years (future years and 1850 for global burden and futures years and 2000 for the excess/avoided future mortality) and then subtracted the mortality estimates. We have revised the text to explain this (p. 6, lines 1-5):

"To estimate ozone mortality, we apply the exposure-response function to the difference in ozone concentrations, while for PM2.5 mortality we apply the exposure-response function to concentrations in each year (future years and 2000) and then subtract the mortality estimates. We therefore estimate 'avoided' / 'excess' premature mortality due to decreases / increases in air pollutant concentrations in the future years relative to 2000 concentrations."

*The numbers in Table S3 do not seem to be consistent with year 2030 mortalities in Figure 4: In Table S3 only 2 models predict a global mean decrease in PM2.5 for RCP2.6 in 2030. In Figure 4 all models except 1 show a decrease in mortalities by 2030...Similar for the other RCPs; most flagrant for RCP8.5 where all PM2.5 appears to increase globally but only 1 model leads to an increase in mortality. How to explain this?*

The numbers in Table S3 are global averages and are often close to zero in 2030. The spatial distribution of concentrations, how they overlay on baseline mortality rates, and the magnitudes of baseline (2000) and future concentrations (which determine their place in the IER exposure-response curves) have significant impacts on the mortality estimates due the non-linearity in the IER model. We have checked both the calculation of mortality and the calculation of population-weighted concentrations to ensure that both were done correctly.

We thank the Referee for this comment, and think that it is a nice way to illustrate the importance of the nonlinearity of the IER function. We have added text to show this (p. 10, lines 3-8):

"Future $PM_{2.5}$-related mortality estimates are influenced by the nonlinearity of the IER function. For example, in RCP8.5 in 2030, all models project an increase in global population-weighted concentration (Table S3) but all models except one show decreases in global $PM_{2.5}$-related mortality (Figure 4). This outcome results in part because $PM_{2.5}$ increases are projected in regions with high concentrations (particularly East Asia) that are on the flatter part of the IER curve, whereas $PM_{2.5}$ decreases in regions with low concentrations (North America and Europe) have a steeper slope and therefore a greater influence on global mortality."

*Table S4: should be mentioned as 'CHANGE' in mortalities between year 2000 pollution levels and respective scenario/year pollution levels. Also on Page 11, "Global future premature mortality rises from 264,000 (-39,300 to 648,000) deaths in 2030 to 316,000 (-310 187,000 to 1.38 million) deaths in 2100" may cause confusion as these are again changes compared to 2000 pollution levels.*

The caption of Table S4 (now Table S5) has been revised to "Change in global respiratory premature ozone mortality in 2030, 2050 and 2100 for all RCPs (considering the change in future ozone concentrations relative to 2000 concentrations), showing the multi-model average (deaths/year) for RCP2.6, RCP4.5 and RCP6.0 deterministic estimates and the empirical mean with 95% CI in parenthesis for RCP8.5 probabilistic estimates (including uncertainty in the RRs and across models). These results correspond to Figure 1. All numbers are rounded to three significant digits."

The caption of Table S6 (now Table S7) was revised in a similar way.

The text on p. 9 (lines 6-8) has been changed to:
"Global future premature mortality changes from 264,000 (-39,300 to 648,000) deaths in 2030 to 316,000 (-187,000 to 1.38 million) deaths in 2100."

*The fact that the range spans from negative to positive implies that the result is not significantly different from 0?*

We do not include statistical significance testing to evaluate whether results are significantly different from zero. Rather, we present results such that the reader can understand both the results for individual models with uncertainty from the concentration-response function and the net uncertainty when evaluating over all models.

*What has been the benefit of the multi-model analysis? And what can be learned from analyzing the RCP scenarios? Are the outcomes plausible in the light of the implicitly assumed rather stringent pollution controls?*

We have improved the Discussion and Conclusion sections taking into account these comments, as shown, for example, in the following excerpts:

(Discussion: p. 12, lines 1-7):
"In all RCP scenarios but RCP8.5, stringent air pollution controls lead to substantial decreases in ozone concentrations through the 21st century, relative to 2000. For RCP8.5, the higher

baseline GHG (including methane) and air pollutant emissions lead to increases in future ozone concentrations. In contrast, global PM2.5 concentrations show a decreasing trend across all RCP scenarios. These changes in air pollutant concentrations, combined with projected increases in baseline mortality rates for chronic respiratory diseases, drive ozone mortality to become more important relative to PM2.5 mortality over the next century.

The importance of conducting health impact assessments with air pollutant concentrations from model ensembles, instead of from single models, is highlighted by the differences in sign of the change in mortality among models, and by the marked impact of the spread of model results on overall uncertainty in our mortality estimates."

Conclusion (p. 13, lines 19-24):
"These reductions in ambient air pollution-related mortality reflect the decline in most emissions projected in the RCPs, but the large range of results from the four RCPs highlights the importance of future air pollutant emissions for ambient air quality and global health. Mortality estimates differ among models and we find that, for most cases, the contribution to overall uncertainty from uncertainty associated with modeled air pollutant concentrations exceeds that from the RRs."

*The results section is dry and hard to digest with long lists of numbers of mortality changes per scenario, per region, with differences between models – all things that are much easier to read from the figures than in the text. For the reader it is hard to keep an overview and grasp the major message. Suggest to reduce and condense this section to most salient observations that are maybe not directly evident from the figures.*

We made minor changes to the Results section itself - to provide less emphasis on particular numerical results – but have made significant changes to the Discussion and Conclusions sections where the major points are now reiterated more clearly.

*Discussion section: it looks like there is an increasing relative importance of O3 as health impact compared to PM for the future (what is the relative contribution of each pollutant to total pollution mortality burden in each year, each scenario?) – this may be worth a few lines of discussion.*

We have added text in the Discussion and Conclusions sections taking into account this comment (p. 12, lines 1-7, and p. 13, lines 31-34):

"In all RCP scenarios but RCP8.5, stringent air pollution controls lead to substantial decreases in ozone concentrations through the 21st century, relative to 2000. For RCP8.5, the higher baseline GHG (including methane) and air pollutant emissions lead to increases in future ozone concentrations. In contrast, global PM2.5 concentrations show a decreasing trend across all RCP scenarios. These changes in air pollutant concentrations, combined with projected increases in baseline mortality rates for chronic respiratory diseases, drive ozone mortality to become more important relative to PM2.5 mortality over the next century."

"A strong decline in PM2.5 concentrations for all RCPs together with demographic trends in the 21st century (with a projected substantial increase in exposed population) lead to a rising importance of ozone relative to PM2.5 for the global burden of ambient air pollution-related mortality."

*It is surprising that for the same emission scenarios, models have such different outcomes. Does the resolution play a role here? What could be done to improve the exposure estimate? Downscaling techniques? Use of regional models? Is it possible to evaluate the error made by using course resolution models?*

We do not have an easy way to separate the influence of resolution on health outcomes. Previous work that is now cited in the paper (Punger and West, 2013; Li et al., 2015) suggests that there is a bias in estimating health effects from using coarse resolution models that is greater for PM2.5 than for ozone. However, we expect that resolution does not play a big role in the difference between models. That is, there may be a bias from coarse grid resolution relative to fine resolution – and we have added text to acknowledge this point (below) – but the bias caused by resolution from one coarse grid to another should be fairly small. Instead, the difference is caused by differences in modeled concentrations, reflecting the different meteorology and atmospheric chemistry within the different models.

We have added the following sentences to the Discussion section (p. 12, lines 13-17).

"The differences in air pollutant concentrations reported by the ACCMIP models reflect different treatments of atmospheric dynamics and chemistry, chemistry-climate interactions, and natural emissions in each model. Although there is likely a bias in estimating health effects using air pollutant concentrations from coarse resolution models (Li et al., 2015; Punger and West, 2013), particularly for PM2.5, we do not expect resolution to be an important factor for the differences in simulated concentrations across coarse resolution models."

*It would be nice to see a graph summarizing other paper's results and this one (with error bars) for projected mortality burdens and to discuss what could be learned from this comparison.*

As we now discuss more fully in the Introduction, there are several studies that are comparable, but there are many differences among these studies in the pollutants and health effects considered, the concentration-response functions used, the scenarios modeled and the time periods evaluated. Because of these large differences we suggest that a systematic comparison over all of the literature would be difficult and would not yield the meaning that the Referee asks for. We choose not to compare with the whole literature, but focus on comparing with Lelieveld et al. (2015), who used comparable health estimation methods but different future scenarios.

# The effect of future ambient air pollution on human premature mortality to 2100 using output from the ACCMIP model ensemble

Raquel A. Silva[1], J. Jason West[1], Jean-François Lamarque[2], Drew T. Shindell[3], William J. Collins[4], Stig Dalsoren[5], Greg Faluvegi[6], Gerd Folberth[7], Larry W. Horowitz[8], Tatsuya Nagashima[9], Vaishali ~~Naik[10]~~Naik[8], Steven T. Rumbold[7,*], Kengo ~~Sudo[11]~~Sudo[10], Toshihiko ~~Takemura[12]~~Takemura[11], Daniel ~~Bergmann[13]~~Bergmann[12], Philip Cameron-~~Smith[13]~~Smith[12], Irene ~~Cionni[14]~~Cionni[13], Ruth M. ~~Doherty[15]~~Doherty[14], Veronika ~~Eyring[16]~~Eyring[15], Beatrice ~~Josse[17]~~Josse[16], I. A. ~~MacKenzie[15]~~MacKenzie[14], David ~~Plummer[18]~~Plummer[17], Mattia ~~Righi[16]~~Righi[15], David S. ~~Stevenson[15]~~Stevenson[14], Sarah ~~Strode[19]~~Strode[18,~~20~~19], Sophie ~~Szopa[21]~~Szopa[20] and Guang ~~Zeng[22]~~Zeng[21,**]

[1]Environmental Sciences and Engineering, University of North Carolina, Chapel Hill, North Carolina, USA
[2]NCAR Earth System Laboratory, National Center for Atmospheric Research, Boulder, Colorado, USA
[3]Nicholas School of the Environment, Duke University, Durham, North Carolina, USA
[4]Department of Meteorology, University of Reading, Reading, United Kingdom
[5]CICERO, Center for International Climate and Environmental Research-Oslo, Oslo, Norway
[6]NASA Goddard Institute for Space Studies and Columbia Earth Institute, New York, New York, USA
[7]Met Office Hadley Centre, Exeter, United Kingdom
[8]NOAA Geophysical Fluid Dynamics Laboratory, Princeton, New Jersey, USA
[9]National Institute for Environmental Studies, Tsukuba, Japan
~~[10]UCAR/NOAA Geophysical Fluid Dynamics Laboratory, Princeton, New Jersey, USA~~
~~[11]Earth~~ [10]Earth and Environmental Science, Graduate School of Environmental Studies, Nagoya University, Nagoya, Japan
~~[12]Research~~ [11]Research Institute for Applied Mechanics, Kyushu University, Fukuoka, Japan
~~[13]Lawrence~~ [12]Lawrence Livermore National Laboratory, Livermore, California, USA
~~[14]Agenzia~~ [13]Agenzia Nazionale per le Nuove Tecnologie, l'Energia e lo Sviluppo Economico Sostenibile (ENEA), Bologna, Italy
~~[15]School~~ [14]School of GeoSciences, University of Edinburgh, Edinburgh, United Kingdom
~~[16]Deutsches~~ [15]Deutsches Zentrum für Luft- und Raumfahrt (DLR), Institut für Physik der Atmosphäre, Oberpfaffenhofen, Germany
~~[17]GAME~~[16]GAME/CNRM, Meteo-France, CNRS—Centre National de Recherches Meteorologiques, Toulouse, France
~~[18]Canadian~~ [17]Canadian Centre for Climate Modeling and Analysis, Environment Canada, Victoria, British Columbia, Canada
~~[19]NASA~~ [18]NASA Goddard Space Flight Center, Greenbelt, Maryland, USA
~~[20]Universities~~ [19]Universities Space Research Association, Columbia, Maryland, USA
~~[21]Laboratoire~~ [20]Laboratoire des Sciences du Climat et de l'Environnement, LSCE-CEA-CNRS-UVSQ, Gif-sur-Yvette, France
~~[22]National~~ [21]National Institute of Water and Atmospheric Research, Lauder, New Zealand
*Now at National Centre for Atmospheric Science (NCAS), University of Reading, Reading, United Kingdom
**Now at NIWA, Wellington, New Zealand

*Correspondence to*: J. J. West (jjwest@email.unc.edu)

**Abstract.** Ambient air pollution from ground-level ozone and fine particulate matter ($PM_{2.5}$) is associated with premature mortality. Future concentrations of these air pollutants will be driven by natural and anthropogenic emissions and by climate change. Using anthropogenic and biomass burning emissions projected in the four

Representative Concentration Pathway scenarios (RCPs), the ACCMIP ensemble of chemistry-climate models simulated future concentrations of ozone and $PM_{2.5}$ at selected decades between 2000 and 2100. We use output from the ACCMIP ensemble, together with projections of future population and baseline mortality rates, to quantify the human premature mortality impacts of future ambient air pollution. Future air pollution-related premature mortality in 2030, 2050 and 2100 is estimated for each scenario and for each model using a health impact function based on changes in concentrations of ozone and $PM_{2.5}$ relative to 2000 and projected future population and baseline mortality rates. Additionally, the global mortality burden of ozone and $PM_{2.5}$ in 2000 and each future period is estimated relative to 1850 concentrations, using present-day and future population and baseline mortality rates. The change in future ozone concentrations relative to 2000 is associated with excess global premature mortality in some scenarios/periods, particularly in RCP8.5 in 2100 (316 thousand deaths/year), likely driven by the large increase in methane emissions and by the net effect of climate change projected in this scenario, but it leads to considerable avoided premature mortality for the three other RCPs. However, the global mortality burden of ozone markedly increases from 382,000 (121,000 to 728,000)~~less than 0.4 million~~ deaths/year in 2000 to between 1.09 and 2.36 million deaths/year in 2100, across RCPs, mostly due to the effect of increases in population and baseline mortality rates. ~~Decreases in~~ $PM_{2.5}$ concentrations decrease relative to 2000 in all scenarios, due to projected reductions in emissions, and are associated with avoided premature mortality ~~in all scenarios~~, particularly in 2100: between -2.39 and -1.31 million deaths/year for the four RCPs ~~due to the reductions in emissions projected in these scenarios~~. The global mortality burden of $PM_{2.5}$ is estimated to decrease from 1.70 (1.30 to 2.10) million deaths/year in 2000 to between 0.95 and 1.55 million deaths/year in 2100 for the four RCPs, due to the combined effect of decreases in $PM_{2.5}$ concentrations and changes in population and baseline mortality rates. Trends in future air pollution-related mortality vary regionally across scenarios, reflecting assumptions for economic growth and air pollution control specific to each RCP and region. Mortality estimates differ among chemistry-climate models due to differences in simulated pollutant concentrations, ~~and~~ which is the greatest contributor to overall mortality uncertainty for most cases assessed here, supporting the use of model ensembles to characterize uncertainty. Increases in exposed population and baseline mortality rates of respiratory diseases magnify the impact on premature mortality of changes in future air pollutant concentrations and explain why the future global mortality burden of air pollution can exceed the current burden, even where air pollutant concentrations decrease.

## 1 Introduction

Ambient air pollution has adverse effects on human health, including premature mortality. Exposure to ground-level ozone is associated with respiratory mortality (e.g. Bell et al., 2005; Gryparis et al., 2004; Jerrett et al., 2009; Levy et al., 2005). Exposure to fine particulate matter with aerodynamic diameter less than 2.5 μm ($PM_{2.5}$) is associated with mortality due to cardiopulmonary diseases and lung cancer (e.g. Brook et al., 2010; Burnett et al., 2014; Hamra et al., 2014; Krewski et al., 2009; Lepeule et al., 2012). Previous studies have estimated the present-day global burden of disease due to exposure to ambient ozone and/or $PM_{2.5}$ (e.g., Apte et al., 2015; Evans et al., 2013; Forouzanfar et al., 2015~~Lim et al., 2012~~), with several studies estimating this burden using only output of global atmospheric models (Anenberg et al., 2010; Fang et al., 2013a; Lelieveld et al., 2013; Rao et al., 2012~~,~~; Silva et al.,

2013). However, few studies have evaluated how the global burden might change in future scenarios (~~e.g.~~ Lelieveld et al., 2015; Likhvar et al., 2015; West et al., 2007). Other global studies have estimated future air pollution-related mortality as a by-product of analyses of other future changes, such as the effects of climate change or of climate change mitigation (e.g., Fang et al., 2013b; Selin et al., 2009; West et al., 2013), but do not focus on the range of plausible future mortality as their main purpose. Similarly, studies at local and regional scales have evaluated the mortality impact of changes in air quality due to future climate change (Bell et al., 2007; Chang et al., 2010; Fann et al., 2015; Heal et al., 2012; Jackson et al., 2010; Knowlton et al., 2004, 2008; Orru et al., 2013; Post et al., 2012; Sheffield et al., 2011; Tagaris et al., 2009) but few such studies have evaluated changes beyond 2050.

Future ambient air quality will be influenced by changes in emissions of air pollutants and by climate change. Changes in anthropogenic emissions will likely dominate in the near-term (Kirtman et al., 2013 and references therein), and depend on several socio-economic factors including economic growth, energy demand, technological choices and developments, demographic trends and land use change, as well as air quality and climate policies. Climate change will affect the ventilation, dilution, and removal of air pollutants, the frequency of stagnation, photochemical reaction rates, stratosphere−troposphere exchange of ozone, and natural emissions (Fiore et al., 2012, 2015; Jacob and Winner, 2009; von Schneidemesser et al., 2015; Weaver et al., 2009). Climate change is likely to increase ozone in polluted regions during the warm season, particularly in urban areas and during pollution episodes. In remote regions, however, ozone is likely to decrease due to greater water vapor concentrations, which increase the loss of ozone by photolysis and subsequent formation of hydroxyl radicals (Doherty et al, 2013). The effects of climate change on $PM_{2.5}$ concentrations are generally uncertain as changes in temperature affect both reaction rates and gas to particle partitioning as well as wildfires and biogenic emissions, and vary regionally primarily due to differing projections of changes in precipitation (Fiore et al., 2012, 2015; Fuzzi et al., 2015; Jacob and Winner, 2009; von Schneidemesser et al., 2015; Weaver et al., 2009).

The Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP) simulated preindustrial (1850), present-day (2000) and future (2030, 2050 and 2100) concentrations of ozone and $PM_{2.5}$ with an ensemble of 14 state-of-the-art chemistry climate models (Table S1) (Lamarque et al., 2013, Stevenson et al., 2013) to support the IPCC's Fifth Assessment Report. Using modeled 1850 and 2000 concentrations from this ensemble, we showed previously that exposure to present-day anthropogenic ambient air pollution is associated with 470 (95% Confidence Interval (CI): 140, 900) thousand deaths/year from ozone-related respiratory diseases, and 2.1 (1.3, 3.0) million deaths/year from $PM_{2.5}$-related cardiopulmonary diseases and lung cancer (Silva et al., 2013). These results were obtained for a wider range of cardiopulmonary diseases and using a different exposure-response model for $PM_{2.5}$ mortality than the present study, as discussed later.

The ACCMIP models simulated future air quality for specific periods through 2100, for four global greenhouse gas (GHG) and air pollutant emission scenarios projected in the Representative Concentration Pathways (RCPs) (Van Vuuren et al., 2011a and references therein). The four RCPs were developed by different research groups with different assumptions regarding the pathways of population growth, economic and technological development, and air quality and climate policies. Anthropogenic radiative forcing in 2100 ranges from a very low level in the mitigation scenario RCP2.6 (Van Vuuren et al., 2011b), to medium levels in the two stabilization scenarios, RCP4.5

(Thomson et al., 2011) and RCP 6.0 (Masui et al., 2011), to a high level in the very high baseline emissions scenario RCP8.5 (Riahi et al., 2011). All RCPs assume increasingly stringent air pollution controls as countries develop economically, leading to decreases in air pollutant emissions that reflect the different methods of the different RCP groups (e.g., Smith et al., 2011)~~, and~~. But as assumptions are similar among the RCPs, the four scenarios do not span the range of possible futures published in the literature for short-term species. For example, other studies have simulated scenarios in which air pollution controls are kept at current levels while underlying trends (e.g., energy use) increase overall emissions (Lelieveld et al., 2015; Likhvar et al., 2015). While most air pollutants are projected to decrease, ammonia increases in all RCPs due to the projected increase in population and food demand, and methane increases in RCP8.5 because of its projected rise in livestock and rice production. However, these scenarios follow different pathways in different regions. In some regions, emissions increase to mid-century before decreasing, while in other regions emissions are already decreasing at present and continue decreasing to 2100. Models in the ACCMIP ensemble incorporate chemistry-climate interactions, including mechanisms by which climate change affects ozone and $PM_{2.5}$, although models do not all include the same mechanisms of interactions and do not always agree on the net effect of these interactions (von Schneidemesser et al., 2015).

Using modeled ozone and $PM_{2.5}$ concentrations from the ACCMIP ensemble, we estimate the future premature human mortality associated with exposure to ambient air pollution. Our premature mortality estimates are obtained using a health impact function, combining the relative risk of exposure to changes in air pollution with future exposed population and cause-specific baseline mortality rates. We estimate overall future premature mortality considering the difference in air pollution associated with 2030, 2050 and 2100 emissions and climate relative to that resulting from 2000 emissions and climate. Mortality estimates are obtained at a sufficiently fine horizontal resolution (0.5°x0.5°) to capture both global and regional effects and inform regional and national air quality and climate change policy, but are not expected to capture local scale (e.g., urban) air pollution effects.

## 2 Methods

### 2.1 Ambient ozone and $PM_{2.5}$ concentrations

Concentrations of ozone and $PM_{2.5}$ in surface air are calculated for the present day (2000) and for the 2030, 2050 and 2100 decades for the four RCPs using the output of simulations by the ACCMIP ensemble of chemistry-climate models. As described by Lamarque et al. (2013) not all models are truly coupled chemistry climate models. OsloCTM2 and MOCAGE are chemical transport models driven by offline meteorological fields, and UM-CAM and STOC-HadAM3 do not model the feedback of chemistry on climate.

All ACCMIP models used nearly identical anthropogenic and biomass burning emissions for the present day and future, but they used different natural emissions (e.g. biogenic volatile organic compounds, ocean emissions, soil and lightning $NO_x$), which mostly impacted emissions of ozone precursors (Lamarque et al., 2013; Young et al., 2013) and natural aerosols (i.e., dust and sea salt). Model output shows good agreement with recent observations, both for ozone (Young et al., 2013) and for $PM_{2.5}$ (Shindell et al., 2013), although models tend to overestimate ozone in the Northern Hemisphere and underestimate it in the Southern Hemisphere, and to underestimate $PM_{2.5}$,

particularly in East Asia. Future surface concentrations of air pollutants vary across scenarios and models, but ozone is projected to decrease except in RCP8.5, mostly associated with the large increase in methane concentrations specific to this scenario and the effect of climate change in remote regions (von Schneidemesser et al., 2015; Young et al., 2013).

We obtained hourly and monthly output from the ACCMIP ensemble simulations for a base year (2000) and for future projections under the four RCPs (2030, 2050 and 2100), with each time period corresponding to simulations of up to 10 years, depending on the model. Only two models reported results for all four RCP scenarios and the three future time periods – GFDL-AM3 and GISS-E2-R. $PM_{2.5}$ is calculated as a sum of aerosol species reported by six models (see Supplemental Material), and four of these models also reported their own estimate of total $PM_{2.5}$ (Table S1). Our $PM_{2.5}$ formula includes nitrate; since this species was reported by three models only, we calculate the average nitrate concentrations in each cell reported by these models and add this average to $PM_{2.5}$ for the other models, following Silva et al. (2013). We use our $PM_{2.5}$ estimates to obtain all mortality results, and perform a sensitivity analysis using the $PM_{2.5}$ concentrations reported by four models using their own $PM_{2.5}$ formulas, which differed among models, as ~~reported in~~done by Silva et al. (2013). The native grid resolutions of the 14 models varied from 1.9°x1.2° to 5°x5°; we regrid ozone and $PM_{2.5}$ species surface concentrations from each model to a common 0.5°x0.5° horizontal grid to take maximum advantage of how the grids of different models overlap, following Anenberg et al. (2009, 2014) and Silva et al. (2013).

Ozone and $PM_{2.5}$ concentrations are calculated in each grid cell for each model separately. For both pollutants, we use identical metrics to those reported in the epidemiological studies we considered for the health impact assessment (next section):

- Seasonal average of daily 1-hr maximum ozone concentration, for the six consecutive months with highest concentrations in each grid cell;
- Annual average $PM_{2.5}$ concentration.

Among the 14 models, five models reported only monthly ozone concentrations, while the remaining models reported both hourly and monthly values. We calculate the ratio of the seasonal average of daily 1-hr maximum to the annual average of monthly concentrations, for each scenario/year, for those that reported both hourly and monthly concentrations. Then we apply that ratio to the annual average of monthly ozone concentrations for the former five models, as previously done by Silva et al. (2013). The differences in ozone and $PM_{2.5}$ concentrations between future year (2030, 2050 and 2100) and 2000 are shown in Tables S2 and S3, for each model. For ten world regions (Figure S1), we also estimate regional multi-model averages for each scenario/year (Figures S2 and S3).

### 2.2 Health impact assessment

We estimate future air pollution-related cause-specific premature mortality using ~~similar~~ generally the same methods ~~to~~ as those used by Silva et al. (2013)~~, except for our~~ to obtain present-day estimates, but with two important differences: (1) we use ~~of~~ the recently published Integrated Exposure-Response (IER) model for $PM_{2.5}$ (Burnett et al., 2014) instead of a log-linear model (Krewski et al., 2009), and (2) we use projections of population and baseline mortality rates from the International Futures (IFs) integrated modeling system (Hughes et al., 2011).

We apply a health impact function to estimate premature mortality associated with exposure to ozone and $PM_{2.5}$ ambient air pollution *(*$\Delta$*Mort*) in each grid cell: $\Delta Mort = y0 * AF * Pop$, where *y0* is the baseline mortality rate (for the exposed population), $AF = 1 - 1/RR$ is the attributable fraction, *RR* is the relative risk of death attributable to a change in pollutant concentrations, (RR=1 if there is no increased risk of death associated with a change in pollutant concentrations), and *Pop* is the exposed population (adults aged 25 and older). We ~~take the~~ calculate changes in premature mortality by applying the change in pollutant concentrations ~~due to future emissions and climate as the difference between concentrations~~ in each future year (2030, 2050 and 2100) ~~and in~~ relative to year 2000 ~~and calculate the impact of that concentration change on premature mortality~~ concentrations - the present-day state of air pollution - to the future population. To estimate ozone mortality, we apply the exposure-response function to the difference in ozone concentrations, while for $PM_{2.5}$ mortality we apply the exposure-response function to concentrations in each year (future years and 2000) and then subtract the mortality estimates. We therefore estimate 'avoided' / 'excess' premature mortality due to decreases / increases in air pollutant concentrations in the future years relative to 2000 concentrations. This approach differs from a calculation of the global burden of air pollution-related mortality since we use 2000 rather than 1850 concentrations as baseline. We estimate mortality changes due to future concentration changes, relative to the present, to avoid applying the health impact function at very low concentrations where there is less confidence in the exposure-response relationship. For example, the simulated 1850 air pollutant concentrations are often below the lowest measured value of the American Cancer Society study (Jerrett et al., 2009; Krewski et al., 2009). For illustration, we also estimate mortality relative to 1850 concentrations, which could be regarded as global burden of disease calculations, following Silva et al. (2013).

For each model, we estimate ozone-related mortality due to chronic respiratory diseases (RESP), using RR from Jerrett et al. (2009). We also estimate $PM_{2.5}$-related mortality due to ischemic heart disease (IHD), cerebrovascular disease (STROKE), chronic obstructive pulmonary disease (COPD) and lung cancer (LC), using RRs from the IER model (Burnett et al., 2014). We use RR per age group for IHD and STROKE and RR for all-ages for COPD and LC. We apply the IER model instead of RRs from Krewski et al. (2009), used by Silva et al. (2013), as the newer model should better represent the risk of exposure to $PM_{2.5}$, particularly at locations with high ambient concentrations. In the IER model, the concentration-response function flattens off at higher $PM_{2.5}$ concentrations yielding different estimates of excess mortality for identical changes in air pollutant concentrations in less-polluted vs. highly-polluted locations. Specifically, a one unit reduction of air pollution may have a stronger effect on avoided mortality per million people in regions where pollution levels are lower (e.g. Europe, North America, etc.) compared with highly-polluted areas (e.g. East Asia, India, etc.), which would not be the case for a log-linear function (Jerrett et al. 2009; Krewski et al. 2009). Therefore, using the IER model may result in smaller changes in avoided mortality in highly-polluted areas than using the log-linear model.

Each RCP includes its own projection of total population, but not population health characteristics. For all scenarios, we choose to use a common projection of population and baseline mortality rates per age group from the IFs (Figures ~~S4~~ S6 and ~~S5~~S7). IFs projects population and mortality based on UN and WHO projections from 2010 through 2100, per age group and country, mostly based on three drivers – income, education, and technology (Hughes et al., 2011). Population projections from IFs differ from those underlying each RCP, but lie within the

range of the ~~latter~~ RCPs (Figure ~~S4~~S6). In 2030, global total population in IFs is within 0.08% of that reported for RCP2.6, RCP4.5 and RCP6.0 and 5% lower than for RCP8.5; however, in 2100 IFs projects larger global populations than RCP2.6 (+7%), RCP4.5 (+13%) and RCP6.0 (+2%) and considerably lower than RCP8.5 (-27%). IFs projects rising baseline mortality rates for cardiovascular diseases (CVD) and RESP, globally and in most regions~~,~~ (particularly in East Asia and India), reflecting an aging population. By using projections from IFs, we have a single source of population and baseline mortality rates, assuring their consistency and enabling us to isolate the effect of changes in air pollutant concentrations across the RCPs. Had we used the population projections from each scenario, the magnitude of the changes (increases or decreases in premature mortality relative to 2000) would likely increase in RCP8.5, but decrease in RCP2.6, RCP4.5 and RCP6.0. With the exception of Europe, Former Soviet Union (FSU) and East Asia, where population is projected to decrease in 2100 relative to 2000, had we used present-day population and baseline mortality we would have obtained lower estimates for excess or avoided mortality in each scenario/year, as projected increases in population and baseline mortality magnify the impact of changes in air pollutant concentrations. Therefore, we estimate the overall effect of future air pollution (due to changes in emissions and climate change) considering the population that will potentially be exposed to those effects. We also obtain different estimates of changes in future mortality than if we had calculated the global burden in each year, using air pollutant concentrations, population and baseline mortality rates in that year, and subtracted the present-day burden. Our results do not reflect the potential synergistic effect of a warmer climate on air pollution-related mortality, i.e. we do not account for potential changes in the exposure-response relationships at higher temperatures (Pattenden et al. 2010; Wilson et al., 2014 and references therein).

Country-level population projections for 2030, 2050 and 2100 are gridded to 0.5°x0.5° using ArcGIS 10.2 geoprocessing tools, assuming that the spatial distribution of total population within each country is unchanged from the 2011 LandScan Global Population Dataset at approximately 1 km resolution (Bright et al., 2012), and that the exposed population is distributed in the same way as the total population within each country. IFs projections of mortality rates for CVD are used to estimate baseline mortality rates for IHD and STROKE considering their present-day proportion in CVD (using GBD 2010 baseline mortality rates), as are RESP projections for COPD and malignant neoplasms for LC. IFs projections for 2010 are comparable to GBD 2010 (Lozano et al., 2012) estimates for CVD (+0.04%), RESP (+2.5%) and neoplasms (-12%). We estimate the number of deaths per 5-year age group per country using the country level population. The resulting population and baseline mortality per age group at 30"x30" are regridded to the same 0.5°x0.5° grid as the concentrations of air pollutants.

Uncertainty from the RRs is propagated separately for each model-scenario-year to mortality estimates in each grid cell, through 1000 Monte Carlo (MC) simulations, i.e. we repeat the calculations in each grid cell ~~for~~ 1000 times using random sampling of the RR variable. For ozone, we use the reported 95% Confidence Intervals (CIs) for RR (Jerrett et al., 2009) and assume a normal distribution, while for $PM_{2.5}$ we use the ~~parameter~~ values for the parameters alpha, gamma, delta and $z_{cf}$ (counterfactual) reported by Burnett et al. (2014) for 1000 MC simulations (GHDx 2013). Then for each of the 1000 simulations, we add mortality over many grid cells to obtain regional and global mortality and estimate the empirical mean and 95% CI of the regional and global mortality results. We assume no correlation between the RRs for the four causes of death; thus we may underestimate the overall

uncertainty for PM2.5 mortality estimates. Uncertainty in air pollutant concentrations is based on the spread of model results by calculating the average and 95% CI for the pooled results of the 1000 MC simulations for each model. This estimate of uncertainty in concentrations does not account for uncertainty in emissions inventories (as the ensemble used identical emissions) or for potential bias in modelled air pollutant concentrations. We also estimate the contribution of ~~uncertainty~~ uncertainties in RR and ~~uncertainty~~ in air pollutant concentrations to the overall uncertainty in mortality estimates using a tornado analysis; we obtained global mortality estimates treating each variable as uncertain individually (year 2000 concentrations, future year concentrations, RR for ozone and the four parameters in the IER model for $PM_{2.5}$) and used central estimates for all other variables, and then calculated the contribution of each variable to the overall uncertainty (when all variables are treated as uncertain simultaneously). Uncertainties associated with population and baseline mortality rates are not reported by IFs, and are not considered in the uncertainty analysis.

## 3 Results

First, we present our estimates of ozone and $PM_{2.5}$-related excess/avoided premature mortality in 2030, 2050 and 2100 for changes in pollutant concentrations between 2000 and each future period, for the four RCPs (sections 3.1 and 3.2, Figures 1 to 7). Figures 1 and 4 show global mortality for the different ACCMIP models. The multi-model average mortality results are shown for individual grid cells (Figures 2 and 5) and for regional totals (Figures 3 and 6). Finally, we include our estimates of the global mortality burden of both air pollutants for future concentrations relative to 1850 concentrations (section 3.3, Figures 8 and 9). In some cases, ~~avoided/excess~~ the changes in future mortality due to ~~a reduction/increase~~ changes in future concentrations relative to 2000 ~~concentrations~~ show~~s~~ a different trend than the global mortality burden~~, ; which~~ this difference reflects the combined effects of future changes in concentrations relative to 1850, exposed population and baseline mortality rates.

### 3.1 Ozone-related future premature mortality

We find that future changes in ozone concentrations are associated with excess global premature mortality due to respiratory diseases in 2030, but avoided mortality by 2100 for all scenarios but RCP8.5 (Figure 1, Table ~~S4~~S5). In 2030, all RCPs show excess multi-model average ozone mortality~~: ~~, ranging from 11,900 (RCP2.6)~~, 100,000 (RCP4.5), 71,200 (RCP6.0) and~~to 264,000 (RCP8.5) deaths/year. For each RCP, however, some models yield avoided mortality in 2030. In 2050, ~~we estimate avoided ozone mortality for RCP2.6 ( 450,000 deaths/year) and RCP4.5 ( 360,000 deaths/year) and excess ozone mortality for RCP6.0 (441,000 deaths/year) and RCP8.5 (246,000 deaths/year); these~~ multi-model averages are obtained from only 3 or 4 models, depending on the scenario, which makes it difficult to compare with the other two periods. In 2100, we estimate excess ozone mortality in RCP8.5 (316,000 deaths/year), but avoided ozone mortality for the other three RCPs from -1.02 million (RCP2.6) to -718,000 (RCP6.0) deaths/year with all models agreeing in sign of the change~~: 1.02 million (RCP2.6), 917,000 (RCP4.5) and 718,000 (RCP6.0) deaths/year~~.

~~For RCP8.5, we propagate input uncertainty to the mortality estimates (Figure 1, Table S4). Global future premature mortality rises from 264,000 ( 39,300 to 648,000) deaths in 2030 to 316,000 ( 187,000 to 1.38 million) deaths in~~

~~2100. Uncertainty in RR leads to coefficients of variation (CV) ranging from 31 to 37% (2030), 31 to 40% (2050) and 16 to 47% (2100) for the different models. Considering the spread of model results, overall CV for the multi-model average mortality increases to 66% (2030), 78% (2050) and 125% (2100). While uncertainty in RR and in modeled ozone concentrations have similar contributions to overall uncertainty in mortality results in 2050 (51% and 49%, respectively), in 2030 modeled ozone concentrations are the greatest contributor (81%), and in 2100 uncertainty in RR contributes the most to overall uncertainty (88%). For 2030, HadGEM2 differs in sign from the other 13 models with (avoided) global mortality totalling -33,900 deaths/year. For 2050, LMDzORINCA differs substantially from the other 3 models with -38,900 deaths/year. For 2100, HadGEM2 is a noticeable outlier with 1.2 million excess deaths/year and MOCAGE differs in sign from the other 12 models with -159,000 deaths/year.~~

Excess ozone-related future premature mortality (Figures 2 and 3, Table ~~S5~~S6) is noticeable in some regions ~~through~~ in 2030 for all RCPs, particularly in India and East Asia for RCP8.5 (~~124,000 and 127,000 deaths/year, respectively~~over 95% of global excess mortality), but all scenarios except RCP8.5 show avoided global ozone-related mortality in 2100. Under this scenario in 2100, there are increases in ozone concentrations in all regions except North America, East Asia and Southeast Asia (Figure S2), likely driven by the projected large increase in methane emissions as well as by climate change. Avoided mortality in those three regions ~~of over 140,000 deaths/year~~ is outweighed by excess mortality in India ~~(292,000 deaths/year)~~, Africa ~~(128,000 deaths/year)~~ and the Middle East ~~(29,800 deaths/year)~~. Also, some regions show different trends in future mortality relative to 2000 depending on the RCP, reflecting the effects of distinct assumptions in each RCP about economic growth and air pollution control with different trends in regional ozone precursor emissions. For example, North America and Europe show decreases in mortality through 2100 in all scenarios, except a slight increase in Europe for RCP8.5 in 2100. In East Asia, mortality peaks in 2050 for RCP6.0, driven by peak precursor emissions in 2050 in this scenario, but it peaks in 2030 for the other three RCPs. India shows peaks in mortality in 2050 followed by decreases for all RCPs but RCP8.5, in which mortality increases through 2100. Africa shows increases in mortality through 2100 for RCP2.6 and RCP8.5, while it peaks in 2050 for RCP4.5 and decreases through 2100 for RCP6.0. Also, the effect of changes in population and baseline mortality rates is noticeable in some regions when comparing the trends in total ozone-related mortality and mortality per million people in each region (Figure ~~S6~~S10). For example, decreases in population projected for 2100 in Europe, FSU and East Asia, are reflected in greater changes in mortality per million people than in total mortality, while the threefold increase in population in Africa amplifies the changes in total mortality.

For RCP8.5, we propagate input uncertainty to the mortality estimates (Figure 1, Table S45). Global future premature mortality ~~rises~~changes from 264,000 (-39,300 to 648,000) deaths in 2030 to 316,000 (-187,000 to 1.38 million) deaths in 2100. Uncertainty in RR leads to coefficients of variation (CV) ranging from 31 to 37% (2030), 31 to 40% (2050) and 16 to 47% (2100) for the different models. Considering the spread of model results, overall CV for the multi-model average mortality increases to 66% (2030), 78% (2050) and 125% (2100). While uncertainty in RR and in modeled ozone concentrations have similar contributions to overall uncertainty in mortality results in 2050 (51% and 49%, respectively), in 2030 modeled ozone concentrations are the greatest contributor (81%), and in 2100 uncertainty in RR contributes the most to overall uncertainty (88%). For 2030, HadGEM2

differs in sign from the other 13 models with (avoided) global mortality totalling -33,900 deaths/year. For 2050, LMDzORINCA differs substantially from the other 3 models with -38,900 deaths/year. For 2100, HadGEM2 is a noticeable outlier with 1.2 million excess deaths/year and MOCAGE differs in sign from the other 12 models with -159,000 deaths/year.

### 3.2 $PM_{2.5}$-related future premature mortality

Global $PM_{2.5}$-related premature mortality, considering the difference in future concentrations and 2000 concentrations, decreases substantially in most scenarios, particularly in 2100 (Figure 4, Table ~~S6~~S7). In 2030, the multi-model average varies from -289,000 (RCP4.5) to 17,200 (RCP8.5) ~~–~~deaths/year, although one model (CICERO-OsloCTM2) shows excess mortality for RCP2.6 and RCP8.5. In 2050, substantial avoided mortality is estimated for all scenarios except RCP6.0 which shows a small increase in mortality (16,700 deaths/year), but this is the average of only three models that do not agree on the sign of the change. In 2100, all scenarios show considerable avoided mortality, ranging from -1.31 million (RCP8.5) to -2.39 million (RCP4.5) deaths/year, reflecting the substantial decrease in emissions of primary $PM_{2.5}$ and precursors~~: -1.93 million (RCP2.6), -2.39 million (RCP4.5), -1.76 million (RCP6.0) and -1.31 million (RCP8.5) deaths/year~~.

In several regions (North America, South America, Europe, FSU and Australia), $PM_{2.5}$ future premature mortality decreases through 2100 for all RCPs (Figures 5 and 6, Table S7). However, in East Asia, Southeast Asia, India, Africa, and the Middle East, for some scenarios, $PM_{2.5}$ mortality increases through 2030 or 2050~~,~~ before decreasing. ~~In East Asia, mortality peaks in 2030 for RCP8.5 and in 2050 for RCP6.0. In Southeast Asia, mortality peaks in 2030 for RCP2.6 and in 2050 for RCP6.0. In India, mortality peaks in 2030 or 2050 for all RCPs except RCP8.5 which still shows an increase in 2100. In Africa, mortality increases through 2100 for RCP2.6 and RCP8.5, but for RCP4.5 it peaks in 2050 and for RCP6.0 it decreases through 2100.~~ The changes in future mortality reflect changes in future $PM_{2.5}$ concentrations relative to 2000 (Figure S3), and a substantial increase in exposed population through the 21st century, particularly in Africa, India and the Middle East (Figure S~~4~~6). That is, any reduction/increase in mortality due to the decrease/increase in pollutant concentrations was amplified by the increases in exposed population. The decreases in population in Europe, FSU and East Asia have similar effects as those mentioned above for ozone-related mortality. For example, while total avoided mortality in 2100 in East Asia decreases compared to 2050, for RCP2.6, RCP4.5 and RCP8.5, total avoided mortality per million people increases in the same scenarios (Figure S~~7~~11). East and South Asia are the regions with the greatest projected mortality burdens, and the variability in $PM_{2.5}$ among models is typically less in these regions than in several other regions globally, depending upon the scenario and year (Figure S9).

Future $PM_{2.5}$-related mortality estimates are influenced by the nonlinearity of the IER function. For example, in RCP8.5 in 2030, all models project an increase in global population-weighted concentration (Table S3) but all models except one show decreases in global $PM_{2.5}$-related mortality (Figure 4). This outcome results in part because $PM_{2.5}$ increases are projected in regions with high concentrations (particularly East Asia) that are on the flatter part of the IER curve, whereas $PM_{2.5}$ decreases in regions with low concentrations (North America and Europe) have a steeper slope and therefore a greater influence on global mortality.

Considering the results of the MC simulations for RCP8.5, premature mortality changes from -17,200 (-386,000 to 661,000) deaths in 2030 to -1.31 (-2.04 to -0.17) million deaths in 2100 (Figure 4, Table ~~S6~~S7). Uncertainty in RR leads to a CV of 11 to 191% for the different models in the three future years. The spread of model results increases overall CV to 1644% (2030), 20% (2050) and 41% (2100). Uncertainty in modeled $PM_{2.5}$ concentrations in 2000 is the greatest contributor to overall uncertainty (59% in 2030, 45% in 2050, and 49% in 2100), followed by uncertainty in modeled $PM_{2.5}$ in future years (40% in 2030, 26% in 2050 and 32% in 2100). Uncertainty in RR has a negligible contribution to overall uncertainty in 2030 (<1%), as the multi-model mean mortality change happens to be near zero (one model projects a large increase while the other five models project decreases), but contributes 29% in 2050 and 20% in 2100.

~~In several regions (North America, South America, Europe, FSU and Australia), $PM_{2.5}$ future premature mortality decreases through 2100 for all RCPs (Figures 5 and 6, Table S7). However, in East Asia, Southeast Asia, India, Africa, and the Middle East, for some scenarios, $PM_{2.5}$ mortality increases through 2030 or 2050, before decreasing. In East Asia, mortality peaks in 2030 for RCP8.5 and in 2050 for RCP6.0. In Southeast Asia, mortality peaks in 2030 for RCP2.6 and in 2050 for RCP6.0. In India, mortality peaks in 2030 or 2050 for all RCPs except RCP8.5 which still shows an increase in 2100. In Africa, mortality increases through 2100 for RCP2.6 and RCP8.5, but for RCP4.5 it peaks in 2050 and for RCP6.0 it decreases through 2100. The changes in future mortality reflect changes in future $PM_{2.5}$ concentrations relative to 2000 (Figure S3), and a substantial increase in exposed population through the 21st century, particularly in Africa, India and the Middle East (Figure S4). The decreases in population in Europe, FSU and East Asia have similar effects as those mentioned above for ozone-related mortality. For example, while total avoided mortality in 2100 in East Asia decreases compared to 2050, for RCP2.6, RCP4.5 and RCP8.5, total avoided mortality per million people increases in the same scenarios (Figure S7).~~

We compared mortality results using our ~~own~~ estimates of $PM_{2.5}$ from the sum of reported species with results using $PM_{2.5}$ reported by four models applying their own formula to estimate $PM_{2.5}$ (Figure 7). The multi-model average future avoided mortality for the four models which reported $PM_{2.5}$ is comparable although lower than the average for our $PM_{2.5}$ estimates for the same models. Individual models do not show the same differences in mortality using their own vs. our $PM_{2.5}$ estimates. Also, for two models (GFDL-AM3 and MIROC-CHEM) the two sources of $PM_{2.5}$ estimates yield mortality changes of different sign in 2030. These results reflect the different aerosol species included by each model to estimate $PM_{2.5}$ (e.g. nitrate is not included by all models).

### 3.3 Global burden on mortality of ozone and $PM_{2.5}$

Here we present estimates of the global burden on mortality of ozone and $PM_{2.5}$ concentrations in the future, considering the four RCPs relative to preindustrial concentrations (1850) and future exposed population and baseline mortality rates (Figures 8 and 9, Tables S8 and S9). For context, we estimate the present-day global burden, using ~~in~~ 2000 concentrations, ~~(using present-day~~ population from Landscan 2011 Population Dataset, and baseline mortality rates from GBD2010~~),~~ to be: 382,000 (121,000 to 728,000) ozone deaths/year and 1.70 (1.30 to 2.10) million $PM_{2.5}$ deaths/year. These estimates are ~~19%~~ 18.7% lower for ozone-related mortality and 19.1% lower for $PM_{2.5}$-related

mortality than those obtained in our previous study (Silva et al., 2013), reflecting; a) more restrictive mortality outcomes (chronic respiratory diseases rather than all respiratory diseases, and IHD+STROKE+COPD rather than all cardiopulmonary diseases~~), ~~); b) updated population and baseline mortality rates~~, and~~; c) the use of the recent IER model (Burnett et al., 2014) for $PM_{2.5}$ (instead of Krewski et al., 2009). Compared with the GBD ~~2010~~ 2013 (Forouzanfar et al. 2015)~~results, these~~, our estimates are ~~15~~17~~6~~% higher ~~than~~ for ozone-related mortality and ~~47~~42% lower ~~than~~ for $PM_{2.5}$-related mortality ~~reported by Lim et al. (2012)~~, likely due to the fact that we estimate the global mortality burden using 1850 concentrations as baseline, while Forouzanfar ~~Lim~~ et al. (2015) consider counterfactual concentrations (theoretical minimum-risk exposure) that are mostly higher for ozone (uniform distribution between 33.3 and 41.9 ppb) and lower for $PM_{2.5}$ (uniform distribution between 5.~~8~~ 9 and 8.~~8~~ 7 μg/m$^3$) than 1850 concentrations. In addition, we consider ozone mortality from all chronic respiratory diseases while Forouzanfar et al. (2015) only account for COPD, and we restrict our mortality estimates to adult population while Forouzanfar et al. (2015) include $PM_{2.5}$ mortality from lower respiratory tract infections in children under 5 years old. As a sensitivity analysis, when we apply a counterfactual of 33.3ppb (instead of using 1850 concentrations), our ozone-related mortality estimates are 23% higher for the multi-model mean, varying between +10% and +52% among models. Similarly, using the IER model counterfactual, our $PM_{2.5}$-related mortality estimates are 22% lower for the multi-model mean, varying between -8% and -44% among models.

For ozone, the global mortality burden increases in all RCPs through 2050 to between 1.84 and 2.60 million deaths/year, and then it decreases slightly for RCP8.5 and substantially for the other RCPs, ranging between 1.09 and 2.36 million deaths/year in 2100. The increase can be explained by the rise in the baseline mortality rates for chronic respiratory diseases magnified by the increase in exposed population, while the decline is likely mostly related to the decrease in concentrations, slightly countered by further population growth (Figure 8). The global burden of mortality from $PM_{2.5}$ shows a declining trend for all RCPs from 2030 to 2100, peaking between 2.4 and 2.6 million deaths/year in 2030 then declining to between 0.56 and 1.55 million deaths/year in 2100, except for RCP6.0 which peaks in 2050 (3.50 million deaths/year) before declining considerably. For $PM_{2.5}$, the increase in exposed population and the decline in concentrations have a much greater effect than changes in baseline mortality rates (Figure 9). These results are similar to those of Apte et al. (2015) who report a stronger effect of projected demographic trends in India and China in 2030 than of changes in baseline mortality rates. Our estimates for the global burden of $PM_{2.5}$ mortality in 2050 (between 1.82 and 3.50 million deaths/year for the four RCPs) are considerably lower than those of Lelieveld et al. (2015) (5.87 million deaths / year for IHD+STROKE+COPD+LC), likely due to the assumption in the RCP scenarios of further regulations on air pollutants, while the Business-As-Usual scenario of Lelieveld et al. (2015) does not assume regulations beyond those currently defined.

To help explain differences between the trends in future global burden (Figures 8 and 9) and in future mortality relative to 2000 (Figures 1 and 4), we estimate the future global burden for two cases: Case A - using 2000 concentrations relative to 1850 and present-day population but future baseline mortality rates; and Case B – using 2000 concentrations relative to 1850 but future population and baseline mortality rates. Case A reflects the effect of future baseline mortality rates on the global burden, if concentrations in future years were maintained at 2000 levels, while Case B reflects the combined effect of population and baseline mortality rates, i.e. it is identical to Case A

except that population changes. The difference between the global burden for each RCP and Case B reflects the effects of changes in future air pollutant concentrations, and nearly equals future mortality relative to 2000 concentrations in Figures 1 and 4. However, Cases A and B are calculated for all 14 models for ozone and 6 models for $PM_{2.5}$ (since all models reported air pollutant concentrations in 2000), while future mortality relative to 2000 is calculated for the models that report each scenario/year.

## 4 Discussion

In all RCP scenarios but RCP8.5, stringent air pollution controls lead to substantial decreases in ozone concentrations through the 21st century, relative to 2000. For RCP8.5, the higher baseline GHG (including methane) and air pollutant emissions lead to increases in future ozone concentrations. In contrast, global $PM_{2.5}$ concentrations show a decreasing trend across all RCP scenarios. These changes in air pollutant concentrations, combined with projected increases in baseline mortality rates for chronic respiratory diseases, drive ozone mortality to become more important relative to $PM_{2.5}$ mortality over the next century.

The importance of conducting health impact assessments with air pollutant concentrations from model ensembles, instead of from single models, is highlighted by the differences in sign of the change in mortality among models, and by the marked impact of the spread of model results on overall uncertainty in our mortality estimates. In most cases assessed here (ozone mortality in 2030 relative to 2000, $PM_{2.5}$ mortality in 2030, 2050 and 2100 relative to 2000), uncertainty in modeled air pollutant concentrations is the greatest contributor to uncertainty in mortality estimates. ~~Uncertainty in future ozone mortality in 2050 relative to 2000 has comparable contributions from uncertainty in RR and in modeled concentrations, while in 2100 uncertainty in RR contributes the most to overall uncertainty in ozone mortality.~~ The differences in air pollutant concentrations reported by the ACCMIP models reflect different treatment of atmospheric dynamics and chemistry, chemistry-climate interactions, and natural emissions in each model (Young et al., 2013). Although there is likely a bias in estimating health effects using air pollutant concentrations from coarse resolution models (Li et al., 2015; Punger and West, 2013), particularly for $PM_{2.5}$, we do not expect resolution to be an important factor for the differences in simulated concentrations across coarse resolution models.

There are several uncertainties and assumptions that affect our results. We applied the same RR worldwide and into the future, despite differences in vulnerability of the exposed population, in composition of $PM_{2.5}$, and in other factors that may support the use of different risk estimates or different concentration-response relationships. These uncertainties can be addressed through additional long-term epidemiological studies, particularly for large cohorts in developing countries, to improve RR estimates globally. These studies should be representative of wider ranges of exposure and air pollutant mixtures than existing studies in the US and Europe, and they should control for confounding factors such as other environmental exposures, use of air conditioning, socio-economic factors, etc. Also, we estimate mortality for adults aged 25 and older, and do not quantify air pollutant effects on morbidity, so we underestimate the overall impact of changes in pollutant concentrations on human health. ~~Uncertainties in projections of~~ Uncertainty is evaluated for a single future population projection, not accounting for the wide range of projections in the literature, and does not reflect uncertainty in ~~and~~ baseline mortality rates, as these are not reported;

uncertainties in both population and baseline mortality rates would be expected to increase with time into the future. ~~were not included in our estimates of uncertainty, and t~~.The spread of model results does not account for uncertainty in emissions inventories, as all ACCMIP models used the same ~~central estimate~~projections of anthropogenic emissions. Moreover, climate and air quality interactions and feedbacks are not sufficiently understood to be fully reflected in modeled air pollutant concentrations, and global models simplify atmospheric physics and chemical processes. This is particularly important when modeling air quality given scenarios of future emissions and climate change. For example, most global models do not fully address climate sensitivity to biogenic emissions (e.g. isoprene, soil NOx and methane) and stratosphere-troposphere interactions (e.g. stratospheric influx of ozone). A better understanding of aerosol-cloud interactions, of the impact of climate change on wildfires, and of the impact of land use changes on regional climate and air pollution is also crucial.

~~These uncertainties can be addressed through additional long-term epidemiological studies, particularly for large cohorts in developing countries, to improve RR estimates globally. These studies should be representative of wider ranges of exposure and air pollutant mixtures than existing studies in the US and Europe, and they should control for confounding factors such as other environmental exposures, use of air conditioning, socio-economic factors, etc.~~

Our results are limited by the range of ~~projected~~ air pollutant emissions projected ~~given~~ by the RCPs, which assume that economic growth strengthens efforts to reduce air pollution emissions. All RCPs ~~consider~~ project reductions in anthropogenic precursor emissions associated with more extensive air quality legislation as incomes rise, except for methane in RCP8.5 and for ammonia in all scenarios. ~~-~~These scenarios together do not encompass the range of plausible air pollution futures for the 21st century, as the RCPs were not designed for this purpose (van Vuuren et al., 2011a). ~~-~~Other plausible scenarios have been considered, such as the Current Legislation Emissions and Maximum Feasible Reductions scenarios used by Likhvar et al. (2015) and the Business-As-Usual scenario ~~considered by~~of Lelieveld et al (2015). As noted above, our global burden estimates for 2050 are considerably lower than the Business-As-Usual scenario of Lelieveld et al. (2015). If economic growth does not lead to stricter air pollution control, emissions and health effects may rise considerably, particularly for scenarios of high population growth in developing countries (Amman et al., 2013).

## 5 Conclusions

Under the RCP scenarios, future $PM_{2.5}$ concentrations ~~are calculated to result in~~lead to decreased global premature mortality versus what would occur with fixed year-2000 concentrations, but ozone-related mortality increases in some scenarios/periods. In 2100, excess ozone-related premature mortality for RCP8.5 is estimated to be 316 thousand (-187 thousand to 1.38 million) deaths/year (likely due to an increase in methane emissions and to the net effect of climate change), while for the three other RCPs ~~there is~~ avoided ozone mortality is between ~~-~~ -718 thousand and -1.02 million deaths/year. For $PM_{2.5}$, avoided future premature mortality is estimated to be between -1.33 and -2.39 million deaths/year in 2100. These reductions in ambient air pollution-related mortality reflect the decline in ~~most~~ pollutant emissions projected in the RCPs, but the large range of results from the four RCPs highlights the importance of future air pollutant emissions for ambient air quality and global health. Mortality estimates differ among models and we find that, for most cases, the contribution to overall uncertainty from uncertainty associated

with modeled air pollutant concentrations exceeds that from the RRs. Increases in exposed population and in baseline mortality rates of respiratory diseases magnify the impact on mortality of the changes in air pollutant concentrations.

Estimating future mortality relative to 2000 concentrations allows us to emphasize the effects of changes in air pollution in these results. However, increases in exposed population and in baseline mortality rates may drive an increase in the future burden of air pollution on mortality. Even in the most optimistic scenarios, the global mortality burden of ozone (relative to 1850 concentrations) is estimated to be over 1 million deaths/year in 2100, compared to less than 0.4 million in 2000~~ (Figure 8)~~. For $PM_{2.5}$, the global burdens in 2030 and 2050 for the four RCPs are greater than the global burden in 2000~~: between 2.4 and 2.6 million deaths/year in 2030 and between 1.8 and 3.5 million deaths/year in 2050~~ but decrease to between 0.56 and 1.55 million deaths/year in 2100, compared to 1.7 million deaths/year in 2000~~ (Figure 9)~~. A strong decline in $PM_{2.5}$ concentrations for all RCPs together with demographic trends in the 21st century (with a projected substantial increase in exposed population) lead to a rising importance of ozone relative to $PM_{2.5}$ for the global burden of ambient air pollution-related mortality.

The RCPs are based on the premise that economic development drives better air pollution control, leading to improved air quality. This trend is apparent in some developing countries now (Klimont et al., 2013), but it is yet to be determined how aggressive many developing nations will be in addressing air pollution. The assumed link between economic development and air pollution control in the RCPs requires new and stronger regulations around the world, as well as new control technologies, for the air pollution decreases in the RCPs to be realized. The projected reductions in mortality estimated here will be compromised if more stringent policies are delayed (e.g., Lelieveld et al., 2015).

**Acknowledgements**

The research here described was funded by a fellowship from the Portuguese Foundation for Science and Technology, by a Dissertation Completion Fellowship from The Graduate School (UNC – Chapel Hill), and by NIEHS grant #1 R21 ES022600-01. We thank Karin Yeatts (Department of Epidemiology, UNC – Chapel Hill) for her help in researching projections of future population and baseline mortality rates, Colin Mathers (WHO) for advising us on the IFs, Peter Speyer (IHME, University of Washington) for providing us access to GBD2010 cause-specific mortality data at the country-level, and Amanda Henley (Davis Library Research & Instructional Services, UNC – Chapel Hill) for facilitating our access to Landscan 2011 Global Population Dataset. The work of DB and PC was funded by the U.S. Dept. of Energy (BER), performed under the auspices of LLNL under Contract DE-AC52-07NA27344, and used the supercomputing resources of NERSC under contract No. DE-AC02-05CH11231. Ruth Doherty, Ian MacKenzie and David Stevenson acknowledge ARCHER supercomputing resouces and funding under the UK Natural Environment Research Council grant: NE/I008063/1. GZ acknowledges the NZ eScience Infrastructure which is funded jointly by NeSI's collaborator institutions and through the MBIE's Research Infrastructure programme.

**References**


Amann, M., Klimont, Z., and Wagner, F.: Regional and Global Emissions of Air Pollutants: Recent Trends and Future Scenarios, Annu. Rev. Environ. Resour., 38, 31–55, doi: 10.1146/annurev-environ-052912-173303, 2013.

Anenberg, S. C., West, J. J., Fiore, A. M., Jaffe, D. A., Prather, M. J., Bergmann, D., Cuvelier, K., Dentener, F. J., Duncan, B. N., Gauss, M., Hess, P., Jonson, J. E., Lupu, A., MacKenzie, I.A., Marmer, E., Park, R. J., Sanderson, M.G., Schultz, M., Shindell, D. T., Szopa, S., Vivanco, M. G., Wild, O., and Zeng, G.: Intercontinental Impacts of Ozone Pollution on Human Mortality, Environ. Sci. Technol., 43, 6482–6487, 2009.

Anenberg, S. C., Horowitz, L. W., Tong, D. Q. and West, J. J.: An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling, Environ. Health Perspect., 118(April), 1189–1195, doi:10.1289/ehp.0901220, 2010.

Anenberg, S. C., West, J. J., Yu, H., Chin, M., Schultz, M., Bergmann, D., Bey, I., Bian, H., Diehl, T., Fiore, A., Hess, P., Marmer, E., Montanaro, V., Park, R., Shindell, D., Takemura, T., and Dentener, F.: Impacts of intercontinental transport of anthropogenic fine particulate matter on human mortality. Air. Qual. Atmos. Health., doi: 10.1007/s11869-014-0248-9, 2014.

Apte, J. S., Marshall, J. D., Cohen, A. J., and Brauer, M.: Addressing Global Mortality from Ambient $PM_{2.5}$. Environ. Sci. Technol., 49, 8057−8066, doi: 10.1021/acs.est.5b01236, 2015.

Bell, M. L., Dominici, F. and Samet, J. M.: A Meta-Analysis of Time-Series Studies of Ozone and Mortality With Comparison to the National Morbidity, Mortality, and Air Pollution Study, Epidemiology, 16(4), 436–445, 2005.

Bell, M. L., Goldberg, R., Hogrefe, C., Kinney, P. L., Knowlton, K., Lynn, B., Rosenthal, J., Rosenzweig, C. and Patz, J. A.: Climate change, ambient ozone, and health in 50 US cities, Clim Change, 82(1-2), 61–76, doi:10.1007/s10584-006-9166-7, 2007.

Bright, E. A., Coleman, P. R., Rose, A. N. and Urban, M. L.: LandScan 2011, Oak Ridge National Laboratory SE, Oak Ridge, TN, 2012.

Brook, R. D., Rajagopalan, S., Pope, C. A., Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., Holguin, F., Hong, Y., Luepker, R. V., Mittleman, M. A., Peters, A., Siscovick, D., Smith, S. C., Whitsel, L. and Kaufman, J. D.: Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association, Circulation, 121(21), 2331–2378, doi:10.1161/CIR.0b013e3181dbece1, 2010.

Burnett, R. T., Arden Pope, C., Ezzati, M., Olives, C., Lim, S. S., Mehta, S., Shin, H. H., Singh, G., Hubbell, B., Brauer, M., Ross Anderson, H., Smith, K. R., Balmes, J. R., Bruce, N. G., Kan, H., Laden, F., Prüss-Ustün, A., Turner, M. C., Gapstur, S. M., Diver, W. R. and Cohen, A.: An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure, Environ Health Perspect, 122(4), 397–403, doi:10.1289/ehp.1307049, 2014.

Chang, H. H., Zhou, J. and Fuentes, M.: Impact of Climate Change on Ambient Ozone Level and Mortality in Southeastern United States, Int. J. Environ. Res. Public Health 7(7), 2866-2880, doi:10.3390/ijerph7072866, 2010.

Doherty, R.M., Wild, O., Shindell, D.T., Zeng, G., MacKenzie, I.A., Collins, W.J., Fiore, A.M., Stevenson, D.S., Dentener, F.J., Schultz, M.G., Hess, P., Derwent, R.G. and Keating, T.J.: Impacts of climate change on surface ozone and intercontinental ozone pollution: A multi-model study. J. Geophys. Res. Atmos., 118(9), 3744-3763, doi:10.1002/jgrd.50266, 2013.

Evans, J., van Donkelaar, A., Martin, R. V., Burnett, R., Rainham, D. G., Birkett, N. J. and Krewski, D.: Estimates of global mortality attributable to particulate air pollution using satellite imagery, Environ Res, 120, 33–42, doi:10.1016/j.envres.2012.08.005, 2013.

Fang, Y., Naik, V., Horowitz, L. W. and Mauzerall, D. L.: Air pollution and associated human mortality: The role of air pollutant emissions, climate change and methane concentration increases from the preindustrial period to present, Atmos Chem Phys, 13, 1377–1394, doi:10.5194/acp-13-1377-2013, 2013a.

Fang, Y., Mauzerall, D. L., Liu, J., Fiore, A. M., and Horowitz, L. W.: Impacts of 21st century climate change on global air pollution-related premature mortality, Climatic Change,, doi: 10.1007/s10584-013-0847-8, 2013b.

Fann, N., Nolte, C. G., Dolwick, P., Spero, T. L., Brown, A. C., Phillips, S. and Anenberg S.: The geographic distribution and economic value of climate change-related ozone health impacts in the United States in 2030, Journal of the Air & Waste Management Association, 65:5, 570-580, doi: 10.1080/10962247.2014.996270, 2015.

Fiore, A. M., Naik, V., Spracklen, D. V., Steiner, A., Unger, N., Prather, M., Bergmann, D., Cameron-Smith, P. J., Cionni, I., Collins, W. J., Dalsøren, S., Eyring, V., Folberth, G. a., Ginoux, P., Horowitz, L. W., Josse, B., Lamarque, J.-F., MacKenzie, I. a., Nagashima, T., O'Connor, F. M., Righi, M., Rumbold, S. T., Shindell, D. T., Skeie, R. B., Sudo, K., Szopa, S., Takemura, T. and Zeng, G.: Global air quality and climate, Chem Soc Rev, 41(19), 6663-6683, doi:10.1039/c2cs35095e, 2012.

Fiore, A. M., Naik, V. and Leibensperger, E. M.: Air Quality and Climate Connections, J Air Waste Manage Assoc, 65(6), 645–685, doi:10.1080/10962247.2015.1040526, 2015.

Forouzanfar, M. H et al.: Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013, The Lancet , 386(10010), 2287 – 2323, doi: 10.1016/S0140-6736(15)00128-2, 2015.

Fuzzi, S., Baltensperger, U., Carslaw, K., Decesari, S., Denier van der Gon, H., Facchini, M. C., Fowler, D., Koren, I., Langford, B., Lohmann, U., Nemitz, E., Pandis, S., Riipinen, I., Rudich, Y., Schaap, M., Slowik, J. G., Spracklen, D. V., Vignati, E., Wild, M., Williams, M., and Gilardoni, S.: Particulate matter, air quality and climate: lessons learned and future needs, Atmos. Chem. Phys., 15, 8217-8299, doi:10.5194/acp-15-8217-2015, 2015.

Global Health Data Exchange (GHDx). 2013. Global Burden of Disease Study 2010 - Ambient Air Pollution Risk Model 1990-2010. Available: http://ghdx.healthmetricsandevaluation.org/record/global-burden-disease-study-2010-gbd-2010-ambient-air-pollution-risk-model-1990-2010 [accessed 8 November 2013].

Gryparis, A., Forsberg, B., Katsouyanni, K., Analitis, A., Touloumi, G., Schwartz, J., Samoli, E., Medina, S., Anderson, H. R., Niciu, E. M., Wichmann, H. E., Kriz, B., Kosnik, M., Skorkovsky, J., Vonk, J. M. and Dörtbudak, Z.: Acute effects of ozone on mortality from the "Air Pollution and Health: A European Approach" project, Am J Respir Crit Care Med, 170(30), 1080–1087, doi:10.1164/rccm.200403-333OC, 2004.

Hamra, G. B., Guha, N., Cohen, A., Laden, F., Raaschou-Nielsen, O., Samet, J. M., Vineis, P., Forastiere, F., Saldiva, P., Yorifuji, T. and Loomis, D.: Outdoor Particulate Matter Exposure and Lung Cancer: A Systematic Review and Meta-Analysis., Environ Health Perspect, (January), doi:10.1289/ehp.1408092, 2014.

Heal, M., Doherty, R., Heaviside, C., Vieno, M., Stevenson, D., Vardoulakis, S.: Health Effects due to Changes in Air Pollution under Future Scenarios, Health effects of climate change in the UK 2012, Chapter 3, 55–82, 2012.

Hughes, B. B., Kuhn, R., Peterson, C. M., Rothman, D. S., Solórzano, J. R., Mathers, C. D. and Dickson, J. R.: Projections De Résultats En Matière De Santé Mondiale De 2005 À 2060 En Utilisant Le Modèle Prévisionnel Intégré International Futures, Bull World Health Organ, 89(7), 478–486, doi:10.2471/BLT.10.083766, 2011.

Jackson, J. E., Yost, M. G., Karr, C., Fitzpatrick, C., Lamb, B. K., Chung, S. H., Chen, J., Avise, J., Rosenblatt, R. A., and Fenske, R. A.: Public health impacts of climate change in Washington State: projected mortality risks due to heat :159–186, doi: 10.1007/s10584-010-9852-3, 2010.

Jacob, D. J. and Winner, D. A.: Effect of climate change on air quality, Atmos Environ, 43(1), 51–63, doi:10.1016/j.atmosenv.2008.09.051, 2009.

Jerrett, M., Burnett, R. T., Pope, C. A., Ito, K., Thurston, G., Krewski, D., Shi, Y., Calle, E. and Thun, M.: Long-Term Ozone Exposure and Mortality, N Engl J Med, 360(11), 1085–1095, doi:10.1056/NEJMoa0803894, 2009.

Kirtman, B., Power, S.B., Adedoyin, J.A., Boer, G.J., Bojariu, R., Camilloni, I., Doblas-Reyes, F.J., Fiore, A.M., Kimoto, M., Meehl, G.A., Prather, M., Sarr, A., Schär, C., Sutton, R., van Oldenborgh, G.J., Vecchi, G. and Wang, H.J.: Near-term Climate Change: Projections and Predictability. In: Climate Change 2013: The Physical Science Basis (Chapter 11). Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change [Stocker, T.F., D. Qin, G.-K. Plattner, M. Tignor, S.K. Allen, J. Boschung, A. Nauels, Y. Xia, V. Bex and P.M. Midgley (eds.)]. Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA, 2013.

Klimont, Z., Smith, S.J. and Cofala, J: The last decade of global anthropogenic sulfur dioxide: 2000-2011 emissions, Environ Res Lett 8(1), 014003, doi:10.1088/1748-9326/8/1/014003, 2013.

Knowlton, K., Rosenthal, J. E., Hogrefe, C., Lynn, B., Gaffin, S., Goldberg, R., Rosenzweig, C., Civerolo, K., Ku, J. Y. and Kinney, P. L.: Assessing ozone-related health impacts under a changing climate, Environ Health Perspect, 112(15), 1557–1563, doi:10.1289/ehp.7163, 2004.

Knowlton K., Hogrefe C., Lynn B., Rosenzweig C., Rosenthal J., Kinney P.L.: Impacts of heat and ozone on mortality risk in the New York City metropolitan region under a changing climate, Advances in Global Change Research, 30:143-160, 2008.

Krewski, D., Jerrett, M., Burnett, R. T., Ma, R., Hughes, E., Shi, Y., Turner, M. C., Pope III, C. A., Thurston, G., Calle, E. E. and Thun, M. J.: Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality, Respir Rep Heal Eff Inst, 140(140), 5–114, 2009.

Lamarque, J. F., Shindell, D. T., Josse, B., Young, P. J., Cionni, I., Eyring, V., Bergmann, D., Cameron-Smith, P., Collins, W. J., Doherty, R., Dalsoren, S., Faluvegi, G., Folberth, G., Ghan, S. J., Horowitz, L. W., Lee, Y. H., MacKenzie, I. A., Nagashima, T., Naik, V., Plummer, D., Righi, M., Rumbold, S. T., Schulz, M., Skeie, R. B., Stevenson, D. S., Strode, S., Sudo, K., Szopa, S., Voulgarakis, A. and Zeng, G.: The atmospheric chemistry and climate model intercomparison Project (ACCMIP): Overview and description of models, simulations and climate diagnostics, Geosci Model Dev, 6(1), 179–206, doi:10.5194/gmd-6-179-2013, 2013.

Lelieveld, J., Barlas, C., Giannadaki, D. and Pozzer, A.: Model calculated global, regional and megacity premature mortality due to air pollution, Atmos Chem Phys, 13(14), 7023–7037, doi:10.5194/acp-13-7023-2013, 2013.

Lelieveld, J., Evans, J. S., Fnais, M., Giannadaki, D. and Pozzer, a.: The contribution of outdoor air pollution sources to premature mortality on a global scale, Nature, 525(7569), 367–371, doi:10.1038/nature15371, 2015.

Lepeule, J., Laden, F., Dockery, D. and Schwartz, J.: Chronic exposure to fine particles and mortality: An extended follow-up of the Harvard six cities study from 1974 to 2009, Environ Health Perspect, 120(7), 965–970, doi:10.1289/ehp.1104660, 2012

Levy, J. I., Chemerynski, S. M. and Sarnat, J. A: Ozone exposure and mortality: an empiric bayes metaregression analysis, Epidemiology, 16(4), 458–468, doi:10.1097/01.ede.0000165820.08301.b3, 2005.

Likhvar, V. N., Pascal, M., Markakis, K., Colette, A., Hauglustaine, D., Valari, M., Klimont, Z., Medina, S. and Kinney, P.: A multi-scale health impact assessment of air pollution over the 21st century, Sci Total Environ, 514, 439–449, doi:10.1016/j.scitotenv.2015.02.002, 2015.

~~Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya, K., Adair-Rohani, H., Amann, M., Anderson, H. R., Andrews, K. G., Aryee, M., Atkinson, C., Bacchus, L. J., Bahalim, A. N., Balakrishnan, K., Balmes, J., Barker-Collo, S., Baxter, A., Bell, M. L., Blore, J. D., Blyth, F., Bonner, C., Borges, G., Bourne, R., Boussinesq, M., Brauer, M., Brooks, P., Bruce, N. G., Brunekreef, B., Bryan-Hancock, C., Bucello, C., Buchbinder, R., Bull, F., Burnett, R. T., Byers, T. E., Calabria, B., Carapetis, J., Carnahan, E., Chafe, Z., Charlson, F., Chen, H., Chen, J. S., Cheng, A. T. A., Child, J. C., Cohen, A., Colson, K. E., Cowie, B. C., Darby, S., Darling, S., Davis, A., Degenhardt, L., Dentener, F., Des Jarlais, D. C., Devries, K., Dherani, M., Ding, E. L., Dorsey, E. R., Driscoll, T., Edmond, K., Ali, S. E., Engell, R. E., Erwin, P. J., Fahimi, S., Falder, G., Farzadfar, F., Ferrari, A., Finucane, M. M., Flaxman, S., Fowkes, F. G. R., Freedman, G., Freeman, M. K., Gakidou, E., Ghosh, S., Giovannucci, E., Gmel, G., Graham, K., Grainger, R., Grant, B., Gunnell, D., Gutierrez, H. R., Hall, W., Hoek, H. W., Hogan, A., Hosgood, H. D., Hoy, D., Hu, H., Hubbell, B. J., Hutchings, S. J., Ibeanusi, S. E., Jacklyn, G. L., Jasrasaria, R., Jonas, J. B., Kan, H., Kanis, J. a., Kassebaum, N., Kawakami, N., Khang, Y. H., Khatibzadeh, S., Khoo, J. P., Kok, C., et al.: A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-~~

~~2010: A systematic analysis for the Global Burden of Disease Study 2010, Lancet, 380, 2224-2260, doi:10.1016/S0140-6736(12)61766-8, 2012.~~

Lozano, Rafael et al.: Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010, The Lancet , 380 (9859): 2095 – 2128, 2012.

Masui, T., Matsumoto, K., Hijioka, Y., Kinoshita, T., Nozawa, T., Ishiwatari, S., Kato, E., Shukla, P. R., Yamagata, Y. and Kainuma, M.: An emission pathway for stabilization at 6 Wm-2 radiative forcing, Clim Change, 109(1), 59–76, doi:10.1007/s10584-011-0150-5, 2011.

Orru, H., Andersson, C., Ebi, K. L., Langner, J., Åström, C. and Forsberg, B.: Impact of climate change on ozone-related mortality and morbidity in Europe, Eur Respir J, 41(2), 285–294, doi:10.1183/09031936.00210411, 2013.

Pattenden, S., Armstrong, B., Milojevic, A., Barratt, B., Chalabi, Z., Doherty, R. M., Heal, M. R., Kovats, S., Wilkinson, P.: Ozone, heat and mortality in fifteen British conurbations, Occup. Environ. Med., 67, 699-707, doi: 10.1136/oem.2009.051714, 2010.

Post, E. S., Grambsch, A., Weaver, C., Morefield, P., Huang, J., Leung, L. Y., Nolte, C. G., Adams, P., Liang, X. Z., Zhu, J. H. and Mahoney, H.: Variation in estimated ozone-related health impacts of climate change due to modeling choices and assumptions, Environ Health Perspect, 120(11), 1559–1564, doi:10.1289/ehp.1104271, 2012.

Rao, S., Chirkov, V., Dentener, F., Dingenen, R., Pachauri, S., Purohit, P., Amann, M., Heyes, C., Kinney, P., Kolp, P., Klimont, Z., Riahi, K. and Schoepp, W.: Environmental Modeling and Methods for Estimation of the Global Health Impacts of Air Pollution, Environ Model Assess, 613–622, doi:10.1007/s10666-012-9317-3, 2012.

Riahi, K., Rao, S., Krey, V., Cho, C., Chirkov, V., Fischer, G., Kindermann, G., Nakicenovic, N. and Rafaj, P.: RCP 8.5-A scenario of comparatively high greenhouse gas emissions, Clim Change, 109(1), 33–57, doi:10.1007/s10584-011-0149-y, 2011.

Roberts, S.: Interactions between particulate air pollution and temperature in air pollution mortality time series studies, Environ Res, 96(3), 328–337, doi:10.1016/j.envres.2004.01.015, 2004.

von Schneidemesser, E., Monks, P. S., Allan, J. D., Bruhwiler, L., Forster, P., Fowler, D., Lauer, A., Morgan, W. T., Paasonen, P., Righi, M., Sindelarova, K. and Sutton, M. A.: Chemistry and the Linkages between Air Quality and Climate Change, Chem Rev, (1), 150430065937004, doi:10.1021/acs.chemrev.5b00089, 2015.

Selin, N. E., Wu, S., Nam, K. M., Reilly, J. M., Paltsev, S., Prinn, R. G. and Webster, M. D.: Global health and economic impacts of future ozone pollution, Environ Res Lett, 4(4), 044014, doi:10.1088/1748-9326/4/4/044014, 2009.

Sheffield, P. E., Knowlton, K., Carr, J. L. and Kinney, P. L.: Modeling of Regional Climate Change Effects on Ground-Level Ozone and Childhood Asthma, Am J Prev Med, 41(3), 251–257, doi:10.1016/j.amepre.2011.04.017., 2011.

Shindell, D. T., Lamarque, J. F., Schulz, M., Flanner, M., Jiao, C., Chin, M., Young, P. J., Lee, Y. H., Rotstayn, L., Mahowald, N., Milly, G., Faluvegi, G., Balkanski, Y., Collins, W. J., Conley, a. J., Dalsoren, S., Easter, R., Ghan, S., Horowitz, L., Liu, X., Myhre, G., Nagashima, T., Naik, V., Rumbold, S. T., Skeie, R., Sudo, K., Szopa, S., Takemura, T., Voulgarakis, a., Yoon, J. H. and Lo, F.: Radiative forcing in the ACCMIP historical and future climate simulations, Atmos Chem Phys, 13(6), 2939–2974, doi:10.5194/acp-13-2939-2013, 2013.

Silva, R. A., West, J. J., Zhang, Y., Anenberg, S. C., Lamarque, J.-F., Shindell, D. T., Collins, W. J., Dalsoren, S., Faluvegi, G., Folberth, G., Horowitz, L. W., Nagashima, T., Naik, V., Rumbold, S., Skeie, R., Sudo, K., Takemura, T., Bergmann, D., Cameron-Smith, P., Cionni, I., Doherty, R. M., Eyring, V., Josse, B., MacKenzie, I. A., Plummer, D., Righi, M., Stevenson, D. S., Strode, S., Szopa, S. and Zeng, G.: Global premature mortality due to anthropogenic outdoor air pollution and the contribution of past climate change, Environ Res Lett, 8, 034005, doi:10.1088/1748-9326/8/3/034005, 2013.

Smith, S. J., West, J. J., and Kyle, P.: Economically consistent long-term scenarios for air pollutant and greenhouse gas emissions, Clim. Change, 108: 619-627, 2011.

Stevenson, D. S., Young, P. J., Naik, V., Lamarque, J. F., Shindell, D. T., Voulgarakis, A., Skeie, R. B., Dalsoren, S. B., Myhre, G., Berntsen, T. K., Folberth, G. A., Rumbold, S. T., Collins, W. J., MacKenzie, I. A., Doherty, R. M., Zeng, G., Van Noije, T. P. C., Strunk, A., Bergmann, D., Cameron-Smith, P., Plummer, D. A., Strode, S. A., Horowitz, L., Lee, Y. H., Szopa, S., Sudo, K., Nagashima, T., Josse, B., Cionni, I., Righi, M., Eyring, V., Conley, A., Bowman, K. W., Wild, O. and Archibald, A.: Tropospheric ozone changes, radiative forcing and attribution to emissions in the Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP), Atmos Chem Phys, 13(October 2012), 3063–3085, doi:10.5194/acp-13-3063-2013, 2013.

Tagaris, E., Liao, K.-J., Delucia, A. J., Deck, L., Amar, P. and Russell, A. G.: Potential impact of climate change on air pollution-related human health effects., Environ Sci Technol, 43(13), 4979–4988, doi:10.1021/es803650w, 2009.

Thomson, A. M., Calvin, K. V., Smith, S. J., Kyle, G. P., Volke, A., Patel, P., Delgado-Arias, S., Bond-Lamberty, B., Wise, M. A., Clarke, L. E. and Edmonds, J. A.: RCP4.5: A pathway for stabilization of radiative forcing by 2100, Clim Change, 109(1), 77–94, doi:10.1007/s10584-011-0151-4, 2011.

Van Vuuren, D. P., Edmonds, J., Kainuma, M., Riahi, K., Thomson, A., Hibbard, K., Hurtt, G. C., Kram, T., Krey, V., Lamarque, J. F., Masui, T., Meinshausen, M., Nakicenovic, N., Smith, S. J. and Rose, S. K.: The representative concentration pathways: An overview, Clim Change, 109, 5–31, doi:10.1007/s10584-011-0148-z, 2011a.

Van Vuuren, D. P., Stehfest, E., den Elzen, M. G. J., Kram, T., van Vliet, J., Deetman, S., Isaac, M., Goldewijk, K. K., Hof, A., Beltran, A. M., Oostenrijk, R. and van Ruijven, B.: RCP2.6: Exploring the possibility to keep global mean temperature increase below 2°C, Clim Change, 109(1), 95–116, doi:10.1007/s10584-011-0152-3, 2011b.

Weaver, C. P., Liang, X. Z., Zhu, J., Adams, P. J., Amar, P., Avise, J., Caughey, M., Chen, J., Cohen, R. C., Cooter, E., Dawson, J. P., Gilliam, R., Gilliland, A., Goldstein, A. H., Grambsch, A., Grano, D., Guenther, A., Gustafson, W. I., Harley, R. A., He, S., Hemming, B., Hogrefe, C., Huang, H. C., Hunt, S. W., Jacob, D. J., Kinney, P. L., Kunkel, K., Lamarque, J. F., Lamb, B., Larkin, N. K., Leung, L. R., Liao, K. J., Lin, J. T., Lynn, B. H.,

Manomaiphiboon, K., Mass, C., Mckenzie, D., Mickley, L. J., O'Neill, S. M., Nolte, C., Pandis, S. N., Racherla, P. N., Rosenzweig, C., Russell, A. G., Salathé, E., Steiner, A. L., Tagaris, E., Tao, Z., Tonse, S., Wiedinmyer, C., Williams, A., Winner, D. A., Woo, J. H., Wu, S. and Wuebbles, D. J.: A preliminary synthesis of modeled climate change impacts on U.S. regional ozone concentrations, Bull Am Meteorol Soc, 90(12), 1843–1863, doi:10.1175/2009BAMS2568.1, 2009.

West, J. J., Szopa, S. and Hauglustaine, D. A.: Human mortality effects of future concentrations of tropospheric ozone, C. R. Geosci., 339, 775-83, 2007.

West, J. J., Smith, S. J., Silva, R. A., Naik, V., Zhang, Y., Adelman, Z., Fry, M. M., Anenberg, S., Horowitz, L. W. and Lamarque, J.-F.: Co-benefits of global greenhouse gas mitigation for future air quality and human health, Nat Clim Chang, 3(10), 885–889, doi:10.1038/NCLIMATE2009, 2013.

Wilson, A., Rappold, A. G., Neas, L. M. and Reich, B. J.: Modeling the effect of temperature on ozone-related mortality, Ann Appl Stat, 8(3), 1728–1749, doi:10.1214/14-AOAS754, 2014.

Young, P. J., Archibald, A. T., Bowman, K. W., Lamarque, J.-F., Naik, V., Stevenson, D. S., Tilmes, S., Voulgarakis, A., Wild, O., Bergmann, D., Cameron-Smith, P., Cionni, I., Collins, W. J., Dalsøren, S. B., Doherty, R. M., Eyring, V., Faluvegi, G., Horowitz, L. W., Josse, B., Lee, Y. H., MacKenzie, I. A., Nagashima, T., Plummer, D. A., Righi, M., Rumbold, S. T., Skeie, R. B., Shindell, D. T., Strode, S. A., Sudo, K., Szopa, S. and Zeng, G.: Pre-industrial to end 21st century projections of tropospheric ozone from the Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP), Atmos Chem Phys, 13(10), 2063–2090, doi:10.5194/acp-13-2063-2013, 2013.

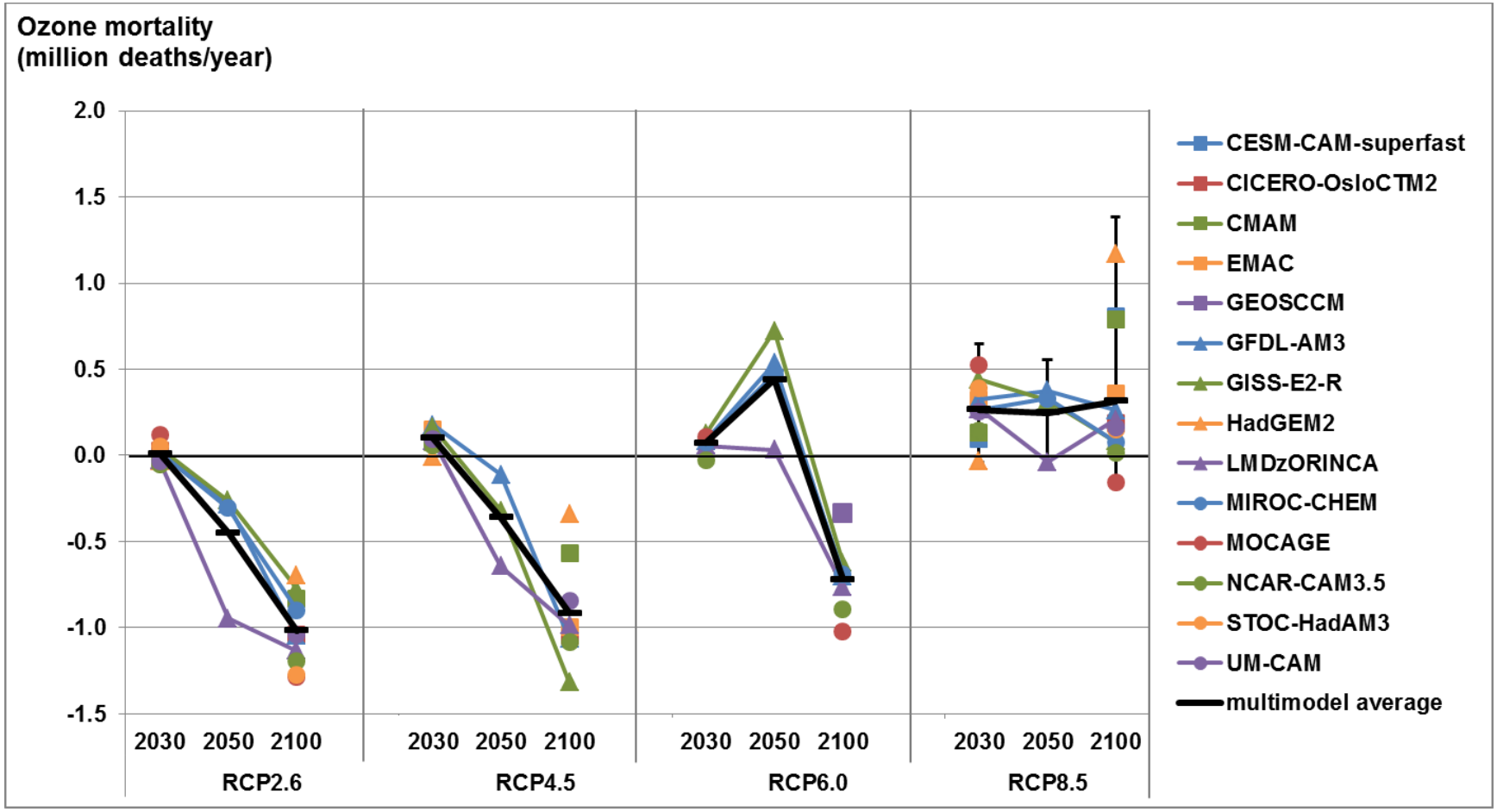


**Figure 1: Estimates of future ozone respiratory mortality for all RCP scenarios in 2030, 2050 and 2100, showing global mortality for 13 models and the multi-model average (million deaths/year), for future air pollutant concentrations relative to 2000 concentrations. Uncertainty for the multi-model average shown for RCP8.5 is the 95% CI including uncertainty in RR and across models. Only models with results for the three years have lines connecting the markers.**

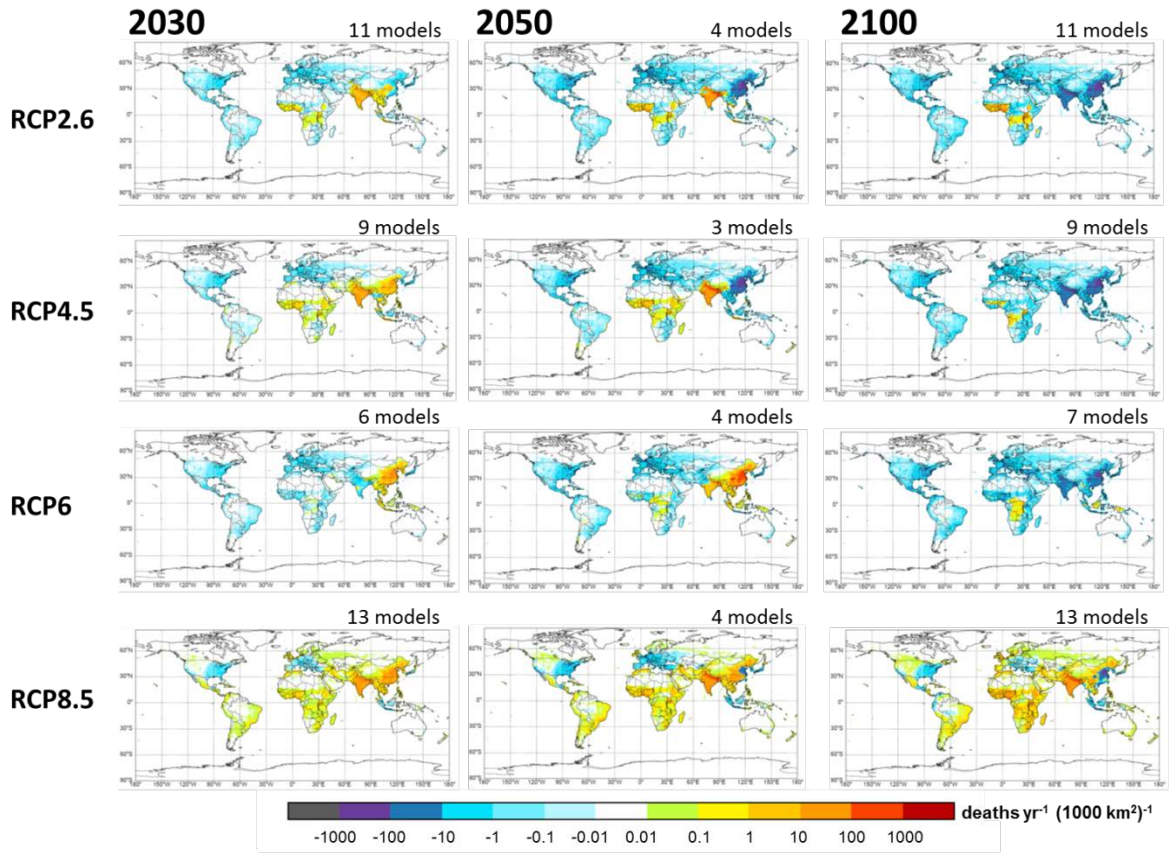


**Figure 2: Future ozone respiratory mortality for all RCP scenarios in 2030, 2050 and 2100, showing the multi-model average in each grid cell, for future air pollutant concentrations relative to 2000 concentrations.**

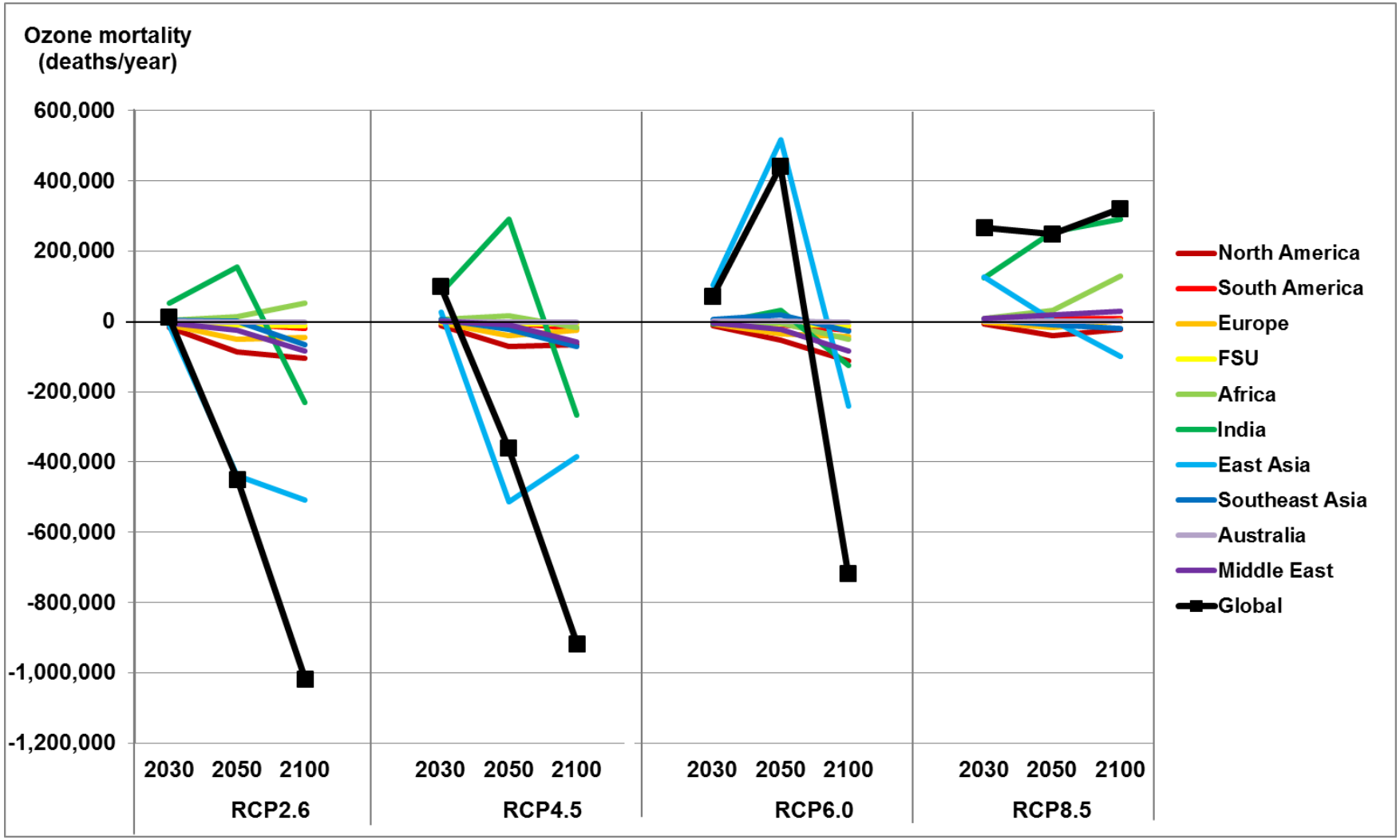


**Figure 3: Future ozone respiratory mortality for all RCP scenarios in 2030, 2050 and 2100, showing the multi-model regional average (deaths/year) in ten world regions (Figure S1) and globally, for future air pollutant concentrations relative to 2000 concentrations.**

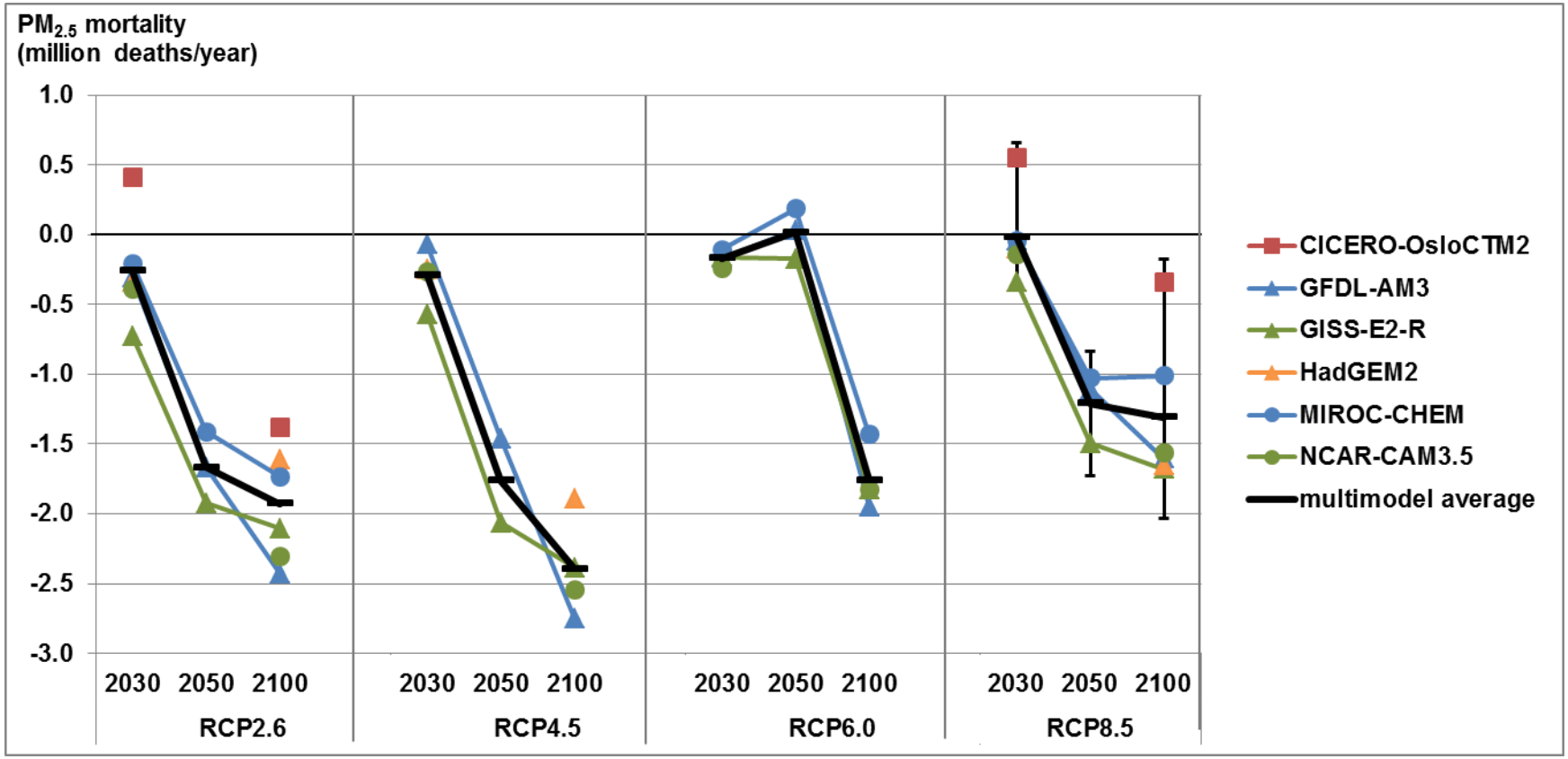


**Figure 4: Estimates of future premature mortality (IHD+STROKE+COPD+LC) for $PM_{2.5}$ calculated as a sum of species, for all RCP scenarios in 2030, 2050 and 2100, showing global mortality for six models and the multi-model average (million deaths/year), for future air pollutant concentrations relative to 2000 concentrations. Uncertainty shown for the RCP8.5 multi-model average is the 95% CI including uncertainty in RR and across models.**

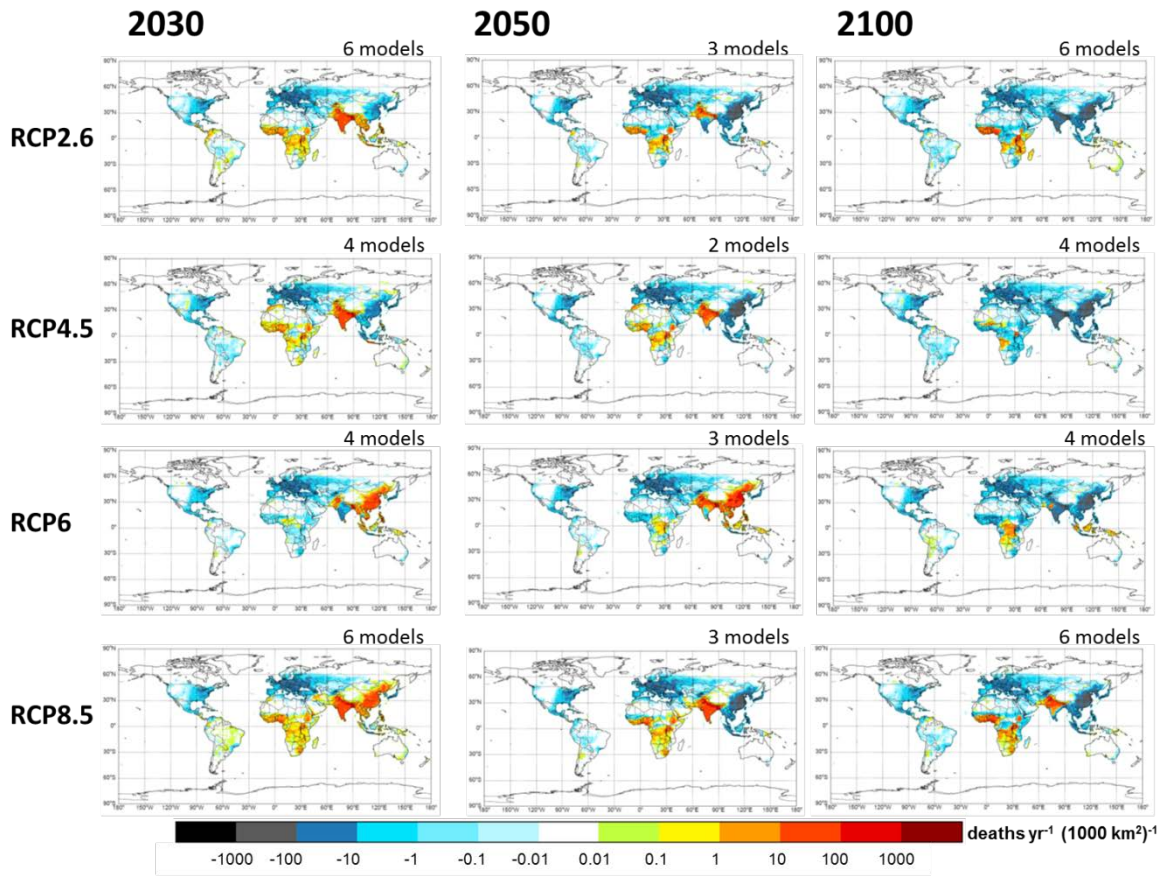


**Figure 5: Future premature mortality (IHD+STROKE+COPD+LC) for $PM_{2.5}$ calculated as a sum of species, for all RCP scenarios in 2030, 2050 and 2100, showing the multi-model average in each grid cell, for future air pollutant concentrations relative to 2000 concentrations.**

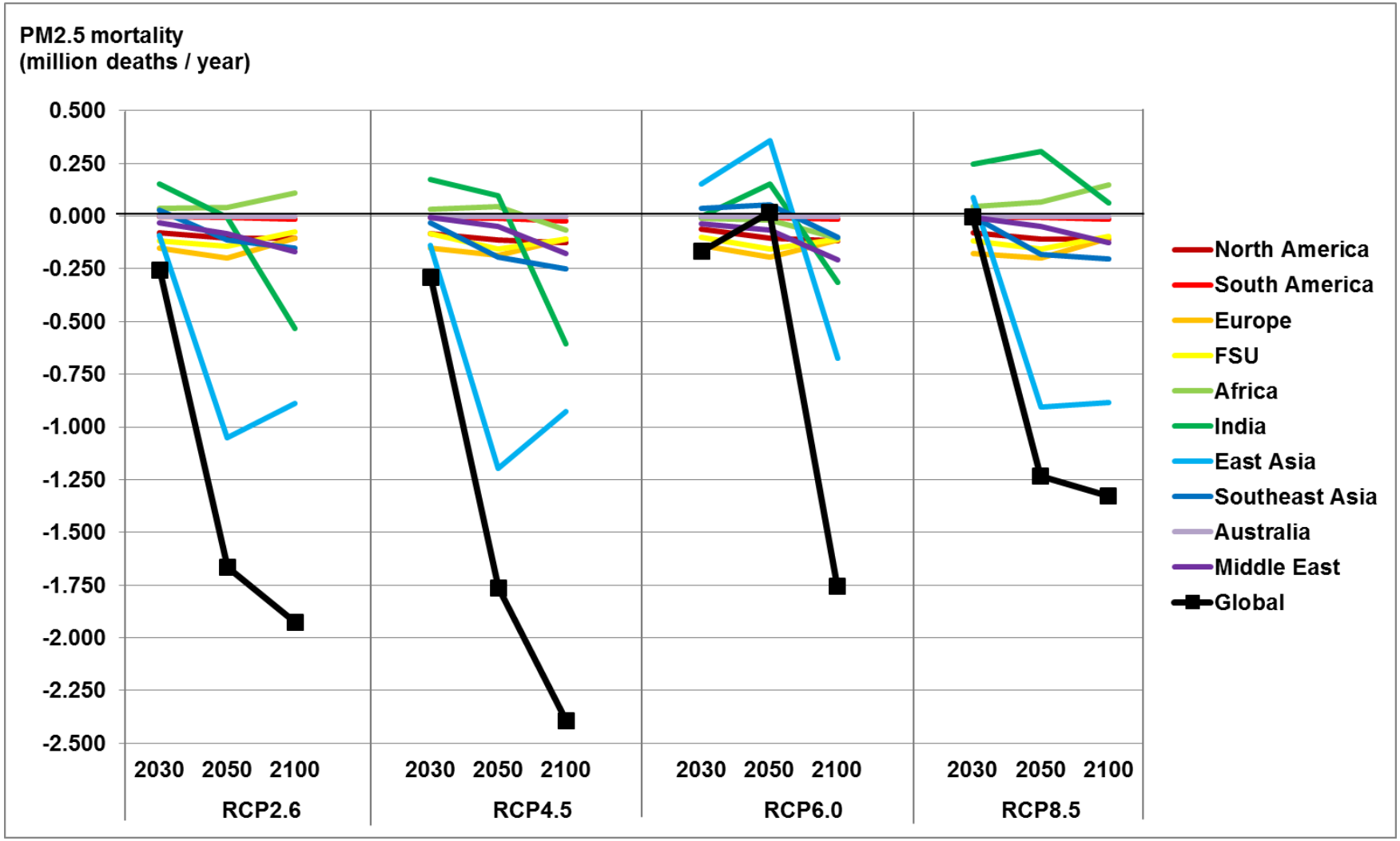


**Figure 6: Future premature mortality (IHD+STROKE+COPD+LC) for $PM_{2.5}$ calculated as a sum of species, for all RCP scenarios in 2030, 2050 and 2100, showing the multi-model regional average (deaths/year) in ten world regions (Figure S1) and globally, for future air pollutant concentrations relative to 2000 concentrations.**

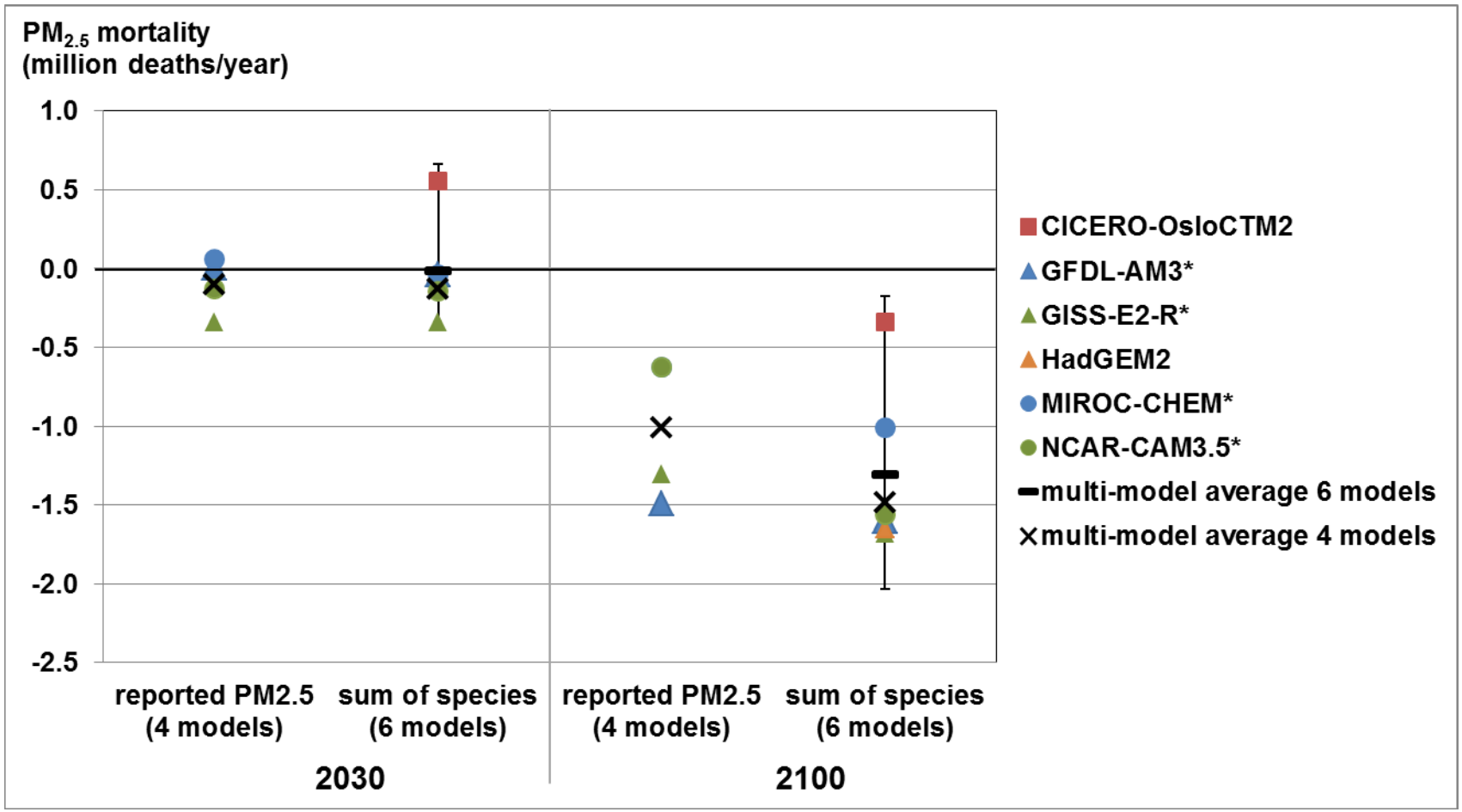


**Figure 7: Estimates of global future premature mortality (IHD+STROKE+COPD+LC) for RCP8.5 in 2030 and 2100, for $PM_{2.5}$ reported by four models and $PM_{2.5}$ estimated as a sum of species for six models, showing global mortality for each model and the multi-model average (million deaths/year), for future air pollutant concentrations relative to 2000 concentrations. Models signaled with * reported their own estimate of $PM_{2.5}$. Uncertainty shown for six models for sum of species is the 95% CI including uncertainty in RR and across models.**

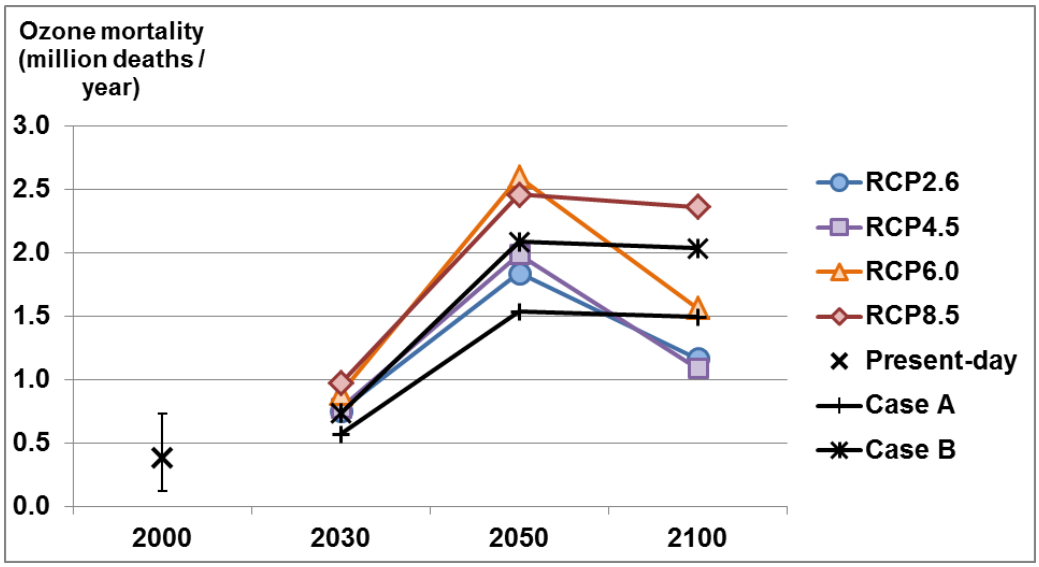


**Figure 8: Global burden on mortality of ozone concentrations relative to 1850, in the present day for 2000 concentrations, showing multi-model average and 95% CI including uncertainty in RR and across models (deaths/year), and in 2030, 2050 and 2100 for all RCPs, showing multi-model averages (deaths/year) given by the deterministic values. Also shown are future burdens using (Case A) 2000 concentrations relative to 1850 and present-day population but future baseline mortality rates and (Case B) 2000 concentrations relative to 1850 but future population and baseline mortality rates.**

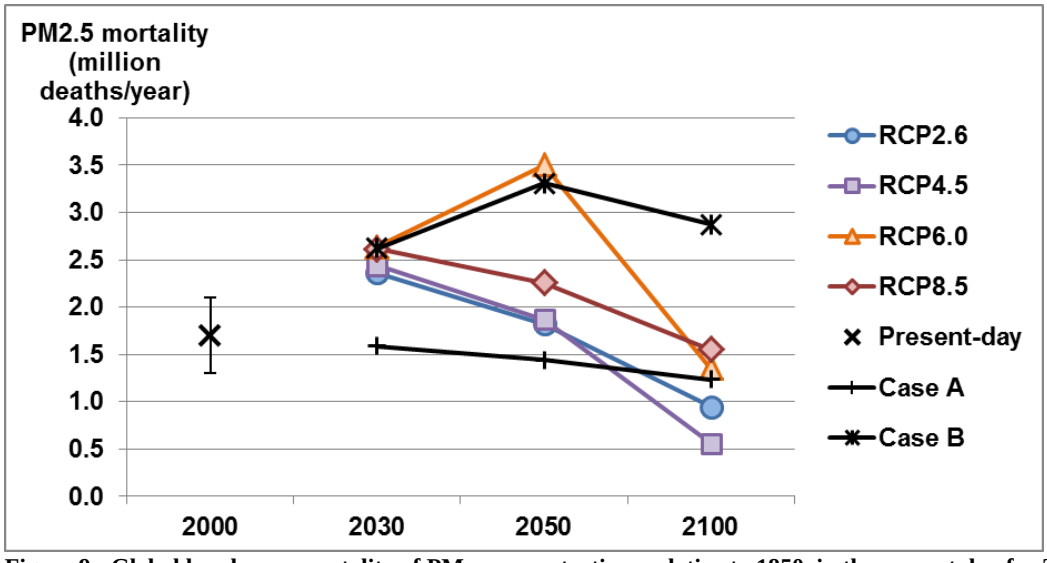


**Figure 9: Global burden on mortality of $PM_{2.5}$ concentrations relative to 1850, in the present day for 2000 concentrations, showing multi-model average and 95% CI including uncertainty in RR and across models (deaths/year), and in 2030, 2050 and 2100 for all RCPs, showing multi-model averages (deaths/year) given by the deterministic values. Also shown are future burdens using (Case A) 2000 concentrations relative to 1850 and present-day population but future baseline mortality rates and (Case B) 2000 concentrations relative to 1850 but future population and baseline mortality rates.**

# The effect of future ambient air pollution on human mortality to 2100 using output from the ACCMIP model ensemble – SUPPLEMENTAL MATERIAL

Raquel A. Silva, J. Jason West, Jean-François Lamarque, Drew T. Shindell, William J. Collins, Stig Dalsoren, Greg Faluvegi, Gerd Folberth, Larry W. Horowitz, Tatsuya Nagashima, Vaishali Naik, Steven T. Rumbold, Kengo Sudo, Toshihiko Takemura, Daniel Bergmann, Philip Cameron-Smith, Irene Cionni, Ruth M. Doherty, Veronika Eyring, Beatrice Josse, I. A. MacKenzie, David Plummer, Mattia Righi, David S. Stevenson, Sarah Strode, Sophie Szopa and Guang Zeng

## 1 Air pollutant ambient concentrations

The Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP) included contributions from several modeling groups. While up to 14 models reported ozone concentrations (depending on the scenario), only up to 6 models reported species used in the calculation of $PM_{2.5}$ concentrations, and up to 4 models reported their own estimate of $PM_{2.5}$ concentrations (Table S1).

Figure S1 shows ten world regions used for all regional calculations presented below.

Global population-weighted differences (Future year – 2000) in ozone and $PM_{2.5}$ concentrations for the different models are shown in Tables S2 and S3, respectively, while regional multi-model average differences are shown in Figures S2 and S3. For the global burden calculations, we use 1850 air pollutant concentrations reported by each model as counterfactual; for reference, we show the multi-model average concentrations in each grid cell (Figures S4 and S5).

For both pollutants, metrics are consistent with the underlying epidemiological studies for the health impact assessment:

- Seasonal (6-month) average of daily 1-hr maximum ozone concentration;
- Annual average $PM_{2.5}$ concentration.

$PM_{2.5}$ concentration is estimated using the sum of $PM_{2.5}$ species mass mixing ratios reported by six models:

Estimated PM2.5=BC + OA + SO4 + SOA + NO3 + NH4 + 0.25*SS + 0.1*Dust,

where BC – Black Carbon, Dust, OA – (Primary) Organic Aerosol corrected to include species other than carbon, SO4 - Sulfate, SOA – Secondary Organic Aerosol, and SS – Sea Salt, following Fiore et al. (2012) and Silva et al. (2013). The factors 0.25 and 0.1 are intended to indicate the fractions of sea salt and dust that are in the $PM_{2.5}$ size fraction.

## 2 ~~Future~~ Population and Baseline Mortality Rates

Table S4 includes present-day estimates of baseline mortality rates for cardiovascular diseases, chronic respiratory diseases and neoplasms given by IF projections for 2010 and GBD 2010.

Figure ~~S4~~ S6 shows future total and exposed population in 2030, 2050 and 2100 estimated from International Futures (IFs) country-level population per age group, used in the health impact assessment, as well as United Nations (UN) and Representative Concentration Pathway scenarios (RCPs) totals as context.

Figure ~~S5~~ S7 shows baseline mortality rates for chronic Respiratory diseases (RESP, ICD-9[1] BTL: B347), ischemic heart disease (IHD, ICD-9: 410-414), cerebrovascular disease (STROKE, ICD-9: 430-435, 437.0-437.2, 437.5-437.8), chronic obstructive pulmonary disease (COPD, ICD-9: 490-492.8, 494, 496) and lung cancer (LC, ICD-9 BTL: B101) estimated from IFs country-level mortality rates of cardiovascular diseases, chronic respiratory diseases and malignant neoplasms. Here we show average values for the exposed population (adults age 25 and older), but we used age distributed values for IHD and STROKE in the premature mortality calculation to align with available relative risks of exposure for these diseases.

**3 Detailed results**

Table ~~S4~~ S5 shows the multi-model average global future ozone premature mortality, including uncertainty for RCP8.5, while Table ~~S5~~ S6 shows the multi-model average across ten world regions. Table ~~S6~~ S7 shows the multi-model average global $PM_{2.5}$ mortality (IHD+STROKE+COPD+LC), including uncertainty for RCP8.5, while Table ~~S7~~ S8 shows the multi-model average across ten world regions. The multi-model average corresponds to the average of estimates given by the available models for each scenario/period. Figures S8 and S9 show the coefficient of variation in each grid cell of future air pollution-related premature mortality for all RCP scenarios in 2030, 2050 and 2100. Figures S10 and S11 show future global and regional air pollution-related premature mortality per million people in 2030, 2050 and 2100, for all RCPs relative to 2000.

[1] ICD-9 - International Classification of Diseases, revision 9.

Tables ~~S8~~ S9 and ~~S9~~ S10 show the global burden on mortality of ozone and $PM_{2.5}$ concentrations in 2000 relative to 1850, using present-day population and baseline mortality rates, and in 2030, 2050 and 2100 for all RCPs relative to 1850, using future population and baseline mortality rates. Also shown are two alternative cases for global burden calculation, using: A) 2000 concentrations relative to 1850 and present-day population but future baseline mortality rates; B) 2000 concentrations relative to 1850 but future population and baseline mortality rates.

~~Figures S6 and S7 show future global and regional air pollution-related premature mortality per million people in 2030, 2050 and 2100, for all RCPs relative to 2000.~~

**References**

Cameron-Smith, P., Lamarque, J.-F., Connell, P., Chuang, C., and Vitt F.: Toward an Earth system model: atmospheric chemistry, coupling, and petascale computing, J. Phys.: Conf. Ser. 46, 343-350, doi:10.1088/1742-6596/46/1/048, 2006.

Collins, W. J., Bellouin, N., Doutriaux-Boucher, M., Gedney, N., Halloran, P., Hinton, T., Hughes, J., Jones, C. D., Joshi, M., Liddicoat, S., Martin, G., O'Connor, F., Rae, J., Senior, C., Sitch, S., Totterdell, I., Wiltshire, A. and Woodward, S.: Development and evaluation of an Earth-System model-HadGEM2, Geosci Model Dev, 4, 1051-75, 2011.

Donner, L. J., Wyman, B. L., Hemler, R. S., Horowitz, L. W., Ming, Y., Zhao, M., Golaz, J. C., Ginoux, P., Lin, S. J., Schwarzkopf, M. D., Austin, J., Alaka, G., Cooke, W. F., Delworth, T. L., Freidenreich, S. M., Gordon, C. T., Griffies, S. M., Held, I. M., Hurlin, W. J., Klein, S. A., Knutson, T. R., Langenhorst, A. R., Lee, H. C., Lin, Y. L., Magi, B. I., Malyshev, S. L., Milly, P. C. D., Naik, V., Nath, M. J., Pincus, R., Ploshay, J. J., Ramaswamy, V., Seman, C. J., Shevliakova, E., Sirutis, J. J., Stern, W. F., Stouffer, R. J., Wilson, R. J., Winton, M., Wittenberg, A. T. and Zeng, F. R.: The Dynamical Core, Physical Parameterizations, and Basic Simulation Characteristics of the Atmospheric Component AM3 of the GFDL Global Coupled Model CM3, J Climate, 24, 3484-519, 2011.

Fiore, A. M., Naik, V., Spracklen, D. V., Steiner, A., Unger, N., Prather, M., Bergmann, D., Cameron-Smith, P. J., Cionni, I., Collins, W. J., Dalsøren, S., Eyring, V., Folberth, G. a., Ginoux, P., Horowitz, L. W., Josse, B., Lamarque, J.-F., MacKenzie, I. a., Nagashima, T., O'Connor, F. M., Righi, M., Rumbold, S. T., Shindell, D. T., Skeie, R. B., Sudo, K., Szopa, S., Takemura, T. and Zeng, G.: Global air quality and climate, Chem Soc Rev, 41(19), 6663, doi:10.1039/c2cs35095e, 2012.

Institute for Health Metrics and Evaluation (IHME): Global Burden of Disease Study 2010 (GBD 2010) Results by Cause 1990-2010 - Country Level, Seattle, United States, 2013.

Jöckel, P., Tost, H., Pozzer, A., Bruhl, C., Buchholz, J., Ganzeveld, L., Hoor, P., Kerkweg, A., Lawrence, M. G., Sander, R., Steil, B., Stiller, G., Tanarhte, M., Taraborrelli, D., Van Aardenne, J. and Lelieveld., J: The atmospheric chemistry general circulation model ECHAM5/MESSy1: consistent simulation of ozone from the surface to the mesosphere, Atmos Chem Phys, 6, 5067-104, 2006.

Josse, B., Simon, P., and Peuch, V.-H.: Rn-222 global simulations with the multiscale CTM MOCAGE, Tellus, 56B, 339–356, 2004.

Koch D., Schmidt G. A. and Field C. V.: Sulfur, sea salt, and radionuclide aerosols in GISS ModelE, J Geophys Res, 111, D06206, 2006.

Lamarque J.-F., Kyle G. P., Meinshausen M., Riahi K., Smith S. J., van Vuuren D. P., Conley A. J. and Vitt F.: Global and regional evolution of short-lived radiatively-active gases and aerosols in the Representative Concentration Pathways, Clim Change, 109, 191-212, 2011.

Lamarque, J.-F., Shindell, D. T., Josse, B., Young, P. J., Cionni, I., Eyring, V., Bergmann, D., Cameron-Smith, P., Collins, W. J., Doherty, R., Dalsoren, S., Faluvegi, G., Folberth, G., Ghan, S. J., Horowitz, L. W., Lee, Y. H., MacKenzie, I. A., Nagashima, T., Naik, V., Plummer, D., Righi, M., Rumbold, S. T., Schulz, M., Skeie, R. B., Stevenson, D. S., Strode, S., Sudo, K., Szopa, S., Voulgarakis, A., and Zeng, G.: The Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP): overview and description of models, simulations and climate diagnostics, Geosci. Model Dev., 6, 179-206, doi:10.5194/gmd-6-179-2013, 2013.

Naik, V., Horowitz, L. W., Fiore, A. M., Ginoux, P., Mao, J., Aghedo, A. M. and Levy, H.: Impact of preindustrial to present-day changes in short-lived pollutant emissions on atmospheric composition and climate forcing, J Geophys Res Atmos, 118, 8086–8110, doi:10.1002/jgrd.50608, 2013.

Oman L. D., Ziemke J. R., Douglass A. R., Waugh D. W., Lang C., Rodriguez J. M. and Nielsen J. E.: The response of tropical tropospheric ozone to ENSO, Geophys Res Lett, 38, 2011.

Righi, M., Eyring, V., Gottschaldt, K.-D., Klinger, C., Frank, F., Jöckel, P. and Cionni, I.: Quantitative evaluation of ozone and selected climate parameters in a set of EMAC simulations, Geosci. Model Dev., 8, 733–768, doi:10.5194/gmd-8-733-2015, 2015.

Scinocca J. F., McFarlane N. A., Lazare M., Li J. and Plummer D.: Technical Note: The CCCma third generation AGCM and its extension into the middle atmosphere, Atmos Chem Phys, 8**,** 7055-74, 2008.

Shindell D. T., Pechony O., Voulgarakis A., Faluvegi G., Nazarenko L., Lamarque J-F, Bowman K., Milly G., Kovari B., Ruedy R., Schmidt, G. A.: Interactive ozone and methane chemistry in GISS-E2 historical and future climate simulations, Atmos Chem Phys, 13, 2653–89, 2013.

Silva, R. A., West, J. J., Zhang, Y., Anenberg, S. C., Lamarque, J.-F., Shindell, D. T., Collins, W. J., Dalsoren, S., Faluvegi, G., Folberth, G., Horowitz, L. W., Nagashima, T., Naik, V., Rumbold, S., Skeie, R., Sudo, K., Takemura, T., Bergmann, D., Cameron-Smith, P., Cionni, I., Doherty, R. M., Eyring, V., Josse, B., MacKenzie, I. A., Plummer, D., Righi, M., Stevenson, D. S., Strode, S., Szopa, S. and Zeng, G.: Global premature mortality due to anthropogenic outdoor air pollution and the contribution of past climate change, Environ Res Lett, 8, 034005, doi:10.1088/1748-9326/8/3/034005, 2013.

Skeie R. B., Berntsen T. K., Myhre G., Tanaka K., Kvalevag M. M. and Hoyle C. R.: Anthropogenic radiative forcing time series from pre-industrial times until 2010, Atmos Chem Phys, 11, 11827-57, 2011.

Stevenson D. S., Doherty R. M., Sanderson M. G., Collins W. J., Johnson C. E. and Derwent R. G.: Radiative forcing from aircraft NOx emissions: Mechanisms and seasonal dependence, J Geophys Res, 109, 2004.

Szopa S., Balkanski Y., Schulz M., Bekki S., Cugnet D., Fortems-Cheiney A., Turquety S., Cozic A., Deandreis C., Hauglustaine D., Idelkadi A., Lathiere J., Lefevre F., Marchand M., Vuolo R., Yan N. and Dufresne J.-L.: Aerosol and ozone changes as forcing for climate evolution between 1850 and 2100, Climate Dynamics, doi: 10.1007/s00382-012-1408-y, 2012.

Teyssedre H., Michou M., Clark H. L., Josse B., Karcher F., Olivie D., Peuch V. H., Saint-Martin D., Cariolle D., Attie J. L., Nedelec P., Ricaud P., Thouret V., Van Der A. R. J., Volz-Thomas A. and Cheroux F.: A new tropospheric and stratospheric Chemistry and Transport Model MOCAGE-Climat for multi-year studies: evaluation of the present-day climatology and sensitivity to surface processes, Atmos Chem Phys**, 7,** 5815-60, 2007.

Van Vuuren, D. P., Edmonds, J., Kainuma, M., Riahi, K., Thomson, A., Hibbard, K., Rose, S. K.: The representative concentration pathways: An overview, Clim Change, 109, 5–31, doi:10.1007/s10584-011-0148-z, 2011.

Watanabe S., Hajima T., Sudo K., Nagashima T., Takemura T., Okajima H., Nozawa T., Kawase H., Abe M., Yokohata T., Ise T., Sato H., Kato E., Takata K., Emori S. and Kawamiya M.: MIROC-ESM 2010: model description and basic results of CMIP5-20c3m experiments, Geosci Model Dev, 4**,** 845-72, 2011.

Zeng G., Pyle J. A. and Young P. J.: Impact of climate change on tropospheric ozone and its global budgets, Atmos Chem Phys**,** 8**,** 369-87, 2008.

Zeng G., Morgenstern O., Braesicke P. and Pyle J. A.: Impact of stratospheric ozone recovery on tropospheric ozone and its budget, Geophys Res Lett**,** 37, L09805, 2010.

**Tables and Figures**

Table S1 – Models that reported ozone, $PM_{2.5}$ species and $PM_{2.5}$ (mmrpm2p5) concentrations for ACCMIP, with type of ozone output (h – hourly, m – monthly) and number of reported $PM_{2.5}$ species.

| Model | Institution | Contact | Ozone | $PM_{2.5}$ | References |
|---|---|---|---|---|---|
| CESM-CAM-superfast | LLNL | Dan Bergmann, Philip Cameron-Smith | h | - | Lamarque et al., 2013; Cameron-Smith, et al., 2006 |
| CMAM | CCCMA, Environment Canada | David Plummer | h | - | Scinocca et al., 2008 |
| EMAC | DLR, Germany | Veronika Eyring, Irene Cionni, Mattia Righi | m | - | Jöckel et al., 2006 Righi et al., 2015 |
| GEOSCCM | NASA GSFC | Sarah Strode | h | - | Oman et al., 2011 |
| GFDL-AM3 | NOAA GFDL | Vaishali Naik, Larry Horowitz | h | 8, mmrpm2p5 | Donner et al., 2011; Naik et al., 2013 |
| GISS-E2-R | NASA-GISS | Drew T. Shindell Greg Faluvegi | h | 8, mmrpm2p5 | Koch et al., 2006; Shindell et al., 2013 |
| HadGEM2 | Hadley Centre Met Office, UK | William Collins, Gerd Folbert, Steven Rumbold | m | 6 | Collins et al., 2011 |
| LMDzORINCA | IPSL-LSCE, France | Sophie Szopa | m | - | Szopa et al., 2012 |
| MIROC-CHEM | NIES-JAMSTEC-NagoyaU-KyushuU, Japan | Tatsuya Nagashima, Kengo Sudo, Toshihiko Takemura | h | 6, mmrpm2p5 | Watanabe et al., 2011 |
| MOCAGE | MeteoFrance, France | Beatrice Josse | h | - | Josse et al., 2004; Teyssedre et al., 2007 |
| NCAR-CAM3.5 | NCAR | Jean-François Lamarque | h | 6, mmrpm2p5 | Lamarque et al., 2011, 2012 |
| OsloCTM2 | CICERO and Univ. Oslo, Norway | Stig Dalsoren, Ragnhild Skeie | m | 8 | Skeie et al., 2011 |
| STOC-HadAM3 | University of Edinburgh, UK | Ian MacKenzie, Ruth Doherty, David Stevenson | m | 3 (not used) | Stevenson et al., 2004 |
| UM-CAM | NIWA, New Zealand | Guang Zeng | h | - | Zeng et al., 2008, 2010 |

Table S2 – Global population-weighted differences (Future year – Hist. 2000) in ozone concentrations (ppb) for the 14 models in 2030, 2050, 2100 for the four RCPs. Pollutant concentrations are weighted by exposed population (adults aged 25 and older) in each future year. Models with the symbol * reported only monthly average ozone concentrations.

| Models | 2030 | | | | 2050 | | | | 2100 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
| **CESM-CAM-superfast([a])** | | | | 3.7 | | | | | | | | 9.7 |
| **CMAM** | -1.2 | 2.1 | | 4.4 | | | | | -9.0 | -6.1 | | 9.6 |
| **GEOSCCM** | | | | | | | | | | | -4.7 | |
| **GFDL-AM3** | 0.5 | 4.7 | 1.3 | 9.0 | -1.3 | 1.6 | 2.3 | 5.7 | -8.7 | -9.6 | -8.3 | 5.1 |
| **GISS-E2-R** | 0.2 | 4.2 | 1.5 | 12.0 | 0.1 | 0.2 | 2.9 | 5.6 | -5.1 | -11.7 | -7.6 | 2.9 |
| **MIROC-CHEM** | -0.5 | | 0.9 | 6.9 | -2.3 | | 1.6 | 4.1 | -8.1 | | -8.5 | 1.3 |
| **MOCAGE** | 2.7 | | 1.5 | 15.2 | | | | | -9.4 | | -11.9 | 1.5 |
| **NCAR-CAM3.5** | -2.5 | 0.9 | -2.1 | 3.7 | | | | | -11.7 | -11.2 | -11.4 | 0.9 |
| **UM-CAM** | -1.4 | 2.5 | | 7.1 | | | | | -8.9 | -7.3 | | 3.9 |
| **CICERO-OsloCTM2*** | 0.0 | 2.8 | | 8.2 | | | | | -9.3 | -9.5 | | 4.2 |
| **EMAC*** | | 4.0 | | 9.5 | | | | | | -8.9 | | 5.9 |
| **HadGEM2*** | -0.9 | -0.1 | | 0.5 | | | | | -7.7 | -3.7 | | 13.6 |
| **LMDzORINCA*** | -1.7 | 1.7 | 0.2 | 7.2 | -8.7 | -4.7 | -2.8 | 0.7 | -9.9 | -9.0 | -8.9 | 3.6 |
| **STOC-HadAM3*** | 0.7 | | | 11.0 | | | | | -10.3 | | | 3.5 |

([a]) CESM-CAM-superfast reported concentrations for RCP2.6 and RCP 6.0, but the simulations for these scenarios used an inconsistent SST file and are not a matched set with the other simulations, so they were not considered here.

Table S3 – Global population-weighted differences (Future year – Hist. 2000) in $PM_{2.5}$ concentrations (estimated as a sum of reported species) ($\mu g/m^3$) for the 6 models in 2030, 2050, 2100 for the four RCPs. Pollutant concentrations are weighted by exposed population (adults aged 25 and older) in each future year.

| Models | 2030 | | | | 2050 | | | | 2100 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
| **GFDL-AM3** | 0.1 | 1.1 | 0.03 | 1.9 | -3.4 | -2.3 | 1.4 | -1.0 | -5.6 | -6.3 | -4.8 | -3.3 |
| **GISS-E2-R** | -2.0 | -1.3 | 0.9 | 0.1 | -4.4 | -4.5 | 0.8 | -3.1 | -5.1 | -5.9 | -5.1 | -4.0 |
| **NCAR-CAM3.5** | -0.4 | 0.01 | -0.03 | 1.3 | | | | | -5.7 | -6.4 | -4.9 | -3.9 |
| **MIROC-CHEM** | 0.2 | | 0.5 | 1.0 | -2.7 | | 1.3 | -1.5 | -3.9 | | -3.3 | -2.1 |
| **CICERO-OsloCTM2** | 2.6 | | | 3.8 | | | | | -3.3 | | | -0.4 |
| **HadGEM2** | 0.5 | 0.9 | | 1.7 | | | | | -3.5 | -4.6 | | -3.9 |

Table S4 – IF projections for 2010 and GBD 2010 estimates of age-standardized mortality rates (deaths per 100,000 people).

| Diseases | IF | GBD 2010 |
|---|---|---|
| Cardiovascular | 234.9 | 234.8 |
| Chronic Respiratory | 58.4 | 57.0 |
| Neoplasms | 106.9 | 121.4 |

Table ~~S4~~ S5 – Change in ~~Global~~ global respiratory premature ozone mortality in 2030, 2050 and 2100 for all RCPs (considering the change in future ozone concentrations relative to 2000 concentrations), showing the multi-model average (deaths/year) for RCP2.6, RCP4.5 and RCP6.0 deterministic estimates and the empirical mean with 95% CI in parenthesis for RCP8.5 probabilistic estimates (including uncertainty in the RRs and across models). These results correspond to Figure 1. All numbers are rounded to three significant digits.

| | | |
|---|---|---|
| | **2030** | |
| **RCP2.6** | 11,900 | |
| **RCP4.5** | 100,000 | |
| **RCP6** | 71,200 | |
| **RCP8.5** | 264,000 | (-39,300 , 648,000) |
| | **2050** | |
| **RCP2.6** | -450,000 | |
| **RCP4.5** | -360,000 | |
| **RCP6** | 441,000 | |
| **RCP8.5** | 246,000 | (-59,600 , 556,000) |
| | **2100** | |
| **RCP2.6** | -1,020,000 | |
| **RCP4.5** | -917,000 | |
| **RCP6** | -718,000 | |
| **RCP8.5** | 316,000 | (-187,000 , 1,380,000) |

Table ~~S5~~ S6 – Premature ozone-related respiratory mortality in ten world regions relative to 2000 concentrations: (a) 2030, (b) 2050, (c) 2100, showing the multi-model average (deaths/year) of the deterministic results. All numbers are rounded to three significant digits.

**(a) 2030**

| Region | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
|---|---|---|---|---|
| **North America** | -17,000 | -12,500 | -10,900 | -8,200 |
| **South America** | -2,710 | -500 | -3,260 | 1,840 |
| **Europe** | -8,870 | -5,590 | -7,190 | -880 |
| **Former Soviet Union** | -2,200 | -1,030 | -1,600 | 660 |
| **Africa** | 2,100 | 6,440 | -3,520 | 9,020 |
| **India** | 52,900 | 82,000 | -6,440 | 124,000 |
| **East Asia** | -11,300 | 25,700 | 103,000 | 127,000 |
| **Southeast Asia** | 2,980 | 5,010 | 4,890 | 5,980 |
| **Australia** | -280 | -120 | -100 | 20 |
| **Middle East** | -3,630 | 930 | -3,700 | 7,460 |

**(b) 2050**

| Region | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
|---|---|---|---|---|
| **North America** | -85,500 | -70,200 | -52,100 | -41,100 |
| **South America** | -8,180 | -4,910 | -8,920 | 8,530 |
| **Europe** | -49,400 | -40,000 | -34,800 | -16,600 |
| **Former Soviet Union** | -9,760 | -6,390 | -5,710 | -440 |
| **Africa** | 13,100 | 16,600 | -5,520 | 30,500 |
| **India** | 154,000 | 290,000 | 32,200 | 256,000 |
| **East Asia** | -439,000 | -514,000 | 518,000 | 3,830 |
| **Southeast Asia** | 900 | -21,300 | 19,600 | -9,920 |
| **Australia** | -1,260 | -590 | -490 | 250 |
| **Middle East** | -24,800 | -9,930 | -21,100 | 18,300 |

**(c) 2100**

| Region | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
|---|---|---|---|---|
| **North America** | -104,000 | -66,300 | -111,000 | -21,100 |
| **South America** | -19,800 | -20,200 | -25,900 | 7,950 |
| **Europe** | -44,600 | -24,900 | -41,600 | 2,390 |
| **Former Soviet Union** | -12,500 | -8,180 | -11,100 | 1,290 |
| **Africa** | 51,100 | -16,000 | -49,400 | 128,000 |
| **India** | -230,000 | -267,000 | -125,000 | 292,000 |
| **East Asia** | -509,000 | -383,000 | -241,000 | -99,700 |
| **Southeast Asia** | -65,000 | -71,400 | -28,000 | -21,000 |
| **Australia** | -1,620 | -990 | -1,510 | 790 |
| **Middle East** | -83,400 | -58,100 | -83,200 | 29,800 |

Table ~~S6~~ S7 – Change in ~~Global~~ global premature $PM_{2.5}$ mortality (IHD+Stroke+COPD+LC) in 2030, 2050 and 2100 for all RCPs (considering the change in future $PM_{2.5}$ concentrations relative to 2000 concentrations), showing multi-model average (deaths/year) for RCP2.6, RCP4.5 and RCP6.0 deterministic estimates and the empirical mean with 95% CI in parenthesis for RCP8.5 probabilistic estimates (including uncertainty in the RRs and across models). These results correspond to Figure 4. All numbers are rounded to three significant digits.

| | | |
|---|---|---|
| | **2030** | |
| **RCP2.6** | -258,000 | |
| **RCP4.5** | -289,000 | |
| **RCP6** | -169,000 | |
| **RCP8.5** | 17,200 | (-386,000 , 661,000) |
| | **2050** | |
| **RCP2.6** | -1,670,000 | |
| **RCP4.5** | -1,760,000 | |
| **RCP6** | 16,700 | |
| **RCP8.5** | -1,210,000 | (-1,730,000 , -835,000) |
| | **2100** | |
| **RCP2.6** | -1,930,000 | |
| **RCP4.5** | -2,390,000 | |
| **RCP6** | -1,760,000 | |
| **RCP8.5** | -1,310,000 | (-2,040,000 , -174,000) |

Table ~~S7~~ S8 – Premature $PM_{2.5}$ mortality (IHD+Stroke+COPD+LC) in ten world regions: (a) 2030, (b) 2050, (c) 2100, showing the multi-model average (deaths/year) of the deterministic results. All numbers are rounded to three significant digits.

**(a) 2030**

| Region | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
|---|---|---|---|---|
| **North America** | -77,800 | -83,500 | -59,700 | -77,100 |
| **South America** | -570 | -6,100 | -6,960 | -6,290 |
| **Europe** | -153,000 | -152,000 | -137,000 | -176,000 |
| **Former Soviet Union** | -119,000 | -82,000 | -101,000 | -116,000 |
| **Africa** | 35,100 | 31,800 | -10,200 | 46,100 |
| **India** | 150,000 | 176,000 | -2,690 | 245,000 |
| **East Asia** | -90,100 | -137,800 | 151,000 | 86,000 |
| **Southeast Asia** | 27,800 | -30,700 | 36,200 | -430 |
| **Australia** | -560 | -180 | -440 | -30 |
| **Middle East** | -30,700 | -4,430 | -37,400 | -7,230 |

**(b) 2050**

| Region | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
|---|---|---|---|---|
| **North America** | -106,000 | -114,000 | -104,000 | -107,000 |
| **South America** | -7,550 | -9,550 | -6,720 | -7,940 |
| **Europe** | -198,000 | -187,000 | -193,000 | -200,000 |
| **Former Soviet Union** | -144,000 | -158,000 | -154,000 | -156,000 |
| **Africa** | 40,200 | 46,000 | -21,100 | 66,100 |
| **India** | -6,540 | 97,000 | 152,000 | 308,000 |
| **East Asia** | -1,050,000 | -1,200,000 | 356,000 | -906,000 |
| **Southeast Asia** | -113,000 | -193,000 | 52,500 | -182,000 |
| **Australia** | -370 | -390 | -250 | -240 |
| **Middle East** | -81,200 | -47,800 | -64,300 | -47,200 |

**(c) 2100**

| Region | RCP2.6 | RCP4.5 | RCP6.0 | RCP8.5 |
|---|---|---|---|---|
| **North America** | -105,000 | -128,000 | -116,000 | -110,000 |
| **South America** | -15,600 | -21,300 | -12,800 | -15,000 |
| **Europe** | -104,000 | -110,000 | -112,000 | -103,000 |
| **Former Soviet Union** | -75,200 | -109,000 | -111,000 | -97,500 |
| **Africa** | 111,000 | -68,100 | -107,000 | 147,000 |
| **India** | -531,000 | -606,000 | -315,000 | 62,700 |
| **East Asia** | -886,000 | -926,000 | -673,000 | -882,000 |
| **Southeast Asia** | -153,000 | -250,000 | -103,000 | -202,000 |
| **Australia** | 30 | -850 | -770 | -440 |
| **Middle East** | -168,000 | -176,000 | -209,000 | -127,000 |

Table ~~S8~~ S9 – Global burden on mortality of ozone concentrations in the present-day for 2000 concentrations relative to 1850, showing multi-model average and 95% CI including uncertainty in RR and across models (deaths/year), and in 2030, 2050 and 2100 for all RCPs relative to 1850, showing multi-model averages (deaths/year) given by the deterministic values. Also shown, future burdens using (Case A) 2000 concentrations relative to 1850 and present-day population but future baseline mortality rates and (Case B) 2000 concentrations relative to 1850 but future population and baseline mortality rates. These results are plotted in Figure 7. All numbers are rounded to three significant digits.

| | 2000 | 2030 | 2050 | 2100 |
|---|---|---|---|---|
| Present-day | 382,000 (121,000 to 728,400) | | | |
| RCP2.6 | | 756,000 | 1,840,000 | 1,170,000 |
| RCP4.5 | | 775,000 | 1,990,000 | 1,090,000 |
| RCP6.0 | | 891,000 | 2,600,000 | 1,570,000 |
| RCP8.5 | | 972,000 | 2,460,000 | 2,360,000 |
| Case A | | 569,000 | 1,540,000 | 1,490,000 |
| Case B | | 735,000 | 2,090,000 | 2,040,000 |

Table ~~S9~~ S10 – Global burden on mortality of $PM_{2.5}$ concentrations in the present-day for 2000 concentrations relative to 1850, showing multi-model average and 95% CI including uncertainty in RR and across models (deaths/year), and in 2030, 2050 and 2100 for all RCPs relative to 1850, showing multi-model averages (deaths/year) given by the deterministic values. Also shown, future burdens using (Case A) 2000 concentrations relative to 1850 and present-day population but future baseline mortality rates and (Case B) 2000 concentrations relative to 1850 but future population and baseline mortality rates. These results are plotted in Figure 8. All numbers are rounded to three significant digits.

| | 2000 | 2030 | 2050 | 2100 |
|---|---|---|---|---|
| Present-day | 1,700,000 (1,300,000 to 2,100,000) | | | |
| RCP2.6 | | 2,360,000 | 1,820,000 | 948,000 |
| RCP4.5 | | 2,440,000 | 1,870,000 | 559,000 |
| RCP6.0 | | 2,640,000 | 3,500,000 | 1,350,000 |
| RCP8.5 | | 2,620,000 | 2,250,000 | 1,550,000 |
| Case A | | 1,590,000 | 1,440,000 | 1,230,000 |
| Case B | | 2,620,000 | 3,310,000 | 2,880,000 |

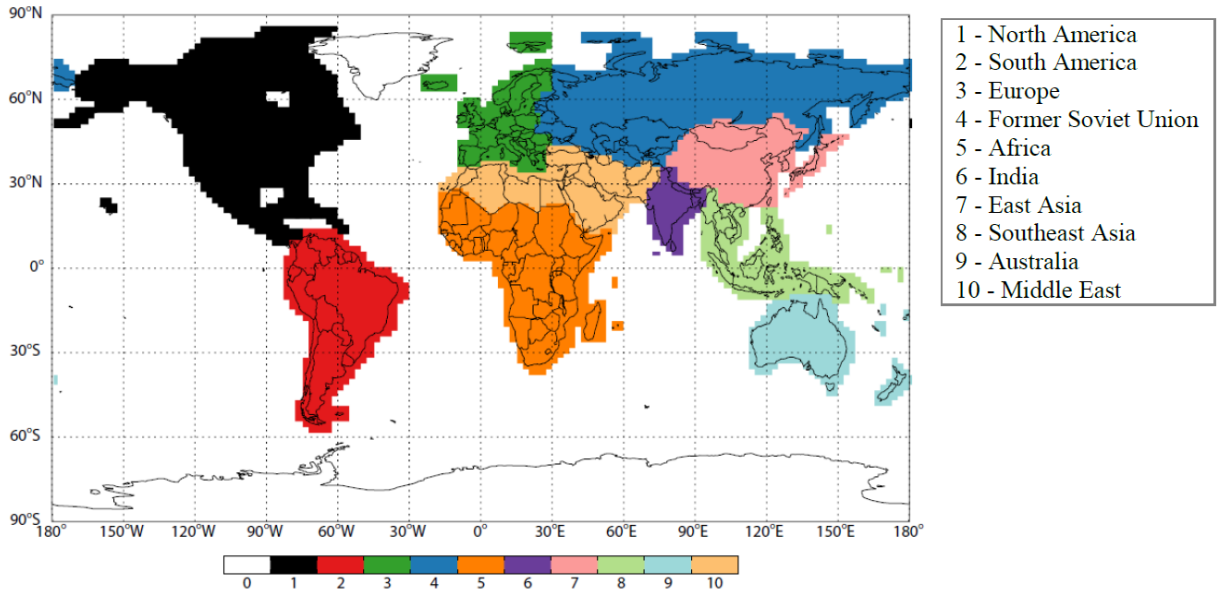


Figure S1 – Ten world regions

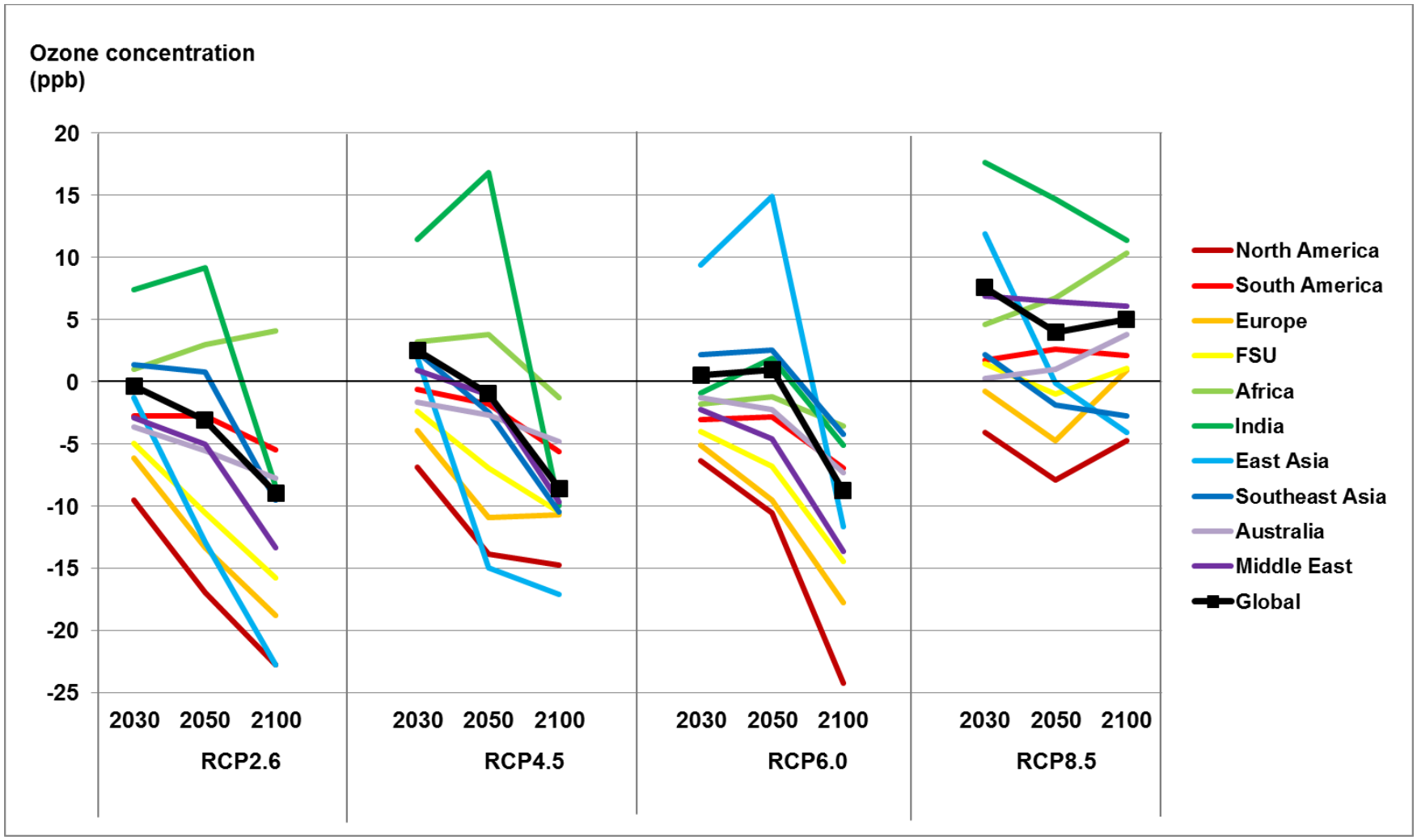


Figure S2 – Regional population-weighted difference in ozone concentrations (ppb) in 2030, 2050 and 2100 relative to 2000. (FSU – Former Soviet Union)

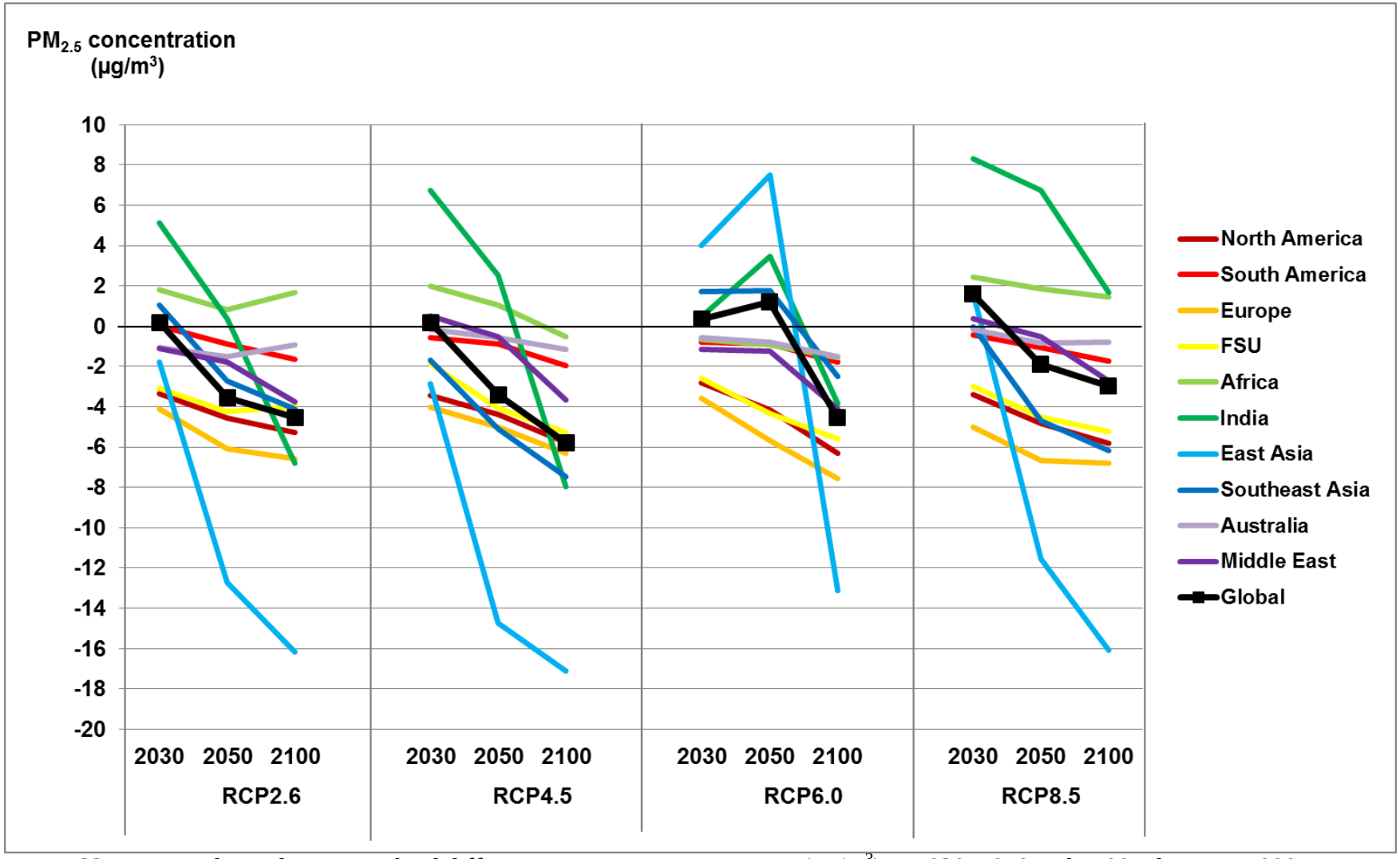


Figure S3 – Regional population-weighted difference in $PM_{2.5}$ concentrations ($\mu g/m^3$) in 2030, 2050 and 2100 relative to 2000.

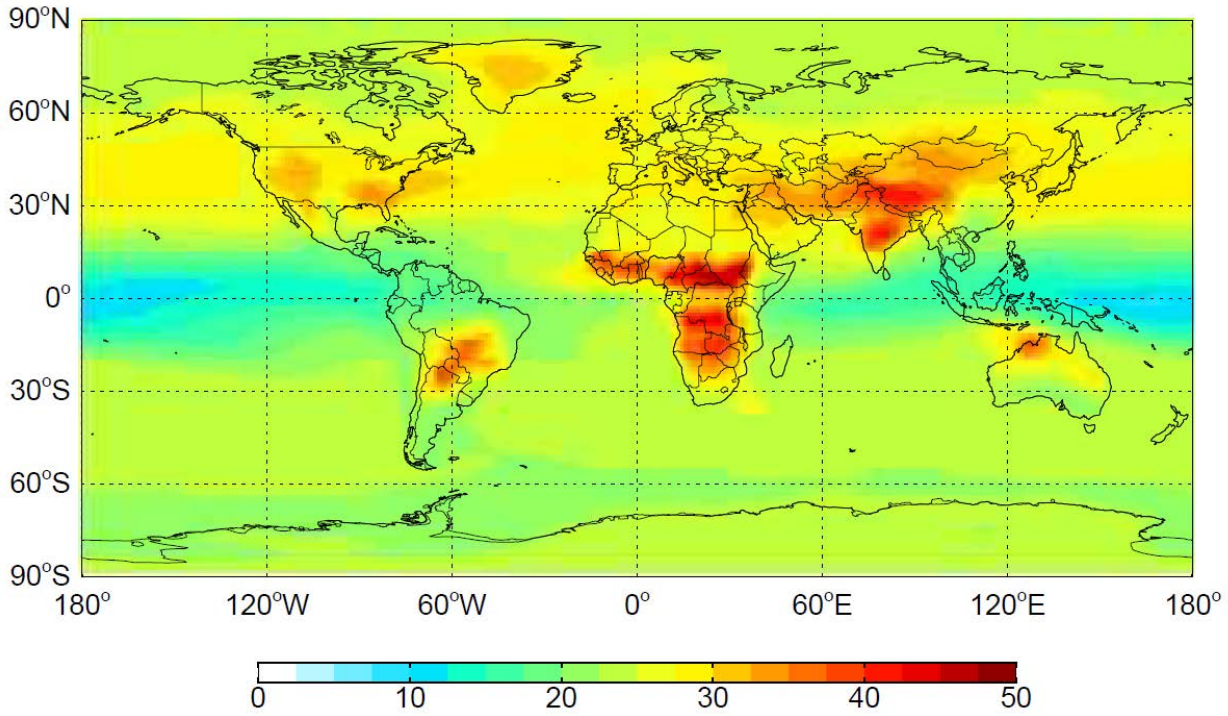


Figure S4 – Spatial distribution of ozone concentrations in 1850 (ppb), showing the multi-model mean in each grid cell.

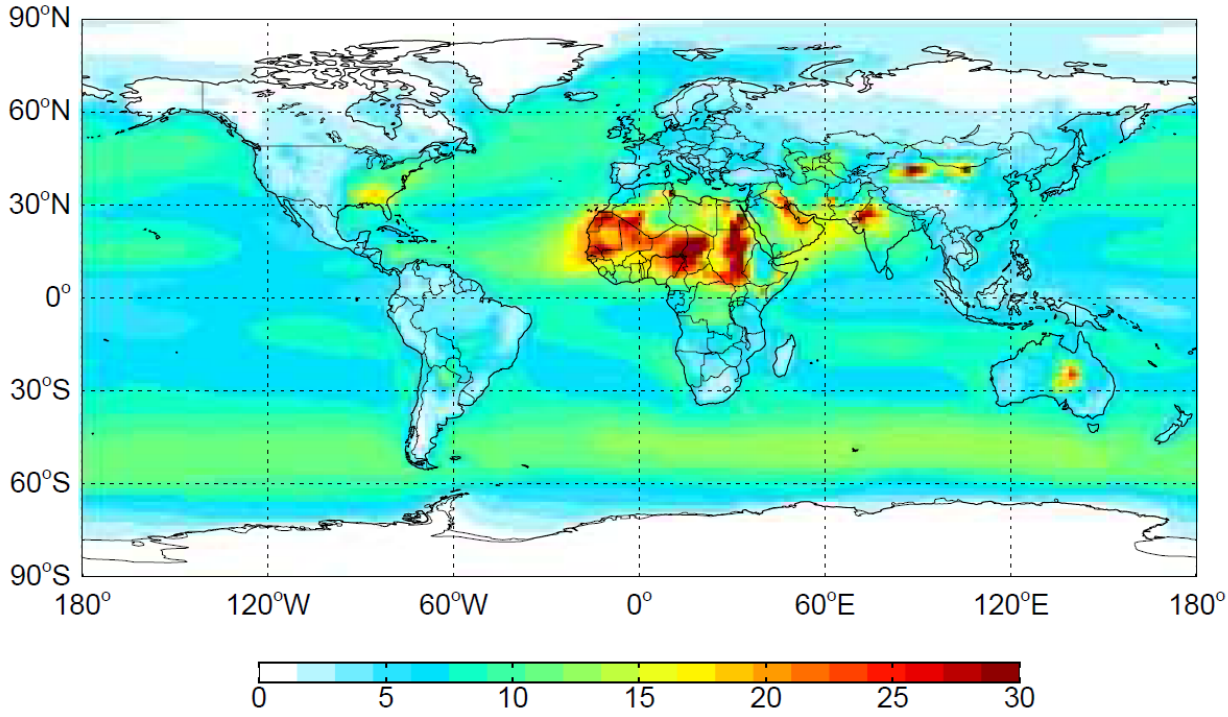


Figure S5 – Spatial distribution of $PM_{2.5}$ concentrations (sum of species) in 1850 ($\mu g/m^3$), showing the multi-model mean in each grid cell.

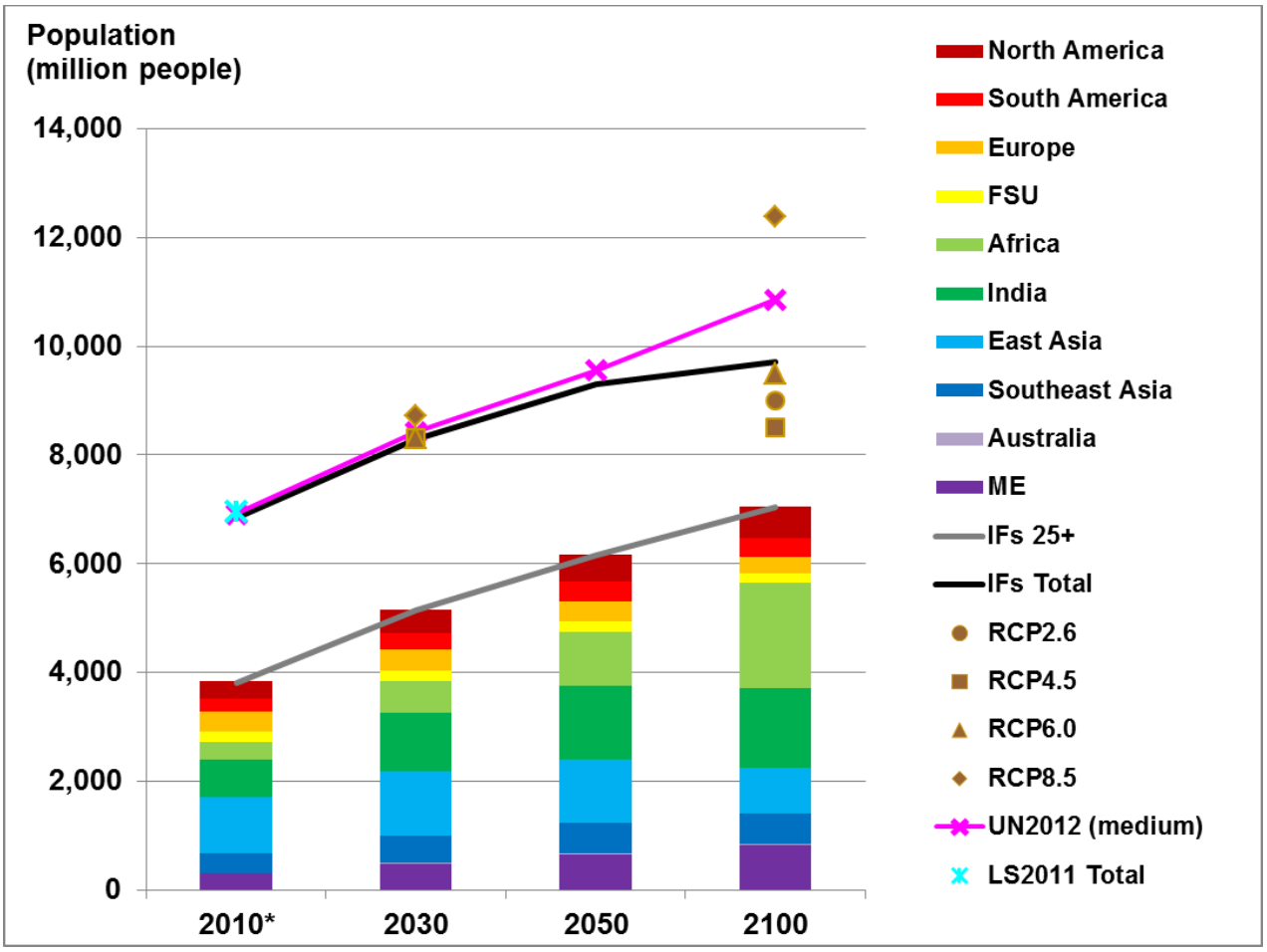


Figure ~~S4~~ S6 – Present-day and future population (millions of people) showing global totals for exposed population (adults 25 and older) from Landscan 2011 (2010) and IFs (2030, 2050, 2100), as well as total population for the RCP scenarios for 2030 and 2100 (Van Vuuren et al., 2011) and for UN Population Prospects 2012 medium fertility scenario for 2030, 2050 and 2100. Also shown are regional exposed populations for IFs.
Sources:

- Oak Ridge National Laboratory (ONRL) - LandScan 2011 Global Population Dataset, http://spruce.lib.unc.edu.libproxy.lib.unc.edu/content/gis/LandScan/. Data retrieved on 12/05/2012.
- Web-Based IFs - The International Futures (IFs) modeling system, version 6.54., www.ifs.du.edu. Data retrieved on 07/2012.
- United Nations, Department of Economic and Social Affairs, Population Division (2013). World Population Prospects: The 2012 Revision. http://esa.un.org/wpp/Excel-Data/population.htm. Data retrieved on 12/03/2013.

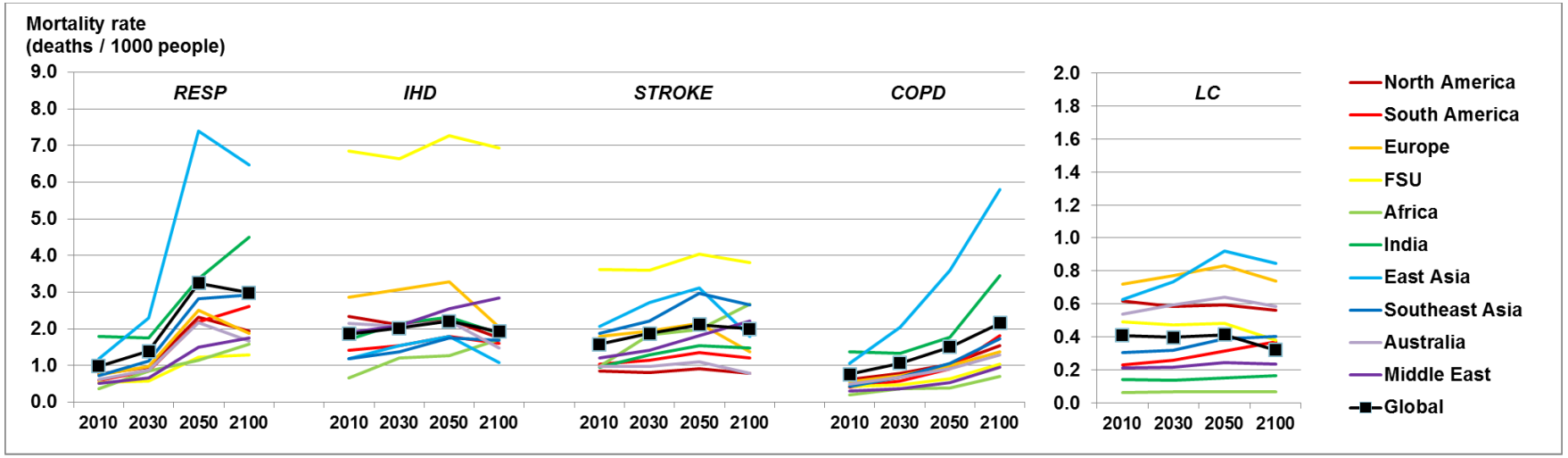


Figure ~~S5~~ S7 – Global and regional average present-day and future baseline mortality rates (deaths per 1000 people per year) for RESP, IHD, STROKE, COPD and LC, for adults aged 25 and older from the Global Burden of Disease Study 2010 mortality dataset (IHME, 2013) and IFs (2030, 2050, 2100). The IHD and Stroke averages are shown for illustration only, since the mortality estimates are obtained using baseline mortality rates per 5-year age group.

Sources:

- Web-Based IFs - The International Futures (IFs) modeling system, version 6.54., www.ifs.du.edu. Data retrieved on 07/2012.
- IHME (2013). Data retrieved from 12/2013 to 03/2014.

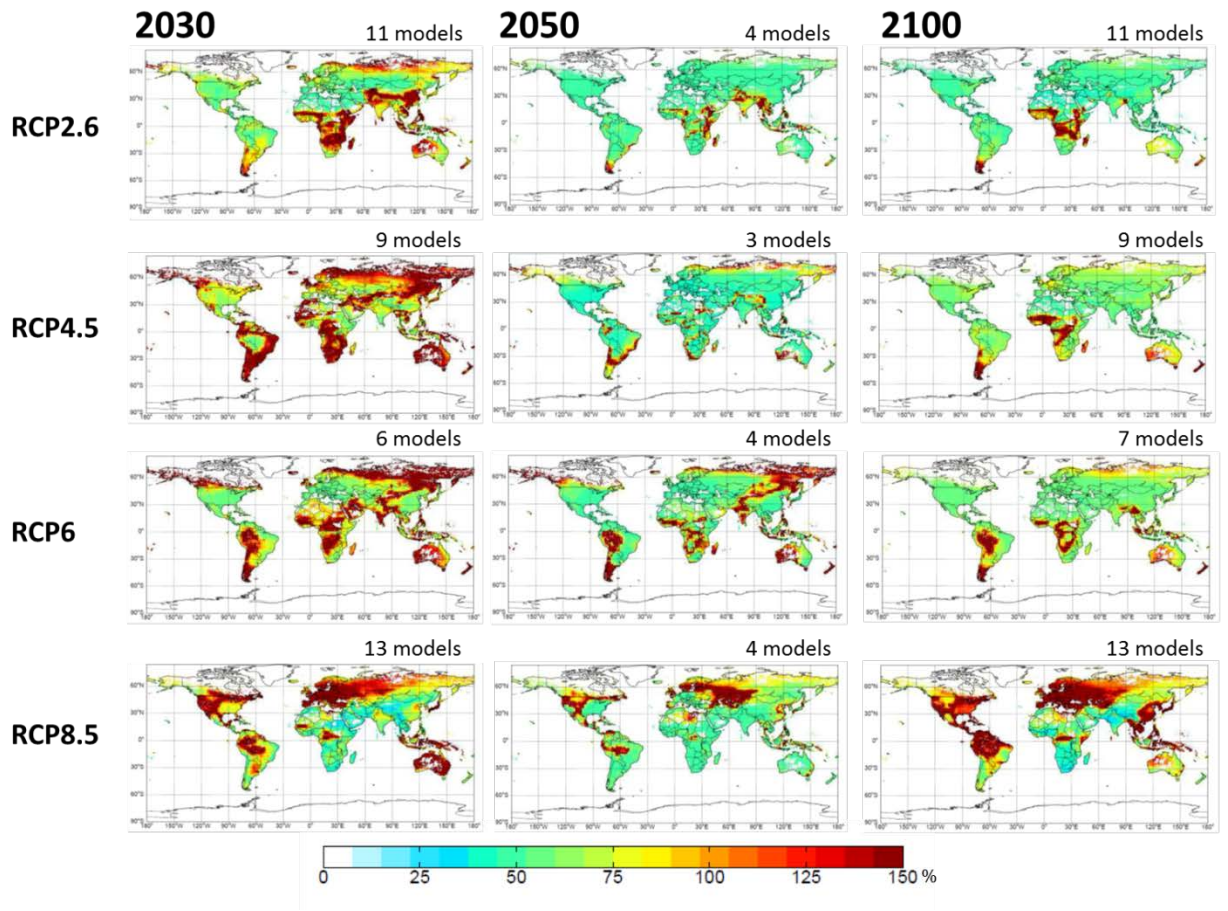


Figure S8 – Spatial distribution of model variability in future ozone respiratory mortality for all RCP scenarios in 2030, 2050 and 2100, showing the coefficient of variation of mortality estimates in each grid cell.

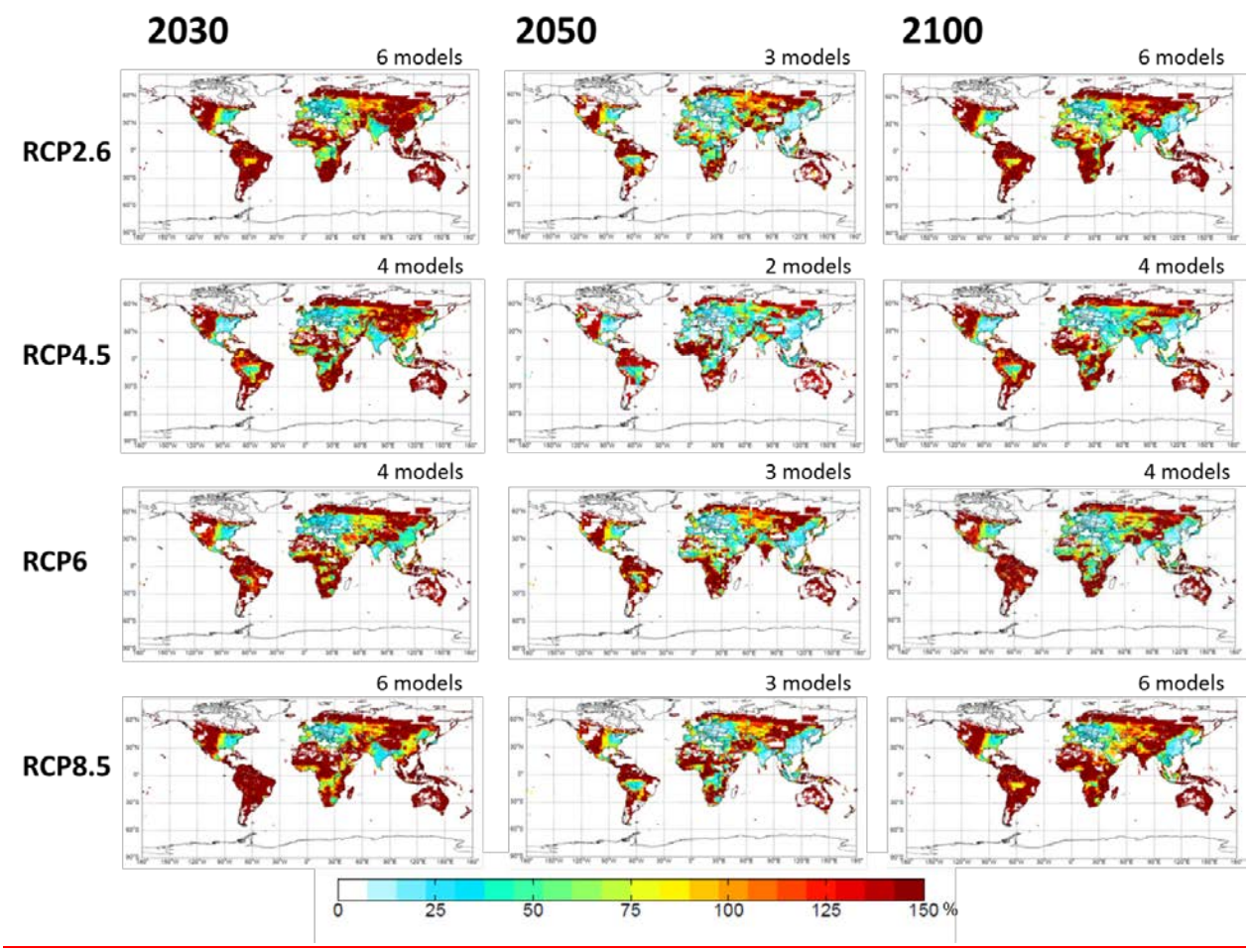


Figure S9 – Spatial distribution of model variability in future premature mortality (IHD+STROKE+COPD+LC) for $PM_{2.5}$ calculated as a sum of species for all RCP scenarios in 2030, 2050 and 2100, showing the coefficient of variation of mortality estimates in each grid cell.

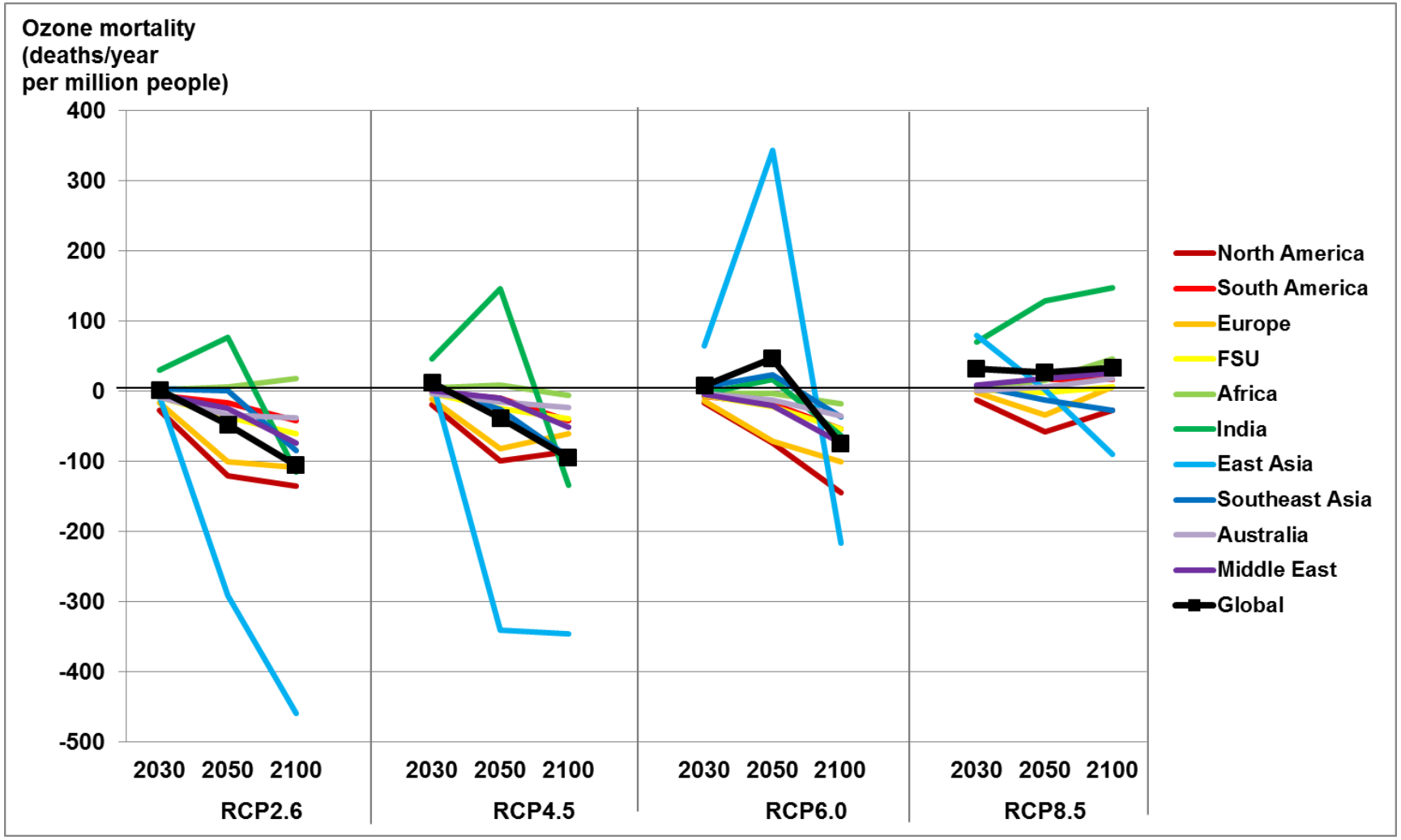


Figure ~~S6~~ S10 – Future ozone respiratory mortality per million people for all RCP scenarios in 2030, 2050 and 2100, showing the multi-model regional average (deaths/year per million people) in ten world regions (Figure S1) and globally, for future air pollutant concentrations relative to 2000 concentrations.

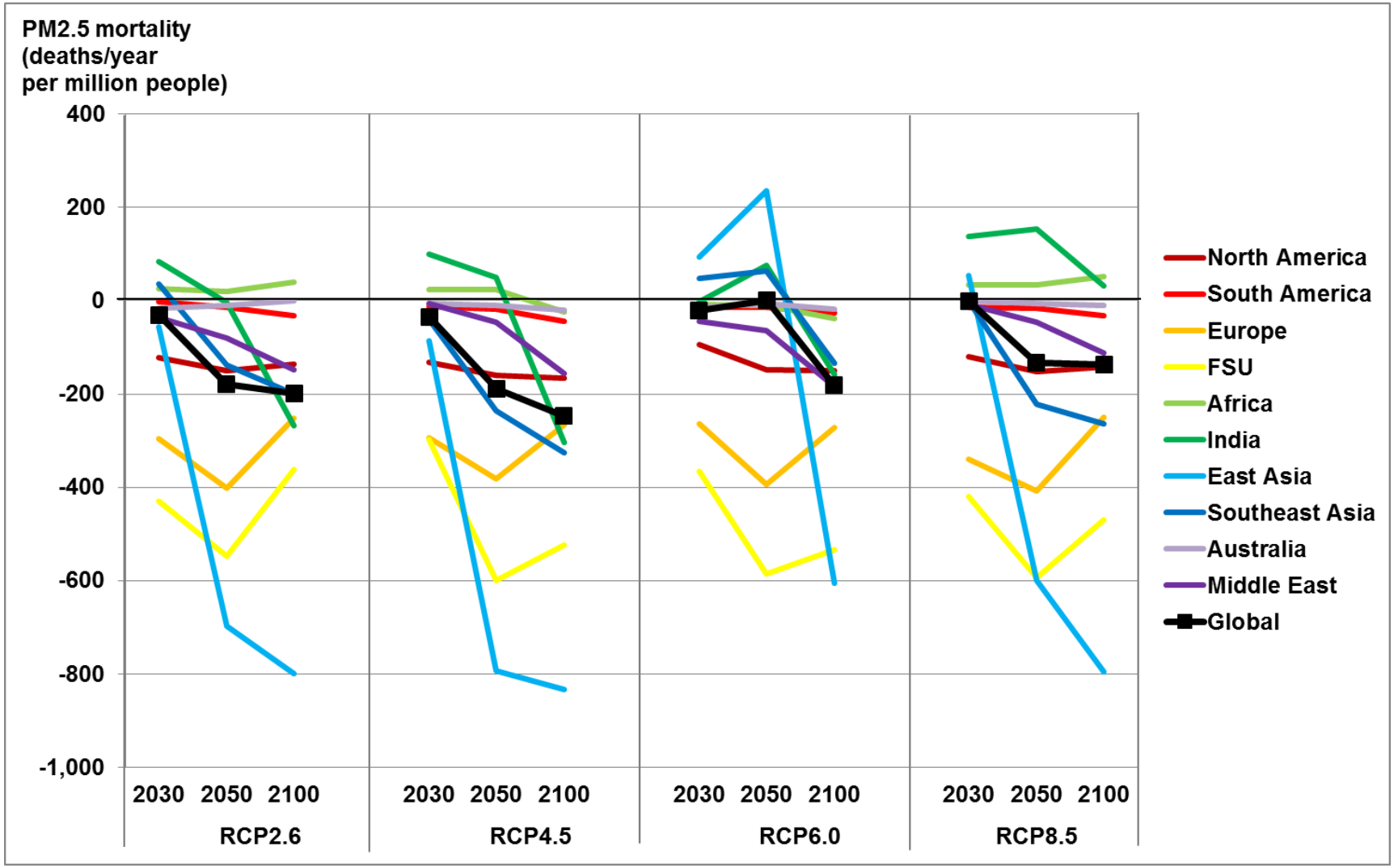


Figure ~~S7~~ S11 – Future $PM_{2.5}$ mortality (IHD+STROKE+COPD+LC) per million people for all RCP scenarios in 2030, 2050 and 2100, showing the multi-model regional average (deaths/year per million people) in ten world regions (Figure S1) and globally, for future air pollutant concentrations relative to 2000 concentrations.