1. Introduction

Ames et al. raise a number of concerns about our recent modeling exercise focused on particulate matter impacts of nine power plants in Illinois. Before responding to their primary critiques, we first wish to provide the context of our analysis and the Cambridge Environmental response, to help frame our discussion. We have conducted two recently published analyses of power plant concentration and health impacts (Levy and Spengler, 2002; Levy et al., 2002), meant to provide estimates that could inform ongoing discussions about power plant regulation in the US. In both of the cases, Cambridge Environmental has been hired by the owners of the power plants (Midwest Generation for this article) to raise questions about the validity of the work in a policy context. Thus, the question before us is not simply an academic question of ideal model specifications, but is whether the uncertainty in our work is sufficient to invalidate its use in estimating population risks from power plants. We address two separate questions in our response—the specific critiques raised by Ames et al. and the importance of these critiques in a policy context.

2. Near-field dispersion and mixing height

The first critique states that our selection of a 45 km radius smoothing window (MNMDAV = 3) in a lakefront region could lead to substantial errors in near-source impacts related to underestimated mixing heights over Chicago. We agree with Ames et al. that the MNMDEV setting could influence the mixing heights of near-shore cells, but argue that this effect is quite small and that mixing heights are more likely overestimated than underestimated in our CALMET modeling.

For example, Fig. 1 shows mixing height contours from CALMET for 3 July 1999 (20Z). This figure shows a sharp gradient in mixing height over the Chicago area, with much lower mixing heights over Lake Michigan. While this gradient is not as sharp as one would find with MNMDAV set to 1, we argue that the mixing heights over Chicago may still be too high, as a result of the techniques used in CALMET to estimate mixing height.

In particular, CALMET does not account for the limiting influence of the lifting condensation level (LCL). In the convective Planetary Boundary Layer (PBL), the LCL provides a rough estimate of the limit of PBL growth. The smaller (horizontal) numbers in Fig. 1 show the LCL in meters based on observed temperature and dewpoint measurements, demonstrating regional mixing height values much lower than our estimated CALMET values. Even if the observed temperatures and dewpoints were too close to the ground and LCL numbers are underestimated, they do not support widespread mixing heights of 1200–1600 m. Thus, it appears unlikely that our modeling process has led to substantial underestimates of mixing height.

In addition, Ames et al. argue specifically that the distance to the maximum ground level concentration (MGLC) of 1–2 km for plants in Chicago is demonstrative of meteorological errors. We disagree. In previous studies of lake breezes on the shores of Lake Michigan (Lyons, 1972, 1975; Lyons and Cole, 1973), lake breeze events were found to occur about 36% of the time from May–August, with minimal occurrence in other seasons due to the requisite temperature gradient between land and water, weak gradient winds, and strong insolation. During our study period, winds from the north-north-east to east occurred about 16% of the time, agreeing with previous estimates. These studies found that power plant plumes along the Chicago shore could be brought...
down to the ground rapidly during strong insolation days, with fumigation within 2–3 km of the shoreline (Lyons and Cole, 1973). Estimated distances to the MGLC varied from 1.8 to 15 km for a similar scenario (Lyons, 1975). Thus, annual average maximum concentrations could, in fact, occur within a few kilometers of plants at the land/water interface, driven by these lake breeze episodes.

Finally, we return to a policy context and ask whether these uncertainties could significantly influence population risks, using some simple bounding calculations. As stated in our article, our central estimate for mortality from the nine power plants combined is 320 deaths/year. Since distance to MGLC for secondary pollutants is relatively invariant across plants and sulfates and nitrates require some time for formation, we first assume that the Ames et al. critique largely relates to primary PM. If we remove all the primary PM effects at all distances from Fisk and Crawford, this would reduce our total mortality estimate to 310 deaths/year.

To further illustrate how the Ames et al. argument about MGLC is inconsequential for risk-based policy decisions, we may assume that all concentration impacts (primary and secondary) for all nine power plants are overestimated by a factor of five at all points within 50 km. Given this extreme assumption, our central impact estimate would be reduced to 270 deaths/year. Simply put, total population exposure to particulate matter from power plants is largely a function of long-range transport of secondarily formed particles, and it is highly unlikely that the modeling concerns raised by Ames et al. would have any effect on risk-based policy decisions. Any reasonable decision maker would consider all of these values to represent “approximately 300 deaths”. A far more significant uncertainty is related to our omission of impacts beyond 500 km from the source.

3. Grid-cell resolution and lake/land breezes

The primary critique raised by Ames et al. in this section is that we used only one surface meteorological station in our modeling domain. This is false and is either a misrepresentation or misunderstanding of our modeling approach. As stated in our article (Levy et al., 2002, p. 1066), “…an ADAS analysis was performed using the RUC analysis for a first-guess field and combining it with the METAR surface observations.”
4. Health effects

Regarding the health evidence, we explicitly stated that discussion of the magnitude of the effect and associated uncertainties were beyond the scope of our paper. However, Ames and colleagues focus on this evidence for the purpose of invalidating our analysis, necessitating a brief discussion here.

Ames et al. argue quite vehemently that the scientific community has a good idea that secondary sulfates and nitrates are not toxic, citing their own review manuscript (in press) and a discussion document from the Netherlands (which is stated to be for external review purposes and not to be cited). They ignore the bulk of the primary health literature. There are three major epidemiological studies considering long-term exposures to sulfate particles (Dockery et al., 1993; Pope et al., 1995; McDonnell et al., 2000). The first two studies found significant associations with sulfate concentrations, relationships confirmed by an independent reanalysis of both studies (Krewski et al., 2000) and a recent follow-up investigation (Pope et al., 2002). The third study did not yield statistical significance, but had a central estimate for male nonsmokers comparable to the central estimates from the other two studies. Numerous studies of short-term exposures to sulfate particles (e.g., Burnett et al., 1998; Fairley, 1999; Gwynn et al., 2000; Mar et al., 2000) have shown significant associations with premature death as well. “Negative” epidemiological evidence has tended to come from studies with insufficient statistical power to detect effects were they to exist (e.g., Klemm and Mason, 2000).

Although meaningful toxicological evidence (using susceptible animals and realistic exposures) is more difficult to come by, some recent laboratory studies provide supporting evidence of potential health effects of sulfate particles. For example, Kleinman et al. (1999) found that an inflammatory response in rats after 5 days of exposure was associated with ozone plus sulfuric acid-coated particles, but not with ozone alone. The same research team found that ammonium bisulfate exposure in older rats led to increased labeling of lung cells, with the most significant effects due to combined ammonium bisulfate, elemental carbon, and ozone exposure (Kleinman et al., 2000). A study in dogs used factor analysis to conclude that sulfur-related exposure was associated with significant decreases in red blood cell counts and hemoglobin concentrations, consistent with potential cardiovascular health effects (Clarke et al., 2000). There is generally a dearth of evidence on nitrate toxicity (positive or negative), although the few published epidemiological studies that measured it have shown significant associations (Fairley, 1999; Hoek et al., 2000), with the exception having minimal statistical power (Klemm and Mason, 2000). While the health literature is clearly broader, more equivocal, and more complex than we can describe in this response, it is clear that Ames et al. vastly misrepresent current scientific understanding.

More generally, our analysis aims to “determine the influence of key atmospheric modeling assumptions on health-based conclusions” (Levy et al., 2002). As such, it is entirely appropriate to use as a reasonable central estimate the default approach used by all past and ongoing analyses in this area (e.g., European Commission, 1995; US EPA, 1999). While future studies may find that sulfates and nitrates are more or less toxic than “average” ambient particles, it is not sensible to argue that the current best estimate is zero toxicity.

5. Conclusions

We thank Ames and colleagues for their careful review of all of our modeling files and are encouraged that their only critiques represent relatively minor influences on health risks. We provided draft manuscripts and input files to all stakeholders to ensure that our analysis was transparent and interpretable, and feedback from both industry and community groups has proven valuable.

We would advise that future critiques of atmospheric modeling for health impact assessment adopt the approach in our article of quantifying the effects of model assumptions rather than asserting that deviations from reality invalidate the findings. We agree with Ames et al. that more validation is required for CALPUFF and other long-range transport models, but urge researchers to focus on health-relevant calculations to help determine which assumptions are important and unimportant for pending policy decisions.

We conclude by arguing that having a standard of perfection from models implies that pollution control policies would be delayed indefinitely awaiting computational refinements (“paralysis by analysis”), and this would be expected to lead to substantial public health impacts in the interim. In this case, we have demonstrated that the issues raised by Ames et al. do not
materially influence the conclusions of our analysis, and we strongly disagree with the contention that our model is invalid for public policy applications.

References


