

Interactive comment on “Model calculated global, regional and megacity premature mortality due to air pollution” by J. Lelieveld et al.

Anonymous Referee #1

Received and published: 2 April 2013

This paper estimates the premature deaths associated with anthropogenic PM_{2.5} and ozone for the whole world, WHO regions, and various megacities. Since these mortality estimates are based on exposure concentrations derived from one particular atmospheric model, and the health impact function methods have been previously used elsewhere with a different atmospheric model to estimate the same thing, the novelty of this paper is limited. This is particularly the case since the new Global Burden of Disease study was published prior to submission of this manuscript. While the outdoor air pollution impacts component of the GBD study has not yet been separately published, the summary article of all risk factors including outdoor air pollution has (Lim et al. 2012), as has the paper on outdoor air pollution exposure concentrations (Brauer et al. 2012), and the results present the state of the art methods and estimates of the global mortality burden of anthropogenic air pollution. Since the innovative methods used for

C885

the GBD study have not yet been published in detail, the authors of the present study should not be held to using those methods; however, the manuscript would be much improved by clearly delineating the novelty, value added, and limitations of the present study in light of the methodological advancements since the methods and results by Cohen et al. (2005) and Anenberg et al. (2010) were published. In addition, the paper contains some questionable assumptions and lacks some details that are necessary to judge the validity of the methods.

Major comments:

- Is it useful to do country rankings as in Table 2 if only using country-specific baseline mortality rates for 36 countries? Baseline mortality rates can significantly influence results for premature air pollution related deaths, so the rankings shown in Table 2 may not be valid if based mainly on regional mortality rates. The same comment applies to the megacity results. If the authors think this is still a valid ranking, appropriate caveats should be made in the explanation.

- The authors state that the value-added of this study is the finer resolution estimates of PM_{2.5} and ozone exposure compared with Anenberg et al. (2010). However, 1.1×1.1 degrees is still very coarse for estimating PM_{2.5} exposure, and the GBD community has now produced estimates of the global burden of outdoor air pollution on mortality using much finer resolution exposure estimates published by Brauer et al. (2012). The GBD study also uses country-specific baseline mortality rates, which as previously noted, have not been used in this paper. While it is useful that the authors have presented the numbers of deaths in megacities, the methods underlying these values are not the most refined possible for any of the underlying inputs to the HIF. The authors need to better articulate the value added of this study given the methods chosen. At the very least, the authors should discuss their methods and results in the context of the new GBD study. In addition, they can specifically compare country mortality results to the estimates given on the Institute for Health Metrics and Evaluation website (<http://www.healthmetricsandevaluation.org/gbd/visualizations/country>) – while the

C886

country-specific results from the GBD study have not yet been published in a journal, they are the results underlying the Lim et al. (2012) global estimates and thus are the result of peer-reviewed methods.

- The comparison to the previous GBD study on p. 7751 needs to be updated with the current study based on Brauer et al. (2012) exposure estimates and mortality results presented by Lim et al. (2012).

- Natural aerosols may cancel out when preindustrial is subtracted from present-day, but the overall total levels of anthropogenic+natural PM2.5 affect the slope of the CRF (see Pope et al. 2009, sensitivity analysis in Anenberg et al. 2012). The implications of modeled concentration and the choice of the shape of the CRF need to be discussed throughout the paper and accounted for in the results. Consider examining the influence of the shape of the CRF in a sensitivity analysis.

- The choice to apply cardiopulmonary RR estimates only to cardiovascular disease rates is not correct. Presumably if PM2.5 is affected mostly cardiovascular disease, RR estimates for cardiovascular disease only would be significantly higher than the RR for total cardiopulmonary disease. The cardiopulmonary RR estimates are also being applied to a substantially smaller amount of baseline deaths (cardiovascular only, excluding respiratory). This is a mismatch that does not make sense, and could be a reason for the much smaller magnitude of estimated PM2.5 deaths compared with Anenberg et al. (2010). Also, are the BMR used for the population age 30+ or all population? Should be 30+ only.

- Methods section – there needs to be more information about the modeling and PM2.5 components included in the definition of PM2.5. Were dust and sea salt included? SOA? What are the sources of emissions and meteorology used to drive the model? Since part of the paper focuses on regional and urban air quality, it is important to know if regional emissions inventories are used or whether the modeling relied solely on global emissions inventories. Also, given the scarcity of monitor observations around

C887

the world (and that comparing gridcell average concentrations to point measurements is mis-matched anyway), how do the model results compare to other models for the 2005 atmosphere? US concentrations and results can be compared with Fann et al. (2011), are there regional studies for Europe and Asia to compare the results to? Should compare concentrations to the global concentrations from modeling, monitoring, and satellites by Brauer et al. (2012).

Minor comments:

- P. 7738, L22 – suggest changing “decreased” to “degraded”

- P7739, L6 – Add references to Lepeule et al. (2012)

- P7740, L 11 – Please give the year of the BMRs used for countries and regions

- P7740, L14 – Unclear which 5 types of mortality you are referring to, when next sentence says you use 3 types of mortality. Please clarify.

- P7741, L11 and L15 – Note that these RRs are for 10 ug/m3 increase in PM2.5

- P7741, L15-19 – Would be cleaner to simply report the 95% CIs next to the RR estimates given above.

- P7742, L11-15 – What are the references to the sulfate, ammonium, and nitrate comparisons, and the BC and OC comparisons for Europe? Please elaborate on the differences found for nitrate. By how much does the model overestimate BC for Europe? By how much does the model overestimate BC and OC in the US and underestimated in China? Please draw some conclusions from this model evaluation, and indicate whether the authors believe that comparing 1.1x1.1 degree model grids to point observations is a valid evaluation? What could be the implications if not? What are the implications of the over- and under-estimates that were found?

- P7743, L15-17 – Was the HIF applied in every gridcell or were the pollutant concentrations somehow aggregated up to the regions before the HIF was applied? Please

C888

clarify.

- P 7744 L14-17 – wording is awkward. Suggest replacing “demands” and changing the wording of the last phrase beginning with “thus. . .”
- P7744 L14-end – it is essential to make clear when citing numbers of deaths whether those deaths are attributable to PM2.5, ozone, both, or are just total baseline deaths in the population. It is very unclear throughout the paper.
- Table 2 – caption needs to say “attributable to PM2.5 and ozone”
- Table 2 – it seems odd that China has an order of magnitude more PM2.5 lung cancer than any other country, but only twice as many PM cardiovascular and ozone respiratory deaths. Please ensure this result is accurate and provide an explanation.
- P7750 L 9-10 – while this may be true for the health endpoints examined by this paper, children may be affected by air pollution via different health endpoints, e.g. pneumonia. The impact of including child health impacts may not be small, though one could cite Cohen et al. (2005) to give an approximate indication for the relative contribution of child vs. adult deaths due to air pollution (based on knowledge at the time).
- P 7751 L22-24 – this is an important methodological choice that should also be described in the Methods section.
- P7752 L1-2 – Anenberg et al. (2010) also used country-specific baseline mortality rates, and for more countries than here. The results were then aggregated to the regional level, but the underlying calculations were made at the gridcell level with country-specific BMRs applied.
- P7751 comparison with Anenberg et al. (2010) – it is very difficult to do this without a table of the preindustrial and present-day concentrations by region for this paper. It is stated in the discussion comparing results to the earlier GBD study that preindustrial PM2.5 concentrations are not much lower than 7.5 ug/m3. Preindustrial PM2.5 concentrations in Anenberg et al. (2010) were close to 0. It could be that a major

C889

difference is the concentration change itself. More information on concentration levels should be presented. As previously stated, another major difference is the application of cardiopulmonary RR estimates to cardiovascular BMRs, which could substantially underestimate PM2.5-related deaths.

References

- Anenberg SC et al 2012 Global air quality and health co-benefits of mitigating near-term climate change through methane and black carbon emission controls *Environ. Health Perspect.* 120 831-839.
- Brauer et al 2012 Exposure assessment for estimation of the global burden of disease attributable to outdoor air pollution *Environ. Sci. Technol.* 46 652-660.
- Fann N, Lamson A, Anenberg SC, Wesson K, Risley D, Hubbell BJ 2011 Estimating the national public health burden associated with exposure to ambient PM2.5 and ozone *Risk Analysis* doi: 10.1111/j.1539-6924.2011.01630.x.
- Lepeule J, Laden F, Dockery D, Schwartz J 2012 Chronic exposure to fine particles and mortality: An extended follow-up of the Harvard Six Cities Study from 1974-2009 *Environ. Health Perspect.* 120 965-970.
- Lim SS et al 2012 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.
- Pope CA III, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, Thun MJ 2009 Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: Shape of the exposure-response relationship *Circulation* 120 941-948.

Interactive comment on *Atmos. Chem. Phys. Discuss.*, 13, 7737, 2013.

C890