Response to reviewer #2:

We thank Reviewer #2 for his/her valuable suggestions. Below are our responses. The reviewer's comments are shown in *italic*, while our responses are shown in plain font.

This paper quantifies that premature PM2.5 and ozone related deaths resulting from total industrial pollution, and the impacts of emission, climate, and methane changes specifically. This is a comprehensive paper that is generally well written. However, there is not enough information in the paper to evaluate the health impact assessment methodology. There are also additional major concerns that need to be addressed.

Major issues:

1) *The health methodology is unclear and may contain serious misjudgments. The authors refer to a submitted paper for more detail on the health methods. Is that paper available or can the authors make it available to the reviewers to judge the health methodology?*

Response (1)

Thank you for pointing this out. We have taken your advice and have added information on the health methodology used to this manuscript. We add the following sentences into Section 2.3: "The appropriate concentration-mortality response factor (β) is used for each pollutant. For $PM_{2.5}$, β is obtained from Krewski et al. (2009), which provides the latest reanalysis of the ACS study (Pope et al., 2002). They found a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentrations was associated with 6% (95% confidence interval, CI, 4-8%), 13% (95% CI, 10-16%) and 14% (95% CI, 6-23%) increase in all-cause, cardiopulmonary, and lung cancer mortality, respectively. For O3, we use (β) from Jerret et al. (2009), for which, a 10 ppb increase in O_3 is associated with a 4% increase in respiratory disease mortality." We also add a new table 2 with mortality rate and population data.

2) *Although it is difficult to understand, it seems that the authors quantify respiratory mortality for ozone but all-cause mortality for PM2.5. At this point, the health literature has been able to discern more about how PM2.5 affects health than was known at the time of the first American Cancer Society publications (including Pope et al. 2002). It is now widely appreciated that PM2.5 is most strongly associated with cardiovascular effects, while ozone is more strongly associated with respiratory effects. I agree with the authors' choice to apply long-term respiratory mortality relationships for ozone based on the results from Jerrett et al. (2009). However, I have some issues with the PM methodology. While it may be true that cause-specific mortality data from many parts of the world may be inaccurate, the authors are then*

using baseline mortality rates for respiratory disease to quantify ozone mortality. In addition, they are extrapolating these relationships for the US to the rest of the world where people are dying of very different diseases, all of which get lumped into "all-cause mortality." This is a majorly flawed assumption – in my opinion less defensible than trusting the cause-specific baseline mortality rates (which by the way are used by the WHO in the Global Burden of Disease study along with many other high-profile scientific assessments). Finally, the authors need to provide guidance on whether the all-cause PM and respiratory ozone mortality can be summed together or whether there may be some overlap between the two.

Response (2)

Previously, we believed that all-cause mortality data was more robust than mortality data on specific diseases especially in the less developed world (i.e., Africa). We therefore chose to use all-cause mortality for the case where it was applicable (i.e., for PM2.5).

However, the reviewer's perspective is well supported and we have now switched our analysis to use disease-specific mortality data for PM2.5 (including cardiopulmonary mortality and lung cancer mortality) as well as for O3 in the revised version. We have revised the abstract, Section 2.3, Section 5, Section 6, Table 2, Table 3, Figure 5 and Figure 6 correspondingly. Although mortality results reported now changes to disease-based numbers, our major conclusion about the relative importance of changing emissions, climate and methane on mortalities remains the same.

3) *In addition to making the methods contained within the submitted paper available, to allow the reviewer to judge the health methodology it is imperative to provide more information on the range of ozone and PM concentrations simulated by the model for each scenario (by region even better), the PM species included in the PM definition (are dust and sea salt included?), population-weighted average change in concentration for each set of scenarios compared, and the regional population and baseline mortality rates used to do the health quantification.*

Response (3)

We have now added a new table (Table 3) that includes populations, and baseline mortality rates for respiratory disease, lung cancer and cardiopulmonary disease for each region discussed in the paper. However, since we have 5 different simulations, it is hard to put a range of PM2.5 and O3 for each scenario and each region into a table. Besides, we feel that we already have a figure (i.e., Figure 3) in the paper that shows the values of PM2.5 and O3 in each model grid. Therefore we decide not to add the range of air pollutant concentrations into the table.

Fine sea salt and dust (with a radius less than 2.5 μm) are included as $PM_{2.5}$. $PM_{2.5}$ was defined in the introduction of this manuscript, "$PM_{2.5}$, including sulfate, nitrate, organic carbon (OC), black carbon (BC), secondary organic carbon, fine dust and sea salt, is either directly emitted from various sources or produced via chemical reactions between directly-emitted gas-phase precursors (including SO_2, NO_x, NH_3 biogenic VOCs etc.) and atmospheric oxidants (i.e., OH, H2O2, O3). " We add the definition of PM2.5 into the method part, i.e., Section 2.2, as well, see the first sentence in the revised manuscript "To investigate the change in concentrations of O_3 and $PM_{2.5}$ (including simulated sulfate, nitrate, small dust, small sea salt, organic carbon, black carbon, secondary organic carbon) during the industrial period, we use AM3 time-slice simulations for "1860" and "2000".

4) *In general, it would be useful to describe the health methods inside this paper in more detail, as ACP readership may not be familiar with them. At the very least, the relative risk estimates should be noted in the text, not just in Table 2.*

Response (4)

Yes, good point. We agree with the reviewer and have added a description of the health methodology to the paper. Please check Response (1) and (2)

5) *I have concerns about the results of the model evaluation in Section 3. The authors state that bias over populated areas in the US and Europe ranges from 4-10ppb and correlation coefficients are >0.7 in the US and >0.95 in Europe. Figure 1 shows that bias in the Northeast and Southeast US is around 14ppb, higher than the range given in the text for populated areas in the US. Some of the biases in Europe also reach much higher than 10ppb. These results do not demonstrate to me that the model is predicting ozone concentrations well for the locations where we have monitored observations.*

Response (5)

Thanks to the reviewer for pointing this out. The bias in our GFDL-AM3 2000 simulation (driven by 1995-2004 mean SST and sea ice from a coupled model, GFDL CM3) varies from 3.8 (over Mountainous West US) to 22.6 (over North West US) ppbv, when compared to the CASTNet (1988-2009) and EMEP (1987-2008) observations. We now clarify this in the manuscript.

Our GFDL-AM3 "2000" simulation has been analyzed within the ACCMIP campaign along with other coupled chemistry-climate models, and shows no large differences from other models [*Young et al*., 2012]. Positive bias in surface O3 has been a common problem even in chemistry transport models that are driven with assimilated

meteorological fields. Conclusive explanations and improvements have been elusive, but contributing factors likely include the coarse resolution in global models, isoprene chemistry, stratospheric-tropospheric exchange and insufficient boundary layer ventilation. For example, the bias in the Eastern US in MOZART can be 10-15 ppbv [*Lin et al*., 2008; *Murazaki and Hess*, 2006; *Fang et al*., 2010] and the positive bias occurs systematically in the multimodel estimates of surface O3 for the year 2001 of the Task Force on Hemispheric Transport of Air pollution as well [*Fiore et al*., 2009; *Reidmiller et al*., 2009].

In *Naik et al.* (2012), a similar GFDL-AM3 simulation (driven by observed sea surface temperature (SST) and sea ice during 1981-2000, hence more comparable with the observations) was evaluated, and the bias of AM3 dropped by 1-4 ppbv in almost all regions except the Midwest Plains (Figure 1). The bias in our 2000 simulation may also be resulting from biases in simulated meteorology because we drive the model with SST and sea ice cover simulated by the GDFL coupled model (GFDL-CM3) as described in Section 2.2.

However, because our study focuses on differences in air quality and the associated health effect between “1860” and “2000” simulations, we expect that biases in the individual simulations will at least partly cancel when the difference between the two simulations is examined.

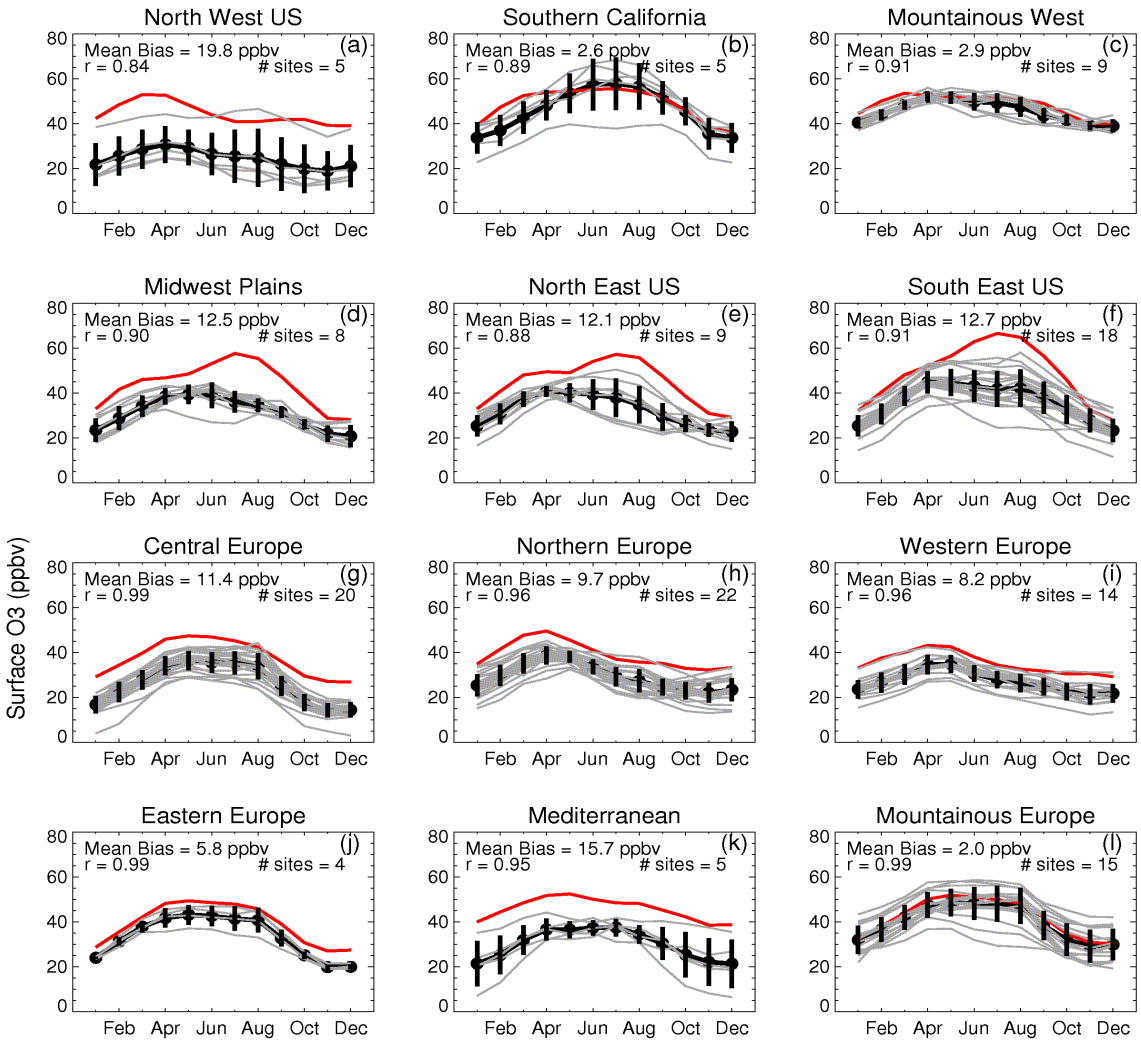

Figure 1. (Cited from *Naik et al*., in revision for JGR) Comparison of GFDL-AM3 simulated monthly mean surface ozone for 1981-2000 (driven by observed SST and Sea Ice, red line) with measurements from CASTNet (http://www.epa.gov/castnet/) in the United States, EMEP (http://www.nilu.no/projects/ccc/emepdata.html) in Europe, and three sites in India. Observed values (black dots) represent the average of climatological monthly mean surface O_3 concentrations for sites (grey lines) falling within each region. Vertical black lines denote standard deviation across the sites. For Indian sites, vertical lines represent standard deviation in measurements.

6) *In addition, the text description of the PM evaluation does not do justice to what looks like an extremely low bias and substantial variation in the direction of that bias in locations in the US, Europe, and Asia. As with ozone, these results do not demonstrate to me that the model is predicting PM well for the locations where there are monitored observations. In addition, many of the relative difference values in Europe and Asia look like they are overpredictions (i.e. red), but there are very few overpredictions shown on the scatterplot. Are some of the data not shown on the*

scatterplot or am I misinterpreting the values in the map? Regardless, more explanation is needed as to what the scatterplot and map depict, and how they should be interpreted for this study.

Response (6)

Evaluation in Europe and Asia focused on sulfate aerosols. Sulfate values are much lower than the total PM2.5 concentrations evaluated for North America.
Values of sulfate aerosols in Europe and Asia are shown as green triangles in the scatter plot. Since most sulfate aerosol values over Europe and Asia are lower than 2 $\mu g/m^3$, the overestimate is not shown as distinctively as the underestimate in PM2.5 over North America (most values between 5 and 20 $\mu g/m^3$, shown as black squares). We have clarified the figure caption to provide this additional information.

7) *Why are preindustrial PM concentrations so high? Can these values be set in the context of other model studies using the same emissions inventories (i.e. other models running ACCMIP emissions)? Previous studies with which I am familiar show preindustrial PM very close to zero. Perhaps the PM2.5 definition includes dust and sea salt, which have been excluded by many studies in the past, including Anenberg et al. (2010)? Please describe in the text what is included in the PM definition and consider whether your definition influences comparisons made to Anenberg et al. (2010). If dust and sea salt are excluded, the degree to which this affects the conclusions regarding relative importance of emissions, climate, and methane depends on why preindustrial PM is so high.*

Response (7)
Our PM2.5 definition includes fine dust and sea salt, which is different from *Anenberg et al.* (2010), and hence our PM2.5 values should be (and are) higher than theirs. We now clarify this in the paper.

However, our preindustrial global mean PM2.5 concentration is more than three times of that in their study, which cannot be explained solely by the difference in definition of PM2.5. Besides, from our analysis, effects of CLIM and TCH4 on PM2.5 are less than 10% of that driven by EMIS. Therefore, we conclude that differences in precursor emissions are the main cause for the different preindustrial PM2.5 concentrations in the studies.

Anneberg et al. (2010) use simulations from *Horowitz et al.* (2006), which applies 0% fossil fuel emission and 10% of their 1990 biomass burning emissions for the preindustrial case. However, the emission inventory we apply in our study [*Lamarque et al*., 2010] reflects recent findings that preindustrial biomass burning emissions are higher than present day over some regions, i.e., the North America [*Mieville et al*., 2010] and that anthropogenic emissions are already significant in 1850. As a result, preindustrial NOx, BC, OC emissions in our study are a factor of 1.8, 3.5, 2.6 higher, respectively, than those in *Horowitz et al*. [2006]. We have already summarized the emission

differences in Table S2 in the supplementary material and also in the text of Section 4.1 in our manuscript.

Currently, there is no published study that compares preindustrial PM2.5 concentrations simulated by various chemistry-climate models using emission inventory of Lamarque et al. (2010). However, GFDL AM3 simulations for the period 2005-2100 using different RCP emission scenarios are analyzed along with many other chemistry-climate models in the ACCMIP project, and the results of PM2.5 simulations are shown in Figure 5 in Fiore et al. (2012). The standard deviations are small in general across these different models, indicating that AM3 simulated PM2.5 are consistent with other models when using the same emission inventory.

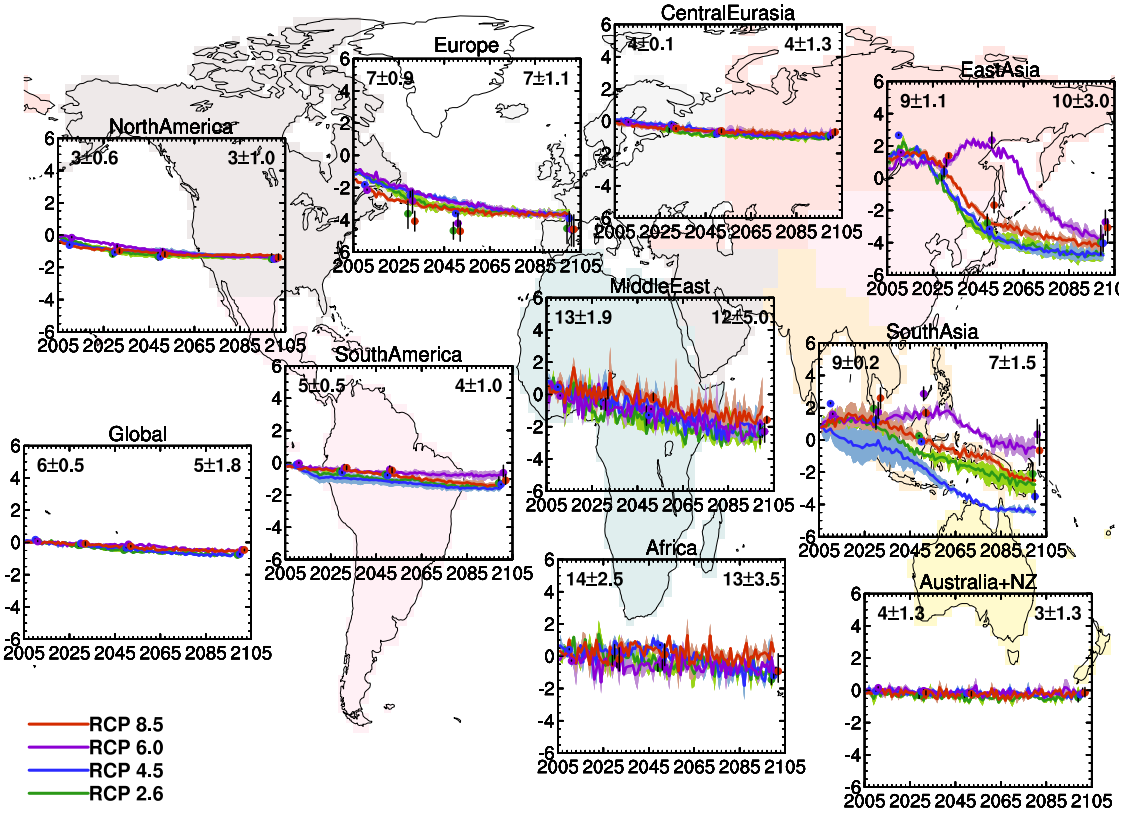

Figure 2. (Cited from Fiore et al., 2012, Figure 5) Changes in annual mean surface ozone (ppb mole fraction) averaged over selected world regions (shaded land regions) following the RCP scenarios. Colored lines denote the 4-model average from transient simulations with CMIP5 chemistry–climate models; shading covers the full range across models (Table S1, ESIz). Filled circles with vertical lines represent decadal multi-model averages from the 2010, 2030, 2050, and 2100 ACCMIP time-slice simulations; the number of models varies with time slice and scenario (Table S1, ESIz). The circles are colored by RCP scenario and offset by a year to clearly distinguish the vertical black lines which denote the full range of ACCMIP model results. Changes are relative to the 1986–2005 reference period for the transient simulations, and relative to the average of the 1980 and 2000 decadal average time slices for the ACCMIP ensemble. The average value for this reference period, averaged across all models is shown in each panel, with the standard

deviation reflecting the model range (transient CMIP5 models on the upper left; ACCMIP models on the upper right). In cases where multiple ensemble members are available from a single model, they are averaged prior to inclusion in the multi-model mean.

8) *There needs to be much more comparison of your results with previous studies, for both the effects of climate on PM and ozone and the health results. Please put the results showing the climate impacts of PM in context by discussing the conclusions of Jacob and Winner (2009), who note that the literature is inconclusive with regard to the directional impact of climate on PM. The impact of methane on ozone-related mortality could be compared with results from West et al. (2006), Anenberg et al. (2009), and Anenberg et al. (2012).*

Jacob, D.J., Winner, D.A. (2009) Effect of climate change on air quality, Atmospheric Environment, 4: 51-63.

West, J. J., A. M. Fiore, L. W. Horowitz, and D. L. Mauzerall (2006) Global health benefits of mitigating ozone pollution with methane emission controls, Proceedings of the National Academy of Sciences, 103(11): 3988-3993.

Anenberg, S.C., J.J. West, A.M. Fiore, D.A. Jaffe, M.J. Prather, D. Bergmann, C. Cuvelier, F.J. Dentener, B.N. Duncan, M. Gauss, P. Hess, J.E. Jonson, A. Lupu, I.A. MacKenzie, E. Marmer, R.J. Park, M. Sanderson, M. Schultz, D.T. Shindell, S. Szopa, M. Garcia Vivanco, O. Wild, and G. Zeng (2009) Intercontinental impacts of ozone pollution on human mortality, Environmental Science and Technology, 43(17):6482-6487.

Anenberg, S.C., J. Schwartz, D. Shindell, M. Amann, G. Faluvegi, Z. Klimont, G. Maenhout, L. Pozzoli, R. van Dingenen, E. Vignati, L. Emberson, N.Z. Muller, J.J. West, M. Williams, V. Demkine, W.K. Hicks, J. Kuylenstierna, F. Raes, V. Ramanathan (2012) Global air quality and health co-benefits of mitigating near-term climate change through methane and black carbon emission controls, Environmental Health Perspec- tives, 120:831-839.

Response (8)

We have added a comparison to these studies in our paper where applicable. Please check Section 4.3.1, 4.4, 5.2 and 6.

Minor issues:

9) *P. 22714 lines 15-19: Explain the standard deviation values given for the concentration*

Response (9)

We added an explanation to the abstract, "(results reported as annual average ± standard deviation of 10 year model simulations)". The standard deviation is defined as the root mean square of variance of the 10-year simulations, as shown in the last sentence of Section 2.2.

10) P. 22714 line 26: Add "above preindustrial"?

Response (10)

The sentence is changed to "However, changing climate and increasing CH_4 concentrations also contribute to premature mortality associated with air pollution globally (by up to 5 % and 15 %, respectively)."

11) P. 22714 lines 26-28 and throughout, it seems odd to lump together climate and methane impacts, since the methane increases since preindustrial presumably are mainly driven by methane emissions. Hence, the methane impact really adds to the impact of the growth in the other emissions as the total impact of anthropogenic emissions since preindustrial. Suggest removing this sentence as it doesn't add anything beyond the preceding sentence anyway.

Response (11)

Methane has a long lifetime (~10yr), and its concentration is prescribed in each simulation rather than being driven by emissions in the GFDL AM3 model.

Besides, due to its longer lifetime, the effect of CH4 concentration increases is a global issue, which means that mitigation results in benefits for health and climate globally. In this respect, CH4 increase and climate change are similar to each other, in that they have a global effect, while differ from changes in emissions of short-lived species, whose effects are primarily local and for which local mitigation efforts provide the largest benefits to the regions that reduce their emissions. Therefore, we added climate and methane effects together to compare a global-scale effect (may impact regions removed from location of emission) vs. local effect (impacts are mostly felt near the location of emission).

12) P. 22715 line 11: This is a factor of 5, not 4.

Response (12)

The measured surface O3 in 2000s **is a factor of 5** of that in 1860s, however, what we mean here is that O3 **increases by a factor of 4**. They are consistent with each other.

13) P. 22716 line 10: Anenberg is misspelled

- P. 22718 line 24: missing "."

- P. 22720 line 3: "its"' misspelled

- P. 22721 line 21: missing "."

Response (13)

Thanks. All corrected.

14) P. 22721 line 15: Anenberg et al. (2010) did not separate out IHD from cardiopulmonary

Response (14)

Corrected.

15) P. 22726 line 18: define somewhere whether biomass burning includes open fires only, or also ag burning and residential cookstoves

Response (15)

They were defined in Section 2.2, Line 24-28 in the original manuscript. "Anthropogenic emissions (including from energy production, industry, land transport, maritime transport, aviation, residential and commercial sectors, solvents, agriculture, agriculture waste burning on fields, and waste) and biomass burning emissions (including from open vegetation fires in forests, savanna and grasslands) of short-lived air pollutants…"

16) P. 22726 line 20-22: This statement implies that anthropogenic emissions increase homogenously. . . not true, even if they do increase everywhere.

Response (16)

We changed the sentence to “Anthropogenic emissions increase during the industrial period almost everywhere while changes in biomass burning emissions driven by human activities vary in sign depending on location. ”

17) P. 22731 line 2: J-value is not defined

Response (17)

J-Values are the values of photochemical reaction rate. We changed it to read “photochemical reaction rate”.

18) P. 22732 line 27: state whether the 15% increase is only valid for pop-weighted total ozone. Would it be a larger fraction of non-population weighted ozone, since methane influences ozone everywhere while shorter-lived precursors influence ozone more near populated areas?

Response (18)

Yes, the reviewer is right. If we consider only the land area, the increase of O3 driven by CH4 concentration increase remains similar when weighted with area as opposed to weighted by population. However, the increase of O3 driven by all factors becomes 20.5 ppbv when weighted by area (compared to 30 ppbv O3 increase when weighted by population). As a result, the fraction becomes 22% when weighted by area. We now make it clear in the paper that this fraction is only valid for population weighted O3; for area-weighted O3, the fraction is higher. We added this into the paper.

19) p. 22736: Do you really need to define a new term for the mortality contribution? No information would be lost and it would read more easily if you eliminate section 5.2.4 with the new term and simply provide the percent contributions of each factor in 5.2.1-5.2.3 (which are currently very short).

Response (19)

The reviewer’s comment is well taken. We have revised this section based on his/her suggestions. Now, Section 5 does not have any subsections.

20) P. 22738 lines 17-18: Suggest rephrasing the reference to SRES and RCP as these have not been previously defined.

Response (20)

We now include reference to these two terminologies, "In the meantime, CH_4 is projected to increase in almost all SRES (Nakicenovic et al., 2000) and RCP (Meinshausen et al., 2011) emission scenarios (except RCP2.6 and SRES B2)."

21) P. 22750: Header for regions is mislabeled

Response (21)

Thanks. Corrected.

22) P. 22753: T-test results are useful, but it is very difficult to see the map underneath since the maps are so small. Is there another way to provide the same information without obscuring the maps? Figure 4 is easier to see since it is bigger

Response (22)

We agree with the reviewer that these two figures are small in the ACPD publication. The size of original plots is reasonable, however. When they were formatted for ACPD, they shrank. We will work with the publication staff to be certain they are of legible size in ACP.

References:

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 Persp., 118, 1189–1195, doi:10.1289/ehp.0901220, 2010.

Fang, Y., A. M. Fiore, L. W. Horowitz, H. Levy II, Y. Hu, and A. G. Russell (2010), Sensitivity of the NOy budget over the United States to anthropogenic and lightning NOx in summer, J. Geophys. Res., 115, D18312, doi:10.1029/2010JD014079

Fiore et al. (2012), Global air quality and climate, Chem. Soc. Rev., 41, 6663–6683, DOI: 10.1039/c2cs35095e

Fiore, A. M., et al. (2009), Multimodel estimates of intercontinental source-receptor relationships for ozone pollution, J. Geophys. Res., 114, D04301, doi:10.1029/2008JD010816

Lin, J.-T., et al. (2008), Global model simulation of summertime U.S. ozone diurnal cycle

and its sensitivity to PBL mixing, spatial resolution, and emissions, Atmos. Environ., 42(36), doi:10.1016/j.atmosenv.2008.08.012

Mieville, A., Granier, C., Liousse, C., Guillaume, B., Mouillot, F., Lamarque, J. F., Gregoire, J. M., and Petron, G.: Emissions of gases and particles from biomass burning during the 20th century using satellite data and an historical reconstruction, Atmos. Environ., 44, 1469–1477, doi:10.1016/j.atmosenv.2010.01.011, 2010.

Murazaki, K. and Hess, 5 P.: How does climate change contribute to surface ozone change over the United States?, J. Geophys. Res., 111, D05301, doi:10.1029/2005jd005873, 2006.

Naik, V., Horowitz, L. W., Fiore, A. M., Mao, J., Aghedo, A., and Levy, H. I.: Preindustrial to present day impact of short-lived pollutant emissions on atmospheric composition and climate, J. Geophys. Res.-Atmos., in revision, 2012.

Reidmiller, D. R., et al. (2009), The influence of foreign versus North American emissions on surface ozone in the US, Atmos. Chem. Phys., 9, 5027–5042, doi:10.5194/acp-9-5027-2009

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. Discuss., 12, 21615-21677, doi:10.5194/acpd-12-21615-2012, 2012.