

**Comments on
Quantification of the Health and Economic Impacts of Air Pollution from
Port-related Goods Movement and Port Activities in California
(Appendix A of the Dec 1, 2005 Draft Emission Reduction Plan for Ports
and International Goods Movement)**

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In my initial review, it seems that you did not use our recent ACS study from LA. Given that 70% of the deaths come from the South Coast Basin, I recommend that you conduct and report this estimates from the LA study as another credible (and probably more relevant) risk estimate for the California population. There could be two specific analyses:

One applying the estimate only to the South Coast and then blending in the higher total from that region with the rest of the state estimated from Pope et al. 2002; and Another applying the LA estimates to the entire state.

Just to clarify what seems to be a misconception in the appendix document, the main estimates presented in the LA paper use EXACTLY the same model as Pope et al. 2002. These estimates are fit with a standard Cox regression model that controls for 44 individual covariates and stratifies for age, sex, race in the baseline. Thus if you want to use the estimates that are the same as the Pope study, then these are available. We intentionally used the same model so such comparisons (and risk estimates) would be available to policymakers for burden assessments and others interested in understanding why the risks in LA were higher.

All of the ecologic confounders and spatial models drive down the estimates or widen the confidence intervals, but they are still about twice as large as the estimates presented in Pope. If you choose to run the sensitivity models using the LA estimates suggested above, I would first use the same ones as Pope without the spatial adjustments. You could if you wanted also report the lower bound with maximal control for neighborhood confounders, but to do this correctly, you would need to account for the spatial variation in the ecologic confounders for the current population in California (which could be quite a chore). But you could report the lower estimate without the more complex analysis as another sensitivity test to supply a lower bound.

The argument currently in the document for not including the LA estimates could be criticized as logically inconsistent. If you did not use the LA estimate because it does not apply to the entire state, then why would another estimate from Pope et al. which includes 116 cities (many of which are very different in pollution mixture and population characteristics than CA)? In fact, if you were trying to match the analysis on the factors that can bias the risk estimates, then the LA

study is arguably more even more relevant as the main estimate by almost all the criteria that matter: (a) the pollution mixture in LA is closer to the pollution mixture across all of CA than the mixture in the 116 cities in Pope et al. which is dominated by sulfate contrasts in the in the lower great lakes; (b) the underlying population characteristics are much closer in the LA study than again in the 116 cities; (c) the relative weight in the model given to CA in the Pope study is less than 10% of the total ACS population in the ACS study (that's my recollection, I'll get you exact numbers soon), while the LA study is 100% based on CA populations; and (d) the spatial resolution of your exposure assignment is if I understand it correctly more of an within-city assessment than a between-city contrast, so again the LA study is a closer match to the health risk assessment. On this last point, I have not reviewed the document in detail, but am relying on your earlier protocol and Arthur Winer's nice description in one of our meetings to discuss the protocol. For all of these reasons, conducting sensitivity analyses on the likely mortality reductions from the LA study estimates is important to the credibility and logical consistency of your chosen dose-response functions and the entire analysis.

Other Comments:

There is a potential problem with the narrow definition of port and goods movement activities. These activities have ramifications that go beyond the immediate trains, trucks, and ships, which are the focus of your study. There are many automobile trips from workers traveling to and from their jobs which need to be taken into account. A more thorough and complete way to understand these impacts would be through an econometric computable general equilibrium model or at least an input-output model. This would give you some idea of the secondary and tertiary ramifications of goods movement. I'm certain that the Finance Department (or equivalent) would have calibrated such a model already, and if they have not, Dr. Sergio Rey of San Diego State University has one that I've used in similar research with him some time ago. I have co-authored a number of papers using the I-O and CGE approach and for the longer term methods development, it would be a good idea to expand this definition. What about the impacts of airports? These are increasingly seen as a major source of pollution. These do not seem to be in the goods movement definition and they should be as far as I can tell. There are a number of estimates that implicate NO₂ as a potential source of health effects. Whether NO₂ is the putative agent, interacts with other pollutants, or serves as good indicator of mobile source pollution is an open question, but I feel that the estimates of NO₂ mortality could be added as a sensitivity analysis (although this raises the issue of overlap with the PM effects). The study by Nafstad et al. (2004) supplies mortality estimates for a Norway, and it would be worth investigating what inclusion of NO₂ does to your estimates. Or you could use recent studies by Burnett et al. for time series estimates (again a sensitivity analysis) The comment that there "strong" associations between air pollution and health may be an overstatement. Strength of association in epidemiology relates to dose-response coefficient size. When

the size is only a 1% increase for time series mortality estimates over a 10 ug/m³ contrast, it is difficult to call this "strong". Even the 6% increase in Pope et al. is not that large an effect (say compared to smoking or ETS for example). The estimates are more properly called "consistent" between places and biologically plausible in the Hill terminology of causation. The key point is that even when the relative risks are small, they affect large populations and as a result have the potential to have sizable impacts on mortality and morbidity. Rose has a famous paper that discusses this point.

There are a number of other papers that should be cited supporting the health effects of living near roads: Hoek et al. 2002 (Lancet); Finkelstein et al. 2004 (AJE); Nafstad et al. 2004(EHP)). All of these deal with mortality and therefore are very relevant to your assessment.

Table A4 should include ischemic heart disease as a separate category for premature death. It is associated with air pollution more strongly than CPD, and in general, respiratory deaths are not usually elevated (6 cities study, my studies with Finkelstein in Hamilton and the ACS study all show this). For ozone, there is a more tenuous relationship, at least to mortality. The ACS studies do not find a significant association. I will read more on this, but my initial reaction is that you could again be seen as inconsistent. If you are going to use time series estimates for ozone mortality (which are smaller) and then chronic estimates for PM (bigger), someone could ask, why have you not used time series for mortality, which would dramatically reduce your estimates. But if you use chronic estimates for ozone, they are not significant. You need to be consistent or it will look like you are just grabbing whatever seems largest (and I know from all the hard work and thoughtful discussion in the document that is not the intent). I can say that our new ACS analysis, which is under preparation, does indicate an ozone effect on all-cause mortality for the national level study, but that is not going to be out for some time.