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Atmospheric Chemistry and Physics Discussions

Interactive comment on "Effect of regional precursor emission controls on long-range ozone transport – Part 2: steady-state changes in ozone air quality and impacts on human mortality" *by* J. J. West et al.

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Response to Anonymous Referee 2.

We thank Referee 2 for his/her thoughtful comments and attention to this manuscript. Referee 2 identifies "weaknesses in the methodology and the application of a global model to examine health effects," which we respond to below.

1. Page 7084. Please explain more clearly the use of the scaling described in lines 9-13 and exactly how the surface ozone concentration change is determined form the

estimated methane response. Is methane dynamic or prescribed in the model? The Fuglestvedt et al. (1999) method for determining the methane change (and secondary ozone effects) based on the initial change in the methane lifetime is useful for determining the global scale responses i.e. global ozone burden change. It is not clear in the text how the spatial changes to surface ozone are determined from these global methane changes. The surface ozone response to a global methane change will not be spatially homogeneous. It will depend on the local short-lived precursor emissions that may easily change over the lifetime of the methane perturbation. How is this taken into account?

The methods used here are described in detail in West et al. (2007a) and Fiore et al. (2009). Because they are presented in detail elsewhere, we chose to summarize these methods as clearly and succinctly as we could in the present paper, while referring the reader to these other sources for more detail. The second paragraph of Section 2 reads:

"Long-term changes in ozone due to changes in CH_4 concentrations are assessed following the methods detailed in West et al. (2007a) and Fiore et al. (2009), which are based on Fuglestvedt et al. (1999) and Naik et al. (2005). Since the 10% regional changes in anthropogenic NO_x emissions are assumed to be sustained in the future, the long-term change in ozone is added to the direct short-term change to give the net change at steady-state. The change in CH_4 lifetime is assessed from the simulated change in OH; on this basis, we estimate the steady-state change in CH_4 concentration using a CH_4-OH feedback factor of 1.33, which was diagnosed for MOZART-2 under the same conditions West et al. (2007a), and is equivalent to the mean of several models Fiore et al. (2009). We then scale the hourly ozone change in each grid cell from a simulation where anthropogenic CH_4 emissions are reduced by 20% under the same conditions West et al. (2007a), to the estimated change in CH_4 at steady state, for each regional NO_x reduction case. We add this long-term change in ozone via CH_4 to the short-term ozone change simulated directly in the model, to give the

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net change at steady state; the steady-state ozone is calculated as a full time-varying three-dimensional field, thereby accounting for the spatial distribution on each day of the net steady-state change in ozone. For sustained emission reductions, the net change in ozone will rapidly reflect the short-term change, and gradually approach the steady-state change with a timescale equal to the 12-year perturbation lifetime of CH_4."

We first calculate the steady-state change in methane from the change in OH, associated with the regional emission change, using standard methods. Then we scale change in ozone from a 20% reduction in methane to the change in methane we calculate – and we do so for each grid cell (in three dimensions) and each hour. This is described mathematically in the supporting information for West et al. (2007a), and we have copied that text here:

"Three-dimensional O_3 concentration fields at steady state for each hour ($[O_3]_{ss}(i,j,k,t)$) are created by adding the long-term change in O_3 due to the change in CH_4 to the short-term O_3 concentration ($[O_3]_{st}$) from the modeled NO_x, NMVOC, and CO emission perturbation runs, where the long-term change in O_3 concentration is calculated by scaling to the -20% CH_4 emission perturbation experiment:

$$[O_3]_{ss}(i,j,k,t) = [O_3]_{st}(i,j,k,t) + ([O_3]_{CH4}(i,j,k,t) - [O_3]_b(i,j,k,t)) (([CH_4]_{ss}-1700)/(1460-1700))$$

where $[O_3]_{CH4}$ is the O_3 concentration in the -20% CH_4 perturbation experiment, $[O_3]_b$ is the O_3 concentration in the base simulation, the CH_4 concentrations in the CH_4 perturbation and base simulations are 1460 ppbv and 1700 ppbv, respectively, and $[CH_4]_{ss}$ is calculated as above for the NO_x, NMVOC, and CO perturbation simulations."

This method is therefore similar to the more common methods, based on Fuglestvedt et al. (1999), which estimate the global change in ozone. However, we account for the spatial and temporal (seasonal/hourly) distributions of the long-term change in ozone by scaling to a methane perturbation experiment. In doing so, we account for the regions of the world where differences in short-lived precursors will influence the effect

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of CH_4 on ozone.

2. Estimates of health effects should not be performed at the current resolution of the model (2.8 x 2.8 degrees) or even at 1 x 1 degree resolution, as concentrations, thus health effects, in urban areas vary significantly across short distances, and such variations cannot be captured by a global model without nesting.

We share the concerns of Referee 2 about the importance of model resolution for assessing health impacts. While some studies use global models to assess the health effects of air pollution, including previous studies from our group, resolution can pose important uncertainties. In this case, the emphasis is on long-range transport, which would be expected to influence mainly the background concentrations of pollutants. While the coarse global model would not be expected to accurately reproduce the ozone peaks in urban areas, it is the best tool available for estimating the changes associated with long-range transport, especially as we find in the companion paper that the transport of ozone is more important than the transport of precursors. The coarse-grid global model is the current state-of-the-art for assessing long-range transport. An improvement on this state of the art would use a regional model nested within the global model, as Referee 2 suggests, but that is clearly impractical for all receptor regions and perturbation experiments considered here.

In addition to its ability to simulate changes in ozone, Referee 2 is correct to question how the changes in ozone in the global model relate to population, particularly in urban areas where there are strong gradients of population over short distances. Again we expect that the changes in ozone due to long-range transport are reasonably well-captured, and we expect that errors from using the global model may be highest within the source region. Indeed, Fiore et al. (2009) find substantial inter-model variation in the HTAP model responses within the source regions, as we discuss in the paper. Future research would be valuable to quantify the possible errors in estimates of population-weighted concentrations and health effects resulting from using coarse resolution global models, relative to models with finer resolution. The authors of this

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paper would like to pursue systematic research addressing these questions in the near future, perhaps using a nested regional model.

In the conclusions section, we have expanded our discussion of these uncertainties and suggest that further research is needed in this area:

"In their multimodel intercomparison, Fiore et al. (2009) find that there is substantial variation between models in their estimated impacts of emission reductions within the source regions, as global models with different emissions have different local sensitivities. Anenberg et al. (2009) show that for this multimodel ensemble, the relative mortalities within versus outside of source regions is strongly influenced by uncertainty in the intra-regional change in ozone among the different models. While a global model is currently the best tool available for addressing long-range transport, estimates of the population-weighted change in concentration within a source region (and mortalities) should be analyzed by regional models at finer scales, that have been tested extensively for local conditions. Further research should also quantify the possible bias from using coarse-resolution global models to estimate health effects."

3. An assumption of the paper is that the methane change is globally homogeneous. How true is this assumption? For example, oxidation capacity varies enormously from region to region.

Methane is long-lived in the atmosphere (perturbation lifetime of 12 years), so that its concentration in the troposphere is nearly uniform with differences between hemispheres of 5-10%, except for very near large sources. Changes in methane emissions from different world regions have been shown to have little influence on the spatial pattern of the change in ozone (Fiore et al., 2008); similarly spatial differences in the destruction of CH_4 by reaction with OH would be expected to have little influence on the long-term ozone. While the CH_4 concentration is assumed to be uniform, however, we account for variability in short-lived precursors and other factors on the spatial distribution of the long-term ozone, as discussed in 1 above. This results in, for example,

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a greater change in ozone due to changes in methane in the Northern Hemisphere than in the Southern Hemisphere.

4. Of the multiple health effects of ozone, why was premature mortality selected as the outcome in this study?

We agree that it will be helpful to clarify this point. We have added this sentence to section 3:

"While ozone may cause other human health effects, we estimate mortalities because it often dominates over other health effects when monetized, and because global data on baseline incidence rates are lacking for other health effects."

5. The actual ozone responses are tiny (ppt) level and are probably dwarfed by inter-annual variability. Please provide a measure of the expected uncertainty range for all the results.

We agree with Referee 2 that we should communicate uncertainty more clearly. In the companion paper to this one, we have taken several steps to improve the communication of uncertainty in our estimates of long-range transport, and reference the variability of long-range transport in studies that have estimated this. As in Part 1, we do not use a coupled chemistry-climate model, and compare all simulations using identical meteorology, for which a single year of meteorology is not unusual in the literature. The inter-annual variability in the total concentration of ozone (at any given location) may be large compared with our estimates of long-range transport, but the more important question for us would be the inter-annual variability in long-range transport itself, which is discussed in the companion paper.

In the present manuscript, it is important for us to communicate uncertainty in the long-term ozone change, and in the estimates on mortality. For the long-term change in methane, we now compare our results with the range from the HTAP model intercomparison, in the footnotes to Table 1 and this new sentence in Section 2:

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"In Table 1, these changes in CH_4 are also compared to the results from 20% regional reductions of NO_x in the HTAP model intercomparison (Fiore et al., 2009). The changes in CH_4 per unit change in NO_x emissions are smaller for the four regions here than in HTAP, but within one standard deviation for three regions (except for EU)."

We likewise guide the reader in interpreting the uncertainty in estimates of long-term ozone, by referring to the HTAP results in this sentence:

"The uncertainty in the long-term surface ozone change can be given by the combination of uncertainties in the CH_4 change ($\pm$20-40%) and the change in global ozone with respect to CH_4 ($\pm$20%), estimated for the several models in the HTAP model intercomparison (Fiore et al., 2009)."

We think that these are worthwhile additions to the paper, especially as this range of changes in methane had not been reported previously in the HTAP publications, and we thank Referee 2 for pushing us in that direction.

For the mortality results, we direct the reader to related studies that we completed earlier, and that included a more thorough uncertainty analysis. We likewise identify and discuss the major sources of uncertainty in the conclusions section. In section 3.2, we focus our analysis of sensitivity on the particular question of the relative mortalities within versus outside of the NA, EU, and FSU source regions.

We have changed our presentation to highlight this uncertainty further. We now report the 95% posterior interval on the results from Bell et al. (2004), when we report the value of Beta. We have likewise changed the text in section 3 to read:

"Estimates of avoided mortalities are uncertain to $\pm$50% due to the uncertainty in β from Bell et al. (2004), or potentially greater uncertainty considering the literature as a whole. Other major sources of uncertainty result from the low-concentration threshold, the application of the ozone-mortality relationship in less industrialized nations, and the uncertainty in the modeled ozone response itself; these uncertainties have

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been explored elsewhere (West et al. 2006; 2007b). Here we emphasize the relative changes in mortality in different receptor regions, due to regional emission reductions and inter-regional transport, rather than the total numbers of estimated mortalities. We test the sensitivity of our major findings for mortalities due to only cardiovascular and respiratory (CR) mortalities, and for alternative low-concentration thresholds."

6. What is the physical meaning of a negative 'avoided mortality'?

Negative avoided mortalities result in this study where concentrations of ozone increase. The rate of mortality would increase due to the increase in ozone, expressed in deaths per year.

7. The dose response relationships have been developed based on much higher observed ozone change levels. Is it appropriate to use these relationships for such tiny ozone changes?

This is an interesting question. The epidemiologic study we use (Bell et al., 2004) makes use of all of the available daily ozone measurements from 95 US cities between 1987 and 2000. Other studies are comparable, but this is a particularly large dataset for a single study, which is partly why we selected this study. These studies regress changes in mortality against changes in ozone concentration. Within that large dataset, some points will be at the extremes of ozone concentration, with many points near the average of observed values, separated by very small differences in concentration. It is not the size of the change in ozone wthat is more or less uncertain in estimating effects on mortality, rather the absolute concentrations at which those changes take place; estimates are more uncertain at very high or very low concentrations of ozone, at which there are fewer observations in the epidemiologic studies. Our use of the average concentration-response relationship from this study to reflect the effects of very small changes in concentration is entirely consistent with the original epidemiologic study.

8. The model health results are interesting and provocative but may not be meaningful because of the coarse grid resolution. Does it make (common) sense that 10% NOx

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emissions reductions in North America have substantial impacts on mortality in India and East Asia or the same reductions in Europe have substantial impacts on mortality in Africa? Table 5 seems to strongly reflect regional population density. Does epidemiological/observational evidence exist to support such long-range influences based on tiny changes in surface pollutants? The India and China regions feature huge and increasing loadings of pollution from local emissions sources. Long-range pollution transport health impacts must be negligible compared to health impacts of local pollution in these regions? In the real world, the US has experienced a 20% increase in NOx emissions in the past 10 years and reductions have also occurred in Europe. Is it possible to detect the influence of these real world perturbations on human health in the developing nations and locally? Have avoided mortalities decreased in Europe due to these NOx reductions?

The common sense explanation of our results is as follows. In very rough terms, the NO_x reduction in NA reduces ozone elsewhere in the Northern Hemisphere by about 10% of the change in ozone in NA itself. However, there are about 500 million people in NA while there are another 5 billion people elsewhere in the Northern Hemisphere. As a result, the effects on mortality outside of the source region are comparable to those within. The high population of other Northern Hemisphere regions is unavoidably an important factor in the results; differences in baseline mortality rates are also taken into account, but population has a stronger influence on the regional results.

Referee 2 is correct to ask about the evidence of health effects in other regions of the world. As stated in the paper, the epidemiological evidence for health effects of ozone outside of the US and Europe is sparse, but while that evidence is inconclusive, it is also not inconsistent with findings in the US and Europe. Referee 2 asks in particular whether long-range influences have been observed to cause health effects, and this has clearly not been done, in part because of the difficulty of separating out the small amount of ozone that is due to long-range influences. In highly polluted regions, the influence of long-range transport is very small in comparison with that of local emissions,

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as we show in the companion to this paper. Nonetheless, the epidemiologic relationship describes the effect of changes in ozone on mortality, and some of the relevant studies suggest that this relationship holds over a wide range of concentrations (e.g., Bell and Dominici, 2006). Had we made other assumptions about the relationships between changes in ozone and mortality, such as in highly polluted regions, this would be inconsistent with the epidemiologic literature. We have expanded this discussion in the conclusions section:

"Our assessment of the effects of long-range transport on mortality is also limited by our understanding of the mortality effects of ozone, and future research should aim to improve this understanding, particularly in developing nations and at higher ozone concentrations than are commonly observed in the US and Europe."

Referee 2 asks whether it is possible to observe the effects of trends in ozone through time on the health of populations. These data are included in the multi-year epidemiologic study that we use (Bell et al., 2004) and in similar studies. One recent study has made exactly this claim for $PM_{2.5}$ (Pope, C. A., Ezzati, M., and Dockery, D. W.: Fine-particulate air pollution and life expectancy in the United States, New England Journal of Medicine, 360, 376-386, 2009), but we are unaware that any study has demonstrated this as clearly for ozone. This is a good area for further research.

9. I found Figure 2 difficult to read.

Figure 2 has been recreated so that the plot is larger, with larger text.

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