



# *memorandum*

**ENTA**

**Abt Associates Inc.**

**Date** December 12, 2005

**To** Bart Croes

**cc** Hien Tran, Linda Tombras Smith, Richard Bode

**From** Aaron Hallberg

**Subject** Goods Movement Risk Assessment Peer Review

All,

It certainly appears that you have done a tremendous amount of work in a very short period of time. In general the approach seems sound, and many areas of the assessment have been explained quite nicely and with an adequate discussion of the uncertainties involved.

My main criticism is simply that a basic explanation of the methodology of the assessment should be included somewhere near the beginning of the appendix. After reading it through my impression is that the methodology involves calculating health impacts for various pollutants in the year 2000, generating impact-per-ton-emitted estimates, and then using these to project impacts in 2005 and various future years based on emissions projections. In order to figure this out, however, I had to get all the way to pages A-41 (for PM) and A-43 (for Ozone).

I also have a number of specific comments on aspects of the methodology and on the appendix itself:

- (1) It seems to me that one of the major sources of uncertainty with the assessment is the assumption that there is a direct correspondence between emissions and exposure. That is, actual estimates of exposure are only generated for the year 2000, and are used just to generate impacts per ton of emissions. All of the health impact numbers presented are then based on the assumption that a linear relationship holds between emissions and health impacts (with a correction for population growth), which is certainly not the case. Given that you did not have the time or resources to use air quality modeling, this may well be the best approach available to you, but you should still discuss the uncertainties involved.
- (2) It appears that Ozone impacts were calculated just for ozone levels above the standard, while PM impacts were calculated all the way down to zero. Is this correct? If so, it seems strange to me and should probably receive some discussion. At the very least, it should be mentioned that the disproportionate impact of PM in the appendix is partially (largely?) an artifact of this distinction between the two pollutants. That is, if ozone impacts were calculated down to background they would be much higher.

- (3) There are two major issues regarding demographic projections:
- a. The composition of California's population is going to change quite a bit between 2005 and 2030, but it appears that only a simple total population ratio was used to project impacts in future years. In general, I would expect this to lead to an underestimate of various health impacts, and especially of premature mortality.
  - b. Baseline incidence rates will also change between 2005 and 2030, but it appears that they are assumed to be constant. In particular, baseline mortality rates should decline, perhaps substantially, leading to an overestimate of premature mortality.
  - c. Note that these two issues may more or less cancel each other out, but it is unclear what the overall impact might be. You may want to try to account for these changes, or at the very least you may want to discuss them as a potential source of uncertainty.
- (4) Many health endpoints typically included in US EPA analyses were left out of the assessment, including Infant Mortality, Chronic Bronchitis, Acute Myocardial Infarction, Cardiovascular Hospital Admissions, Acute Bronchitis, Lower and Upper Respiratory Symptoms. The exclusion of chronic bronchitis is especially troublesome, as it is typically the second highest component of the economic valuation (after premature mortality). Additionally, the chronic bronchitis study typically used by US EPA (Abbey et al, 1995) was actually done in California. See, for example, the Clean Air Interstate Rule regulatory impact analysis at <http://www.epa.gov/interstateairquality/pdfs/finaltech08.pdf>.
- (5) The low and high estimates of economic impacts are presented as 5<sup>th</sup> and 95<sup>th</sup> percentiles of "...an integrated analysis of uncertainties in human health concentration-response functions and the economic values..." (pg. A-25). The method of generating these values, however, seems incorrect to me. If I am reading it correctly (pg. A-48), you are simply multiplying the 5<sup>th</sup> percentile estimate of cases by the 5<sup>th</sup> percentile of the unit value distribution to generate the 5<sup>th</sup> percentile of the combined distribution. The typical approach for this process, assuming that the unit value distribution and the distribution of the C-R function coefficient are independent (and I see no reason not to make this assumption), would be to run a Monte Carlo simulation to generate the composite distribution and then pull the 5<sup>th</sup> percentile, the mean, and the 95<sup>th</sup> percentile from this composite. If the two distributions are not skewed, the mean value from this procedure should be very close to the product of the two means. The 5<sup>th</sup> and 9<sup>th</sup> percentiles, however, will typically be quite different from the simple product of the 5<sup>th</sup> and 95<sup>th</sup> percentiles.
- (6) You might consider updating your premature mortality unit value – EPA has more recently used (see the Clean Air Interstate Rule regulatory impact analysis referenced above) a normal distribution with mean of \$5.5 million (1999 dollars) and 95% confidence interval of \$1.0 million to \$10 million. Obviously, the choice of a premature mortality unit value will largely drive your overall economic impacts, so it is quite important.
- (7) I found the presentation of future economic benefits on page A-6 quite confusing at first – you present a single central estimate of premature mortality, but two central estimates (or a central estimate range) for economic benefits. Later I realized that these two estimates were generated using different discount rates, but this is not explained until page A-56. Indeed, table A-12 is similarly confusing. I would recommend explicitly

presenting these two sets of numbers as different central estimates, rather than as a range. I would also present the confidence intervals separately – it is still not clear to me what these represent.

- a. Does the 5<sup>th</sup> percentile come from the (lower) 7% discount rate and the 95<sup>th</sup> from the (higher) 3%? In this case, in what sense do these numbers represent a 95% confidence interval?
- b. Also – I think you are really presenting a 90% confidence interval (5<sup>th</sup> – 95<sup>th</sup>), not a 95% confidence interval (2.5<sup>th</sup> – 97.5<sup>th</sup>). This should probably get updated throughout.

(8) On page A-82, you say:

“The current methodology used a power of 2.5 in order to optimize the interpolations... Further, the current methodology uses a minimum of 10 monitoring stations and up to a total of 15 in weighting the results to estimate the concentration at each census tract.”

There are two issues here, which it probably makes sense to discuss in the appendix. The first is in what sense using a power of 2.5 (in the inverse distance weighting) “optimizes” the interpolations. I can see how it gives greater weight to nearby monitors, but was some test run which determined that this was the “best” value in some sense, or is it simply based on intuitions about nearby monitors giving more accurate results? Secondly, using the old algorithm for selecting monitors to use in the interpolation process was quite straightforward – everything within 50 km was used. The new procedure is not similarly straightforward – how are the 10 to 15 monitors selected? Are they simply the closest monitors, within some radius? Or are they the monitors that “surround” the interpolation site in some sense? When are 10 used and when 15?

- (9) On page A-90 you mention that annual concentration statistics are reported as geometric means. Are these geometric means used in combination with C-R functions to generate health impacts, or are they strictly for reporting? I would not recommend using them in C-R functions unless the coefficients in these functions were developed using geometric means.
- (10) In response to my previous comment on interpolation to the census tract, you mentioned that “...population-weighted exposures were developed at the county and basin level, consistent with the higher levels of spatial aggregation used in the epidemiologic studies.” The issue, however, is that the population-weighted exposure estimates are based on census-tract population levels and census-tract pollutant concentrations (at least on my reading of the appendix – if this is not the case, you might want to re-write this section). Again, this assumes that people are exposed to ambient pollution according to where they live, and is not consistent with the spatial scales typically used in epidemiologic studies. This is probably not a huge issue, but it seems like you are doing a whole lot of work to generate numbers which are not likely to be any more accurate than a simple county- or basin-wide average.

Again, overall I think you have done a nice job here with the time and resources at your disposal.

Best regards,

Aaron Hallberg