June 20, 2002

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Dear Eric:

Following is a report that provides a detailed and quantitative examination of the models and assumptions used in the Abt Associates study titled "Particulate-Related Health Impacts of Eight Electric Utility Systems". This analysis represents a follow-up of my initial letter dated May 20, 2002.

The key conclusions from my analysis are as follows:

- The atmospheric model applied appropriately accounts for critical atmospheric factors and provides health impact estimates quite similar to estimates from other models.
- The health evidence considered includes the major published studies on particulate matter health effects. The concentration-response function for mortality is a reasonable interpretation of the current literature, and the evidence cited for other health outcomes is representative of the literature as a whole.

Based on the calculations provided, I conclude that the health estimates in the Abt Associates study are well supported by the published literature and represent reasonable central estimates. Substantial bias related to the atmospheric modeling is extremely unlikely, and bias related to the health evidence (in either direction) is only plausible only under extreme interpretations of the literature. I therefore conclude that the Abt Associates study provides useful information from which policy conclusions can be drawn, and information as provided in the attached document can be used to provide plausible upper and lower bounds for the public health impacts of selected power plants.

Jonathan Levy

Evaluation of Methodology in "Particulate-Related Health Impacts of Eight Electric Utility Systems"

Executive Summary

In April 2002, Abt Associates prepared a report titled "Particulate-Related Health Impacts of Eight Electric Utility Systems". In this report, the authors focused on quantifying the health impacts of particulate air pollution from power plants in eight electric utility systems (AEP, Cinergy, Duke, Dynergy, First Energy, SIGECO, Southern, and TVA). They concluded that this subset of power plants could be associated with 5,900 premature deaths per year, 140,000 asthma attacks per year, and 6,000,000 minor restricted activity days per year, among other health outcomes. These estimates were made by forecasting NOx and SO_2 emissions at the selected power plants in 2007, using a relatively simple atmospheric dispersion model to estimate the resulting particulate matter impacts across the US, and using current health evidence to quantify the health impacts from this incremental contribution to particulate matter levels.

In any analysis of this sort, there are numerous uncertainties, many of which are impossible to eliminate. The important question for policy analysis is whether the estimates in this report are significantly biased (in either direction), how large the uncertainties appear to be, and what the major contributors to uncertainty are.

In this review, I focus on the questions of bias related to the atmospheric dispersion model and the use of health evidence. Through detailed comparisons with other similar studies using more complex atmospheric models, I conclude that the atmospheric dispersion model in the Abt Associates report does not appear to be significantly biased. Nitrate impacts are likely more uncertain than sulfate impacts, because of issues related to the atmospheric chemistry, but

this would have a relatively small impact on the total health impacts due to the far greater contribution of sulfates.

Similarly, the decisions regarding relative toxicity of particle types and the choice of studies made by the authors appear appropriate and provide reasonable best estimates of health impacts. Although alternative interpretations of the health literature are available and could lead to significantly different estimates, the concentration-response functions selected by Abt Associates are bounded by estimates available elsewhere and are reflective of current scientific knowledge. The most significant uncertainty is related to the interpretation of cohort mortality studies, but given currently available information, it would be inappropriate to exclude this effect entirely from a comprehensive analysis.

The estimates provided in the text of my review can help decision makers place upper and lower bounds on the potential magnitude of the health effects and determine in which areas further research might help inform policy decisions. In conclusion, supported by a detailed uncertainty analysis, the estimates from the Abt Associates report provide a plausible basis for near-term policy decisions.

Evaluation of Dispersion Model

For the atmospheric modeling, it is important to evaluate the potential biases or uncertainties in the model from a health-relevant perspective. In other words, it would be possible for S-R matrix to display different geographic patterns than other models, but result in similar health impact estimates. Since the Abt Associates report focuses largely on national health impacts, I evaluate S-R matrix from a national perspective as well. Some of the discussion below, including the comparison for power plants in Georgia, is based on the analysis in a manuscript in preparation (*1*).

To compare the results from different atmospheric models in a risk assessment and to allow for model results to be extrapolated to other settings, analysts have developed the concept of the intake fraction (*2*). An intake fraction can be defined simply as the fraction of a pollutant or its precursor emitted that is eventually inhaled or ingested by someone, somewhere. Mathematically, it is defined as:

$$iF = \frac{BR \times \sum_i C_i \times N_i}{Q}$$

where iF = intake fraction; BR = population-average breathing rate (assumed to be 20 m^3/day); C_i = incremental concentration of pollutant at receptor i ($\mu g/m^3$); N_i = number of people at receptor i; Q = emission rate of pollutant or pollutant precursor (μg/day).

Therefore, an intake fraction is a unitless measure that depends on how a pollutant emitted influences ambient concentrations, and on how many people are affected by those concentrations. If the health effects of the pollutant have a linear concentration-response function

with no dose rate dependence, this figure will be directly proportional to health impacts. In other words, if an intake fraction from Source A is double the intake fraction from Source B, then if the sources have the same emission rate, the health impacts from Source A will be double those of Source B. Since the Abt Associates report assumes linearity in concentration-response functions, the intake fraction is an appropriate figure to estimate.

For this case, we are exclusively interested in intake fractions related to particulate matter formation due to SO_2 and NOx emissions, as the Abt Associates report focused on health effects from particulate matter. Clearly, SO_2 emissions lead to the formation of ammonium sulfate particles and NOx emissions lead to the formation of ammonium nitrate particles. However, there is an additional intake fraction we must consider. It is a well-established fact that, under some conditions, changes in SO_2 emissions can influence particle nitrate concentrations. Because ammonium preferentially reacts with sulfate over nitrate, decreases in sulfate concentrations can potentially free up ammonium to react with nitrate. Thus, throughout this text, I will be considering three different intake fractions:

- Sulfate/SO_2: Incremental amount of sulfate inhaled per incremental unit of SO_2 emissions
- Nitrate/NOx: Incremental amount of nitrate inhaled per incremental unit of NOx emissions
- Nitrate/SO_2: Incremental amount of nitrate inhaled per incremental unit of SO_2 emissions

Given these definitions, the question is: Are the intake fractions implied by the S-R matrix analysis similar to those from other modeling studies? We address this question by making two major comparisons:

1. S-R matrix versus CALPUFF for 40 power plants randomly selected across the US (7 of which are in the Abt Associates report)
2. S-R matrix versus CALPUFF for seven power plants in Georgia (all included in the Abt Associates report)

For the first comparison, we are comparing the findings from Wolff (*3*) with findings from the S-R matrix, which was provided to us by Abt Associates. Wolff used CALPUFF to model the intake fractions for primary PM, sulfates, and nitrates for 40 power plants randomly selected across the US. CALPUFF is a transport and dispersion model that models emissions as a sequence of discrete puffs and simulates both dispersion and chemical transformation (*4*). It is generally applied to a small set of sources with limited background pollution data. For these and other reasons, CALPUFF has a somewhat different methodological framework than S-R matrix.

In Wolff (*3*), CALPUFF was used to estimate the incremental concentrations for each source at each of 448 receptor points, spaced every 100 km over a region 1600 km by 2800 km. To estimate intake fractions, Wolff used 1990 meteorological and population data, as taken from ArcView version 3.2. Of note, this implies that the intake fractions estimated by Wolff would be expected to be slightly lower than the intake fractions implied by S-R matrix, which use 2007 population data. The ratio between 2007 and 1990 US populations is roughly 1.17. In addition, CALPUFF default values of parameters such as particle size distribution (mass median diameter = 0.5 μm, geometric standard deviation = 2), background ozone (80 ppb) and ammonia concentrations (10 ppb) were used, as was the MESOPUFF chemical conversion methodology.

For these 40 power plants, Wolff reported mean intake fractions of 2×10^{-7} for sulfate/SO_2 and 3×10^{-8} for nitrate/NOx. This means that for every 10 million grams of SO_2 emitted by power plants, 2 grams of sulfate are inhaled by someone in the US. This is perhaps unintuitive until we make a naïve "back of the envelope" calculation based on an earlier Abt Associates study (*5*). They modeled the benefits of power plant emission controls across the US. Their "Policy Case" resulted in a 7.1 million ton reduction in annual SO_2 emissions. 7.1 million tons per year is equal to about 2×10^{16} µg/day. We can estimate that the average ambient reduction of sulfate from this was roughly 1 µg/m^3 (looking at Exhibit 3.2 in their report). Using our above equation,

$$iF = (20\ m^3/day) * (1\ \mu g/m^3) * (290\ million\ people) / (2 \times 10^{16}\ \mu g/day) = 3 \times 10^{-7}$$

So, this simple calculation demonstrates that the magnitude of the figures is reasonable.

As discussed in Evans et al. (*6*), the nitrate/NOx intake fractions may be underestimated, as Wolff chose to divide all CALPUFF-modeled values by four to reflect known relationships between particle nitrate formation and temperature. Thus, a value of 1×10^{-7} for nitrate/NOx may be more appropriate, with the true value implied by the Wolff analysis likely falling between 3×10^{-8} and 1×10^{-7}. Because of the methodology used by CALPUFF, no nitrate/SO_2 intake fractions were estimated.

In contrast, when we apply S-R matrix to the same 40 power plants, we find mean intake fractions of 3×10^{-7} for sulfate/SO_2, 5×10^{-8} for nitrate/NOx, and -5×10^{-8} for nitrate/SO_2. In other words, S-R matrix would yield an impact due to sulfate particles approximately a factor of two higher than reported in Wolff. However, this difference is tempered somewhat by the

reduced nitrate concentrations per unit SO_2 emissions and by the higher population used by S-R matrix. When looking at NOx emissions, the S-R matrix intake fraction is between the two potential values from Wolff. Thus, there does not appear to be substantial bias in either direction, although S-R matrix yields slightly higher estimates for particle formation due to SO_2 emissions.

However, we note that the power plants in the eight electric utility systems considered in the Abt Associates report are predominantly found in the Midwest and Southeast. Because sulfate and nitrate formation patterns depend on weather patterns, it would be expected that the relationships between the models would differ across regions. Thus, our US-wide comparison for the 40 power plants may not be directly applicable to the Abt Associates analysis.

We can make a more reliable comparison for the purpose of evaluating the Abt Associates report by focusing on the seven power plants modeled in Wolff that were also modeled in the Abt Associates report. The sulfate/SO_2 and nitrate/NOx intake fractions for those seven plants are given in the table on the following page. Nitrate/SO_2 is not presented, as all values are zero in Wolff. All nitrate/NOx values are presented as reported in Wolff (*3*), with the ratios in the table reflecting both interpretations of the Wolff findings.

Plant	Sulfate/SO_2, CALPUFF	Sulfate/SO_2, S-R matrix	Ratio (S-R/CALPUFF)	Nitrate/NOx, CALPUFF	Nitrate/NOx, S-R matrix	Ratio (S-R/CALPUFF)
W H Sammis	1.6×10^{-7}	3.4×10^{-7}	2.1	2.2×10^{-8}	2.5×10^{-8}	1.1/0.3
Gorgas	1.3×10^{-7}	2.9×10^{-7}	2.2	1.6×10^{-8}	4.3×10^{-8}	2.7/0.7
Scherer	1.3×10^{-7}	3.0×10^{-7}	2.3	1.5×10^{-8}	3.9×10^{-8}	2.6/0.6
Gallatin	2.0×10^{-7}	3.5×10^{-7}	1.8	2.5×10^{-8}	4.9×10^{-8}	2.0/0.5
Cardinal	1.6×10^{-7}	3.4×10^{-7}	2.1	2.1×10^{-8}	2.5×10^{-8}	1.2/0.3
Conesville	1.7×10^{-7}	3.7×10^{-7}	2.2	2.3×10^{-8}	3.0×10^{-8}	1.1/0.3
Widows Creek	1.8×10^{-7}	3.5×10^{-7}	1.9	2.2×10^{-8}	4.6×10^{-8}	2.1/0.5

This table corroborates the general findings from the 40 plant comparison. S-R matrix yields sulfate/SO_2 intake fractions that are approximately a factor of two higher than those reported by Wolff. If we adjust the sulfate/SO_2 values for the higher population and reduced nitrate in S-R matrix, the S-R/CALPUFF ratio falls from 1.8-2.3 to 1.3-1.7. For nitrate/NOx, the two interpretations of the Wolff findings bound the S-R matrix estimates in all cases. There are some distinct geographic patterns, with values relatively lower for S-R matrix for the three plants in Ohio, versus the plants in Alabama, Georgia, and Tennessee.

Now, the critical question is: Do our findings mean that S-R matrix has overestimated sulfate formation, that Wolff's CALPUFF analysis has underestimated sulfate formation, or does the truth lie somewhere in between? Although the difference between the models is only a factor of 1.5 (a difference unlikely to lead to radically different policy decisions), understanding this question will help determine if any systematic bias exists in the Abt Associates report. Because

of the numerous differences in model assumptions, it is difficult to draw direct conclusions from the above values. The findings would be somewhat more conclusive if the two models were constructed with as many identical assumptions as possible.

In an ongoing analysis (*1*), we have modeled sulfate and nitrate impacts of seven power plants in Georgia, using both CALPUFF and S-R matrix with essentially identical model assumptions wherever possible (e.g., identical population patterns, same meteorological year, similar background pollution levels). The comparison in the following table is based on a domain within 500 km of Atlanta, making direct comparison with the values reported earlier inappropriate. Note that the nitrate/NOx intake fractions from CALPUFF do not contain the Wolff correction factor.

Plant	Sulfate/SO_2, CALPUFF	Sulfate/SO_2, S-R matrix	Ratio (S-R/CALPUFF)	Nitrate/NOx, CALPUFF	Nitrate/NOx, S-R matrix	Ratio (S-R/CALPUFF)
Bowen	1.6×10^{-7}	1.7×10^{-7}	1.1	6.7×10^{-8}	2.7×10^{-8}	0.4
Hammond	1.6×10^{-7}	1.7×10^{-7}	1.1	7.1×10^{-8}	2.7×10^{-8}	0.4
Harllee Branch	1.5×10^{-7}	1.6×10^{-7}	1.1	5.9×10^{-8}	2.3×10^{-8}	0.4
Jack McDonough	1.7×10^{-7}	1.7×10^{-7}	1.0	7.0×10^{-8}	2.6×10^{-8}	0.4
Scherer	1.5×10^{-7}	1.6×10^{-7}	1.0	5.9×10^{-8}	2.2×10^{-8}	0.4
Wansley	1.5×10^{-7}	1.8×10^{-7}	1.2	6.0×10^{-8}	2.7×10^{-8}	0.4
Yates	1.6×10^{-7}	1.8×10^{-7}	1.1	6.9×10^{-8}	2.7×10^{-8}	0.4

For sulfate/SO_2, the results are essentially identical, and are even closer when we incorporate the negative impact on nitrate formation due to SO_2 emissions in S-R matrix. This implies that the differences between S-R matrix and CALPUFF as implemented in Wolff (*3*) could be due to differences in how Wolff and Abt Associates set up their respective models rather than systematic bias related to the models themselves.

For nitrate/NOx, S-R matrix appears to systematically underestimate impacts, although we note that application of the Wolff correction factor would result in CALPUFF estimates that bound the S-R matrix estimates as above.

To understand the relative importance of these figures, we make some preliminary calculations using S-R matrix. From the above intake fraction estimates for the 40 power plants across the US, there would be about six times more exposure to particulate matter per unit emissions for SO_2 than for NOx. Since SO_2 emissions from power plants exceed emissions of NOx, the true measure of atmospheric modeling uncertainty or bias in the Abt Associates report is the uncertainty related to sulfate modeling, which is relatively insubstantial. This also has implications for our interpretation of the health evidence, as information related to sulfates will be relatively more important than information related to nitrates. In fact, my preliminary calculations using S-R matrix indicate that the sulfate impact from the power plants in the Abt Associates report actually exceeds the total particulate matter impact, due to the negative influence on nitrates.

Thus, we have shown that S-R matrix does not appear to have substantial biases in its estimation of population exposure to particulate matter. However, this does not necessarily imply that the model is correct, as it could be the case that the comparison models were biased for identical reasons as S-R matrix. Since S-R matrix was calibrated to monitored concentrations,

this provides one external checkpoint of the validity of the model. Another way that we can check the validity of S-R matrix is by examining how the non-linear patterns of sulfate and nitrate formation compare with patterns described elsewhere (*7*). Without going into great detail, the methodology used by S-R matrix to determine reactions between ammonium, sulfate, and nitrate leads to nearly identical relationships as documented by West et al. (*7*), providing further support for the validity of the Abt Associates approach.

Evaluation of Health Evidence

The second major aspect of the Abt Associates analysis we must consider is the health evidence and its validity. Since a comprehensive discussion of all health endpoints is beyond the scope of this review, I focus on selected health evidence but briefly consider three broad questions that could significantly alter the interpretation of the literature:

- Is the assumption that sulfate and nitrate particles have equal toxicity as average ambient particles valid?
- Is the assumption that ambient particulate matter levels in the model region are above any potential population threshold valid?
- Is the choice of studies for major health endpoints representative and unbiased?

Clearly, none of these questions can be resolved definitively within this document. But, the important issue is whether the assumptions made in the Abt Associates report reflect a reasonable current interpretation of the literature. In other words, it is not incumbent on Abt Associates to show definitively that all particles have identical toxicity, but rather that it is

equally likely that sulfates and nitrates are more or less toxic than average and that equality is a reasonable best estimate.

For the relative toxicity question, I paraphrase an argument presented in a recent publication (*8*). The two major cohort mortality studies (*9-12*) found significant relationships between premature mortality and sulfate concentrations, with the impact per unit concentration slightly greater than that of $PM_{2.5}$. Significant associations have also been shown in the time-series mortality literature (*13-16*). Time-series mortality studies that have not shown significant effects (*17*) have tended to have insufficient statistical power to detect effects, were they to exist. There is also limited toxicological evidence supporting sulfate health effects, although the evidence is far from conclusive. For example, some studies in rats have found respiratory effects from sulfate particles, especially in conjunction with simultaneous elemental carbon and ozone exposure (*18, 19*). Sulfur-related compounds had an effect on cardiovascular-related endpoints in dogs (*20*). There is little positive or negative evidence for nitrate particles. There is limited time-series evidence indicating positive associations between nitrate and mortality (*13, 21*), with one study that did not find statistical significance suffering from the statistical power problem cited above (*17*).

From this evidence (which represents only a brief overview of a small subset of the literature), I would conclude that it is appropriate in general to assign health impacts to sulfate and nitrate particles, although substantial uncertainties are likely. Although some evidence implies that motor vehicle-related particles could be most toxic (*22*), this study found similar toxicity for coal-related particles as for average ambient particles. In addition, the above evidence and other findings that crustal fine particles are less toxic than combustion particles (*15, 22*) are supportive of the Abt Associates assumptions. Using average particle toxicity may

underestimate or overestimate the impacts, but there is no evidence at this time strongly supportive of specific deviations in either direction. The approach taken by Abt Associates is therefore a reasonable one.

For the second point listed above, Abt Associates correctly points out that assuming a threshold would likely require one to alter the assumed slope of the concentration-response curve at concentrations above the threshold. The net effect of this on estimated benefits would be unclear. However, the literature to date has not demonstrated a threshold. Using mortality as an example, the most recent cohort study (*12*) did not show any evidence of a threshold, with annual average $PM_{2.5}$ concentrations down to approximately 9 $\mu g/m^3$. According to S-R matrix, $PM_{2.5}$ concentrations exceed this level for nearly 90% of the US population, particularly in the vicinity of the power plants modeled in the Abt Associates analysis. Furthermore, time-series studies of mortality (*23*) have found that any potential population threshold would likely be quite low. Given these points along with the fact that incorporating a threshold might either increase or decrease impacts, depending on the assumed functional form, the Abt Associates approach is reasonable and unlikely to contribute to significant bias.

Turning to the final point, I first consider premature mortality in detail, and then briefly discuss morbidity endpoints. There are two major decisions that must be made in incorporating premature mortality into a health impact analysis. The first is whether to rely on evidence from the cohort mortality literature or the time-series mortality literature, and the second is related to which studies are most representative of the selected body of literature.

It is clear on theoretical grounds that one would prefer to use evidence from cohort studies when possible, assuming that those cohort studies correctly characterize the relationship between the pollutant and the health outcome. Although studies have shown that time-series

studies with longer time windows can capture a greater magnitude of effect (*24*), a significant gap would remain provided that the effects of particulate matter are either cumulative or can extend beyond a one month period. The only logical reason to exclude cohort mortality evidence would be if one believed that the findings were spurious.

To evaluate whether this is likely to be the case and to consider the appropriate concentration-response function implied by the cohort mortality literature, we note that there are four primary cohort studies to date that provide some evidence about the effects of air pollution on mortality – the Harvard Six Cities Study (*9*), the American Cancer Society study (*10, 12*), the Adventist Health Study of Smog (*25*), and the Washington University-EPRI Veterans' Cohort Mortality Study (*26*). The first two of these studies are population-based and have undergone an extensive re-analysis (*11*). The Adventist Health Study of Smog was an analysis of residents of California who were Seventh-Day Adventists (a religious organization that largely abstains from smoking, alcohol consumption, and drug use), making it less generalizable to the population at large. The Veterans' Cohort is a study of mild-to-moderate hypertensive veterans receiving medical care for their hypertension at VA hospitals, again a population that may not generalize to the US as a whole. It had also not yet been published in final form at the time of this review. Nevertheless, we consider all four studies to some degree in the analysis of an appropriate concentration-response function.

The following table provides the core findings from the first three of these studies. The findings from the Veterans' Cohort are not included, as this study has not yet been published in final form and because the results are presented in a somewhat different format (fractional risks at mean value of pollutant less background). However, it is worth noting that this study found no

significant positive effect of $PM_{2.5}$ (and in fact, the effect was negative in some models, indicating less mortality at higher levels of $PM_{2.5}$).

The relative risks reported in the first three studies have been translated into percentage increases in mortality per $\mu g/m^3$ increase in $PM_{2.5}$ concentrations. When studies reported more than one value, I have provided a few representative values or a range. To avoid clutter in the table, I have only provided the central estimates from the models in these columns, noting when the estimates are not statistically significant, and have only given the $PM_{2.5}$ estimates. The value used by Abt Associates in their primary mortality estimate is placed in bold.

Study	Publication	% increase in mortality per $\mu g/m^3$ of $PM_{2.5}$	Notes (all table references refer to tables in original publications)
Harvard Six Cities	Dockery et al., 1993	1.2%	Using estimates from Table 3
	Krewski et al., 2000	0.8% - 1.5%	Across models in publication (Tables 3, 7, 14)
American Cancer Society	Pope et al., 1995	0.6%	Based on median PM
	Krewski et al., 2000	0.5%	Using model with mean PM rather than median (Table 31)
		0.1% - 1.2%	Across other models in publication (Tables 38, 46, 50), based on median
	Pope et al., 2002	**0.4%**	Using 1979-1983 concs.
		0.6%	Using 1999-2000 or average concs
Seventh Day Adventist	McDonnell et al., 2000	0.8%	From Table 2 (males only; not statistically significant)

A few key points emerge from this table. First, most available estimates exceed the 0.4% value used by Abt Associates. The lower values from the Krewski et al. (2000) reanalysis were in models including both $PM_{2.5}$ and SO_2. If one were to apply these values, it would be necessary to infer a causal effect of SO_2 on mortality. This inference is not well supported by the literature and has been shown to increase total health impact estimates for power plants substantially (*27*).

Thus, the estimate used by Abt Associates is a somewhat conservative value given the presupposition that a long-term exposure effect exists. If one believes that the findings for hypertensive veterans in the Washington Unversity-EPRI study represent a generalizable relationship and that the analytical methods in this study supercede the methods in the other studies cited above, then the Abt Associates estimate would not be conservative, and one would need to turn to the time-series literature for appropriate mortality estimates. I would conclude that the choice to include mortality from long-term exposure but to use a lower bound value from the literature is a reasonable decision based on currently available evidence.

Finally, I briefly consider the morbidity evidence used by Abt Associates. Broadly, the endpoints are appropriate, as they reflect respiratory and cardiovascular effects of varying ranges of severity, which is consistent biologically with available evidence. The authors took care to remove overlapping health outcomes (such as emergency room visits and hospital admissions), which is appropriate methodologically. Looking at a few specific study choices, the use of the 14-cities study (*28*) for hospital admissions for selected endpoints is appropriate, given that it employs an identical statistical methodology across all cities, minimizing the difficulty in combining evidence across studies. For many other morbidity endpoints, limited information exists in the literature, but the studies chosen by Abt Associates are representative and do not appear significantly biased. For example, for asthma attacks, a recent meta-analysis (*29*)

combined six studies to yield an estimate of a 0.3% increase in asthma attacks per $\mu g/m^3$ increase in daily PM_{10} concentrations. The study selected by Abt Associates implies a concentration-response function approximately a factor of two lower.

Conclusions

This review evaluated two critical aspects of the Abt Associates report. Through careful evaluation of the atmospheric modeling from a health-relevant perspective, we concluded that bias for sulfates was minimal. For nitrates, uncertainties appeared greater, but the small contribution of nitrates to total benefits makes this uncertainty relatively insubstantial. In terms of the health literature, the choices made by Abt Associates for mortality provided estimates at the lower end of the range in the literature provided that cohort evidence is believed. The morbidity estimates are also in line with the prevailing literature. Although significant uncertainties exist and can be quantified using information from this review and other sources, the findings from the Abt Associates report appear reasonable and useful for public policy analysis.

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