Estimated Public Health Impacts of Criteria Pollutant Air Emissions from the

Salem Harbor and Brayton Point Power Plants

Jonathan Levy [1]
John D. Spengler [2]
Harvard School of Public Health
665 Huntington Ave.
Boston, MA 02115

Dennis Hlinka
David Sullivan
Sullivan Environmental Consulting, Inc.
1900 Elkin Street, Suite 240
Alexandria, VA 22308

[1] Research Fellow, Department of Environmental Health
[2] Akira Yamaguchi Professor of Environmental Health and Human Habitation, Director of Environmental Science and Engineering Program

May, 2000

Table of Contents

<u>**Executive Summary:**</u> **Estimated Public Health Impacts of Criteria Pollutant Air**

Emissions from the Salem Harbor and Brayton Point Power Plants

<u>**BACKGROUND**</u>

To estimate the health impacts of emissions and benefits of emission reductions from

power plants that had been "grandfathered" under the Clean Air Act, we developed a damage

model and applied it to two power plants in Massachusetts. Salem Harbor is an 805 megawatt

power plant in Salem, Massachusetts, and Brayton Point is a 1611 megawatt power plant in

Somerset, Massachusetts. Both power plants are largely coal-fired, with approximately one

million short tons of coal burned at Salem and three million short tons of coal burned at Brayton

each year.

For our model, we focused on emissions of sulfur dioxide (SO_2), nitrogen dioxide (NO_2),

and particulate matter (PM_{10}). We considered three emission scenarios for both power plants:

- Actual emissions, derived from reported emission rates between 1996 and 1998

- Lower target emissions, estimated from the application of Best Available Control

  Technology to new coal-fired power plants (0.30 lb/MMBTU for SO_2, 0.15

  lb/MMBTU for NOx, and 0.01 lb/MMBTU)

- Maximum allowable emissions, defined as the emission rate if the plants operated at

  their maximum design capacity heat rate

To estimate the health impacts of these emission scenarios, we defined a population of

interest living in New England, eastern New York, and New Jersey. In our baseline model, we

used the CALPUFF atmospheric dispersion model (Earth Tech, Concord, MA) to estimate the

pollution exposure to the approximately 32 million people across this region. This model

considers detailed meteorological patterns and chemical transformations of the pollutants, along with characteristics of the power plants.

To translate marginal concentration changes from these sources into estimated adverse health effects, we combined demographic information taken from the U.S. Census with epidemiological studies that estimated concentration-response relationships. We took our baseline estimate of premature mortality from the American Cancer Society prospective cohort study, which found a significant relationship between long-term exposure to particulate matter and mortality rates. Epidemiological studies also provided estimates of air pollution effects on morbidity outcomes, such as hospitalizations, asthma attacks, and respiratory symptoms.

MAJOR FINDINGS

In general, ambient concentrations were greatest close to the source for primary pollutants (within 5 miles for PM_{10} and SO_2) and peaked further downwind for secondary particles (approximately 20 miles). Secondary particles are formed in the atmosphere by chemical reactions involving SO_2 and NO_2 emissions. Although diminished by distance from the source, some pollution was distributed across the entire region of interest.

Given these air pollution impacts, some of the estimated health impacts for both actual and lower target levels are depicted in the following table:

	Salem Harbor			Brayton Point		
	Actual	Target	**Benefits (Actual – Target)**	Actual	Target	**Benefits (Actual – Target)**
Premature deaths/year	53	10	**43**	106	25	**81**
Emergency room visits/year	570	110	**460**	1,140	270	**870**
Asthma attacks/year	14,400	2,800	**11,600**	28,900	6,900	**22,000**
Daily incidents of upper respiratory symptoms/year	99,000	19,000	**80,000**	199,000	47,000	**152,000**

This table indicates that our best estimates for the current public health impacts among the 32 million people affected include:

- 53 premature deaths per year from Salem Harbor and 106 premature deaths per year from Brayton Point

- 570 emergency room visits per year from Salem Harbor and 1,140 emergency room visits per year from Brayton Point

- 14,400 asthma attacks per year from Salem Harbor and 28,900 asthma attacks per year from Brayton Point

- 99,000 daily incidents of upper respiratory symptoms from Salem Harbor and 199,000 daily incidents of upper respiratory symptoms from Brayton Point

Additional key findings include:

- Per capita health risks were greatest near the power plants and decreased with distance from the source. However, only 20% of total impacts occurred within 30 miles of the plants (15% for Brayton Point and 32% for Salem Harbor), since more than 90% of affected individuals live beyond 30 miles of the plants.

- Secondary sulfate particles were responsible for a majority of the estimated health effects, associated with a relatively high SO_2 emission rate in comparison with other pollutants.

DISCUSSION AND IMPLICATIONS

There are important public health benefits of reducing current emissions to the lower levels that would be reached by using the best available control technologies required for newer power plants under the 1990 Clean Air Act and required by EPA as retrofits to some older plants.

An estimated 124 premature deaths would be averted per year, along with 1,300 fewer emergency room visits, 34,000 fewer asthma attacks, and 230,00 fewer daily incidents of upper respiratory symptoms.

Although there are uncertainties in these estimates related to the reported confidence in the epidemiological findings and the dispersion model estimates, the above health estimates were relatively insensitive to some of the assumptions inherent in the model, including the geographic boundaries, plant characteristics, and meteorological assumptions. Conversely, estimated effects were sensitive to choice of dispersion model (CALPUFF vs. ISCST3 within 50 km of the source and SLIM3 beyond 50 km). However, comparative analysis demonstrated that the CALPUFF and ISC-SLIM3 models had similar outputs for all but secondary pollutants, indicating that the meteorological assumptions have a limited impact on aggregate impacts but that more refined atmospheric chemistry adds to the accuracy of CALPUFF. Reliance on only short-term mortality studies would lower estimated impacts. However, the Health Effects Institute recently re-analyzed the long-term mortality studies and found the estimated damage function (i.e., relationship between mortality and air pollution) within these studies to be relatively insensitive to key assumptions, increasing the degree of belief in these studies.

In summary, we have applied models validated in past investigations to develop a tool that can be used by policymakers to evaluate the benefits of control options. The findings from this investigation are meant as an input to the decision process. A comprehensive evaluation of the benefits and costs of emission controls would need to consider changes in production efficiency and plant configuration, impacts of additional air pollutants (e.g., ozone, carbon monoxide, mercury, and other air toxics) and water pollutants, and changes in the fuel cycle.

However, this study demonstrates that the health benefits of emission controls in power plants can be quantified with estimates that are reliable.

Jonathan Levy [1]
John D. Spengler [2]
Harvard School of Public Health
665 Huntington Ave.
Boston, MA 02115

Dennis Hlinka
David Sullivan
Sullivan Environmental Consulting, Inc.
1900 Elkin Street, Suite 240
Alexandria, VA 22308

[1] Research Fellow, Department of Environmental Health
[2] Akira Yamaguchi Professor of Environmental Health and Human Habitation, Director of Environmental Science and Engineering Program

Rationale

Air pollution emissions from older power plants can often be much greater than emissions from newer plants, in part because older plants are exempt from modern emission standards required of new plants under the Clean Air Act. Within the state of Massachusetts, five of these "grandfathered" power plants have been targeted for emission reductions, based on their significant contribution to emissions in the state. Governor Paul Cellucci has pledged to cut air pollution from these power plants, and the Massachusetts Department of Environmental Protection is in the process of initiating rulemaking.

One of the primary reasons cited for the proposed emission reduction is the adverse human health impact of power plant emissions. Epidemiological and toxicological evidence suggest that exposure to elevated levels of particulate matter (PM), sulfur dioxide (SO_2, with most health effects associated with sulfate particles), and other combustion pollutants can lead to numerous adverse health effects, ranging from respiratory symptoms to premature death. To make rational policy choices, there is a need to understand and quantify the health benefits associated with changes in power plant emissions. This information can be used in conjunction with estimates of control costs to determine the cost-effectiveness of emission controls.

To understand the magnitude of health impacts from grandfathered power plants in Massachusetts, we focus our analysis on two of the aforementioned five plants – the Salem Harbor and Brayton Point power plants. Salem Harbor is located in Salem, Massachusetts, approximately 17 miles northeast of downtown Boston. Brayton Point is located in Somerset, Massachusetts, approximately 45 miles south of downtown Boston (Figure 1). Both power plants are owned by PG&E Generating.

The goal of our analysis is to estimate the magnitude and distribution of health impacts under three different emission scenarios. Our baseline damage estimate is derived from actual emissions reported by the power plants. Given a set of proposed target emission rates for PM_{10}, NO_2, and SO_2 (based on the emission rates of new coal-fired power plants using Best Available Control Technology), we can estimate the target health effects and therefore determine the expected benefits of controlling emissions from these grandfathered power plants. Finally, since these power plants could theoretically increase generation and their annual emissions in the future, we estimate the health effects under a maximum potential emissions scenario, to understand the range of health values that might be expected from these two plants.

Model Structure and Overview

To estimate the health effects of emissions from Salem Harbor and Brayton Point under different emission scenarios, we apply a damage function model. A damage function model is essentially a quantitative methodology to translate emissions into concentration changes across a defined region and estimate the expected damages from these concentration changes (either in health or monetary terms). Health impacts are determined using epidemiological findings coupled with demographic information. To compare these impacts with the economic costs of control, the impacts are often placed in monetary terms using willingness-to-pay and cost-of-illness studies.

This methodology has been used in multiple recent studies of the health effects of power plants. Three studies in recent years helped to develop the damage function approach and provide power plant damage estimates – studies conducted by the European Commission (EC, 1995a), Hagler Bailly (Rowe *et al.*, 1995), and Oak Ridge National Laboratory and Resources

9

for the Future (ORNL/RFF, 1994). The findings from these studies differed quite widely, due to differences in population distributions, meteorological patterns, assumed health effects, and other model components. A recent publication by Levy and colleagues (1999) compared the differences between these three models and provided an updated damage function model that addressed these differences. According to this publication, a 62 MW fuel oil-fired cogeneration plant in Boston was responsible for an estimated increased risk of 0.3 premature deaths per year, distributed across the Northeast and assuming only short-term mortality impacts (0.9 estimated mortality deaths per year using long-term studies).

In addition, Samet and colleagues estimated the health impacts associated with the Centralia Steam Electric Generating Plant in Washington State (Samet *et al.*, 1997). Centralia is a coal-fired plant with a capacity of 1340 MW, using an electrostatic precipitator as its primary baseline control. This study used the CALPUFF air dispersion model to estimate the effect of Centralia on ambient concentrations within a 150-mile radius of the plant (affected population of 5.5 million people). Based on estimates of the effect of long-term exposure on mortality from the Harvard Six Cities Study (Dockery *et al.*, 1993) and the American Cancer Society CPS II study (Pope *et al.*, 1995), the best estimate for baseline premature mortality was 34 annual deaths due to particulate matter.

Our model adopts a similar approach to the above studies. The choice of models and studies, as well as the preliminary monetary valuation phase, is similar to the choices made in the studies by Samet *et al.* (1997) and Levy *et al.* (1999). Given the structure of the estimated lower target emissions, we focus exclusively on the health effects of power plant emissions of particulate matter, NO_2, and SO_2. Both of these gaseous pollutants contribute significantly to secondary particle formation. Within this category, we only consider direct health effects from

inhalation, excluding secondary pathways through food and water as well as upstream impacts from the entire fuel cycle.

To understand the degree of uncertainty surrounding our central impact estimates, we use Monte Carlo analysis to propagate the uncertainties estimated in each phase of the damage function. This includes our assessments of uncertainties in the concentration estimates from the dispersion models and the health effects per unit concentration change. Any of the assumptions evaluated in the sensitivity analysis are not included in this uncertainty propagation; thus, the confidence intervals presented reflect only the baseline model assumptions and do not reflect model uncertainty. The Monte Carlo analysis is conducted using @RISK (Palisade Corporation, Newfield, NY).

Power Plant Characteristics

Some of the basic characteristics of Salem Harbor and Brayton Point are presented in Table 1. In addition, for our damage function model, stack parameters for the Salem Harbor and Brayton Point plants were compiled from previous modeling studies (Tables 2 and 3).

As mentioned above, we evaluate the impacts of three different emission scenarios. Actual emissions of SO_2, NOx, and filterable PM_{10} were estimated as the three-year average of emission rates between 1996 and 1998. Emission rates were determined as the product of the annual tons of emissions and the number of hours of operation for each unit per year. We also estimated condensable particulate matter emissions using the latest AP-42 emission factors and three-year average heat inputs, given EPA guidance on the most appropriate factors applicable to each stack. According to AP-42, condensable particles are defined as material that is emitted in the vapor state which condenses immediately after emission to form aerosol particles (primarily

sulfate or nitrate). These condensable particles are generally considered to be distinct from both filterable particles and secondary particulate matter.

The lower target emission rates were estimated as typical rates given the application of Best Available Control Technology (BACT) to new coal-fired power plants built in recent years. The derived rates per unit of heat input were 0.30 lb/MMBTU for SO_2, 0.15 lb/MMBTU for NOx, and 0.01 lb/MMBTU for filterable PM_{10}. The average heat input for each power plant between 1996 and 1998 was used to estimate emissions per second for the above pollutants. For condensable particles, the target emission rates were estimated by applying the actual ratio of condensable to filterable particulate matter emissions to the calculated target filterable PM_{10} emission rate. Embedded in the target emission calculations is the assumption that flue gas desulfurization occurs only under the target emissions scenario.

To estimate maximum allowable (potential) emissions of SO_2, NOx, and filterable PM_{10}, we use the maximum design capacity heat rate for the stacks of each power plant. As for the target calculation, we estimate the maximum potential condensable PM_{10} emission rate using the actual ratio of filterable to condensable emissions and the derived filterable potential PM_{10} emission rate. For all scenarios, all emission rates are assumed to be uniform across the year.

Dispersion Modeling

The choice of a pollutant dispersion model can have a significant influence on the estimated health impacts, particularly when considering the geographic distribution of impacts. To understand the potential range of aggregate effects as well as the geographic patterns, we therefore use two separate dispersion modeling schemes. Each dispersion model is intended to estimate the concentrations of the primary pollutants described above (PM_{10}, SO_2, NOx) as well

as secondary sulfate and nitrate particles. Because of the difficulty and complexity of modeling the effects of a single source of NOx emissions on ozone formation across an entire year, as well as the relatively larger impact hypothesized for particulate matter, we do not construct an ozone model for this analysis. The health impacts of ozone related to NOx emissions are therefore omitted from our impact estimates, but are approximated in our uncertainty/sensitivity analysis.

For either model, we selected a receptor grid that corresponded to the geographic area of interest. All receptors were located between 45 degrees N and 40 degrees N and between 67 degrees W and 75 degrees W (Figure 2). This established a receptor region stretching approximately 200 miles in all directions from Boston, including populations in New England, New York, and New Jersey (as well as areas of the Atlantic Ocean in which no human health impacts would occur). In total, this resulted in an affected region of 32,389,920 people, including approximately 6 million in Massachusetts and 13 million in New York. Although this region omits some potentially affected populations (i.e., northern Maine and western New York), it was selected to encompass a significant fraction of the affected population without extending the dispersion model boundaries excessively. Within our investigation, we assume that the population is stable, so that we are analyzing the benefits of control strategies at present rather than future controls.

For our primary model, we select CALPUFF as a state-of-the-art model of meteorological conditions and atmospheric dispersion. CALPUFF is a Lagrangian puff model developed by Earth Tech (Concord, MA) that is programmed to simulate the continuous emission of puffs of pollutants into ambient windfields (EPA, 1998). Through the CALMET program, CALPUFF is able to handle complex three-dimensional windfields, a particularly advantageous feature for power plants located in a coastal area. The initial phase of CALPUFF

analysis involves deriving the CALMET meteorological file, which is a complex process involving the synthesis of numerous data points across time. The method by which the CALMET file was derived is explained in full in Appendix 1. Briefly, a full year (January 26, 1999 through January 25, 2000) of gridded hourly surface, upper air, and cloud cover data were compiled to construct the three-dimensional windfields. To obtain the requisite surface parameters and temperature lapse rate, we used a program created by SSESCO to develop representative data from the monitors in the modeling domain.

The basic coordinate grid for CALMET consisted of 45 grid cells along the x-axis (east-west) and 36 grid cells along the y-axis (north-south), spaced 15 km (9 miles) apart. The coordinate system was converted to a Lambert projection grid, with the southwest corner of the model domain set to (-330, -263), representing a latitude of 40.338 degrees N and a longitude of 76.184 degrees W. Eight vertical layers were incorporated into the CALMET processing, with heights of: 20m, 50m, 100m, 500m, 1500m, 2500m, 3500m, and 4500m.

CALPUFF was run with separate model input files for Brayton Point and Salem Harbor, under the three emission scenarios of maximum potential, lower target, and actual emissions. We used the stack parameters and emission rates as presented in Tables 2 and 3, using AP-42 to estimate that 95% of NOx emissions are initially in the form of NO (with the remainder as NO_2). Building downwash was not incorporated for the Salem Harbor plant, given stack heights above Good Engineering Practice (GEP). For Brayton Point, the surrounding building heights and widths were input into EPA's Building Profile Input Program (BPIP, version 95036) to derive precise directional heights and widths.

Because of the size and complexity of the model, 25 separate model runs were needed across the model year, with 25 semi-monthly concentration files output by the model. The model

restart file option was used in the sequence of model runs for each of the 25 model periods, to keep track of the modeled puffs from one period to the next. The RIVAD/ARM3 chemical transformation mechanism was used to determine the conversion of NOx into NO_3 and HNO_3 as well as SO_2 into SO_4.

To increase the resolution of the model, we applied a nesting factor of two for the sampling grid option (MESHDN). In addition, we used large sets of discrete receptors within 20 km (12 miles) of the power plants (6,097 for Brayton Point and 7,407 for Salem Harbor), to provide greater accuracy at close range. These receptor points corresponded to the census blocks from the U.S. Census, with coordinates converted from UTMs to a Lambert projection grid.

The CALPOST program was used to develop concentration files for all modeled compounds under all emission scenarios for both power plants. A post-processing program was used to compute the time-weighted average of the concentrations from the 25 semi-monthly output files. In order to match the predicted concentrations with the demographic data needed for the impact calculations, our final receptor grid consisted of the geographic centroids of all U.S. census tracts between 45 degrees N and 40 degrees N and between 67 degrees W and 75 degrees W. The post-processing program identified the maximum modeled concentration within 3 km (2 miles) of receptors within 15 km (9 miles) of the plants, and within 10 km (6 miles) of receptors greater than 15 km (9 miles) from the plants. The final output of the post-processor consisted of annual average concentrations for each pollutant (SO_2, NO_2, PM_{10}, SO_4, and NO_3) for each emission scenario. It should be noted that most of the particulate matter is likely in the fine particle fraction ($PM_{2.5}$), but we estimate PM_{10} for comparability with a majority of the epidemiological studies.

Along with CALPUFF, we also apply a simpler modeling scheme to help test model uncertainty and to evaluate the sensitivity of our findings to model selection. A simpler model also allows for more extensive sensitivity analysis over key assumptions, which cannot be conducted in CALPUFF given the complexity and level of detail of the model. For the simpler model, we combine two separate models as applied in the Levy *et al.* (1999) and Rowe *et al.* (1995) studies, allowing for the incorporation of the complete geographic range of influence with detailed near-source information.

Within 50 km (31 miles) of each plant, the Industrial Source Complex Short-Term model (ISCST3, version 98356) was used to evaluate impacts on ambient concentrations. To align this model with CALPUFF, we used 1990 U.S. census data to develop the receptor grid. Placing receptor points at the geographic centroid of each block group within each census tract, receptor grids of 38,225 receptors for Salem Harbor and 29,372 receptors for Brayton Point were established. The total population within 50 km (31 miles) of Salem Harbor is 3,293,127, while there are 1,918,746 people within 50 km of Brayton Point. The latitude, longitude, and elevation height of each receptor were extracted and used in the dispersion model.

Beyond 50 km, concentrations were estimated using the Sector-Average Limited Mixing Mesoscale Model (SLIM3). This dispersion model uses an exponential decay formulation to allow for the chemical conversion and deposition of SO_2 and NO_2, allowing us to estimate secondary sulfate and nitrate particle formation. Previous analysis has demonstrated good concordance between SLIM3 and ISCST3 near the 50 km juncture (Levy *et al.*, 1999; Rowe *et al.*, 1995). For SLIM3, we used the census tract grid described above for the CALPUFF analysis, so that identical populations would be considered for both model regimes. The SLIM3 receptor

grid overlapped the domain of the ISCST3 receptor grid, and any overlapping populations were omitted from the health effect estimation for pollutants estimated in ISCST3.

For the ISC-SLIM3 analysis, we used five-year pre-processed meteorological data sets. For Salem Harbor, meteorological data were derived from 1991-1995 surface data from Logan Airport in Boston and mixing height data from Portland, Maine. For Brayton Point, the 1989-1993 surface data for Providence, Rhode Island and mixing height data for Chatham, Massachusetts were used. Although these are conventional time period selections for ISC-SLIM3 (with the assumption that the use of five years of data reduces the meteorological variability), it should be noted that both of the time periods differ from the one-year period used in CALPUFF.

For both CALPUFF and ISC-SLIM3, some post-processing was necessary to ensure that accurate calculations of secondary sulfate and nitrate particles were made. First, both models report the sulfate and nitrate concentrations as SO_4 and NO_3. However, we are interested in estimating the mass of sulfate and nitrate particles, which are typically in the form of ammonium sulfate [$(NH_4)_2SO_4$] and ammonium nitrate (NH_4NO_3). To convert, we increase the concentrations by the ratios of the molecular mass of the ammonium compounds to the reported compounds, resulting in adjustment factors of 1.37 for sulfates and 1.29 for nitrates.

Second, although sulfate is always found in the particle phase, nitrate concentrations will include both gaseous nitric acid and particle ammonium nitrate (E.H. Pechan and Associates, 1997). Particle nitrate will only be formed at low temperatures and when sufficient ammonia exists to neutralize all of the sulfate. To account for the fact that nitrate would not be likely to form during warm weather, we use the methodology of E.H. Pechan and Associates and divide the estimated nitrate concentrations by four (reflecting the three winter months in which

particulate nitrate would be hypothesized to form). We discuss issues related to chemical

conversion assumptions within our sensitivity analysis.

For the uncertainty propagation, it is generally assumed that estimates from ISCST3 have

a 95% confidence interval between 50% and 150% of the central estimate (Bowers and

Anderson, 1981). As a conservative assumption (given the presumed increased accuracy

associated with CALPUFF), we assume the same confidence interval to apply to CALPUFF. For

SLIM3, we assume that estimates have a 95% confidence interval between 25% and 175% of the

central estimate (Rowe *et al.*, 1995), reflecting the simplicity of the model structure. Because of

the number of necessary differences in model implementation and assumptions, we do not

include model uncertainty (i.e., the differences in predicted findings between ISC-SLIM3 and

CALPUFF) within our confidence limits. The impacts of model uncertainty are addressed within

the sensitivity analysis.

Health Effect Estimation

For the health effect estimates, we rely on a meta-analysis of the epidemiological

literature on both mortality and morbidity effects of particulate matter, NO_2, and SO_2. We largely

select epidemiological studies and the methodology for combining their results while accounting

for potential co-pollutant confounding from the recent publication by Levy *et al.* (1999).

We derive our estimate of premature mortality from long-term exposure to particulate

matter from the American Cancer Society study (Pope *et al.*, 1995), a comprehensive prospective

cohort study following over 500,000 individuals from all 50 states. We estimate that mortality

rates will be approximately 4% higher for a $10 \, \mu g/m^3$ increase in annual average PM_{10}

concentrations (95% confidence interval: 2%, 6%). Because of the chronic nature of power plant

exposure, we assume that this estimate is applicable to the case study and use it for our baseline

estimate, with no mortality effects assumed from gaseous pollutants. Although it is possible that

long-term exposure to NO_2 and SO_2 have independent effects on mortality, any observed effects

would likely be related to the secondary nitrate and sulfate particles formed.

Another type of study that has investigated the relationship between air pollution and

premature mortality is the time-series study, which considers daily changes in air pollution

patterns and associates them with daily changes in number of deaths. These studies are referred

to as "acute mortality" studies, since they consider premature deaths from short-term changes in

air pollution rather than from long-term air pollution patterns ("chronic mortality"). Although

acute mortality studies may estimate different phenomena than chronic mortality studies, we use

only the chronic mortality estimate for our baseline scenario. This may yield a slight

underestimate in the number of estimated premature deaths, although the difference in

concentration-response functions makes the potential underestimate relatively small.

Within our sensitivity analysis, we consider the premature mortality effects if only time-

series studies evaluating acute mortality are included. As in the Levy (1999) study, we use a

random effects model to pool 24 studies addressing acute mortality impacts of criteria air

pollutants. According to this model, we estimate that acute mortality rates will increase by 0.6%

for a 10 μg/m^3 increase in PM_{10} concentrations (95% CI: 0.2%, 0.9%), controlling for the effects

of correlated gaseous pollutants. There is also some weak evidence of an independent effect of

gaseous SO_2. Through our meta-analysis, much of the SO_2 mortality effect can likely be

explained by correlated particulate matter, but the residual effect of 0.04% for a 10 μg/m^3

increase in SO_2 concentrations (95% CI: Non-Significant, 0.08%) is considered in our sensitivity

analysis. It should be noted that for this estimate, as well as some subsequent morbidity

calculations, the 95% confidence interval includes negative values. This does not imply that these pollutants may have protective effects for health, but rather that either the sample sizes in the studies were insufficient to detect the true effect with significance or that the relationship does not exist. We present all negative lower bounds as "Non-Significant" to avoid confusion.

For morbidity outcomes, we largely focus on the effects of particulate matter. There is some evidence of independent effects of gaseous SO_2 and NO_2 for a number of outcomes, but we assume that these effects are related in part to particulate matter and that the residual effects of the gases acting alone are relatively insignificant. Within our analysis, we consider the potential effects of NO_2 on lower respiratory symptoms in children, although the pooled literature estimate is not statistically significant (Appendix 2). For PM_{10}, we evaluate health outcomes including: chronic bronchitis, respiratory and cardiovascular hospital admissions, emergency room visits, asthma attacks, restricted activity days, and upper and lower respiratory symptoms. The studies evaluated and the rationale behind the estimated effects are given in Appendix 2. It should be noted that Appendix 2 contains some information regarding health effects of carbon monoxide and ozone, which are not used in the central impact estimates. The percentage contributions to these health outcomes of each 10 $\mu g/m^3$ increase in PM_{10} concentrations are presented in Table 4.

For all health effects, we derive demographic characteristics of the receptors from 1990 U.S. Census data. We assume asthma prevalence to be uniform across all receptors, given a lack of census tract data on this health outcome.

<u>Results</u>

Using the CALPUFF dispersion model, we can estimate the marginal contribution of each power plant to ambient concentrations at all receptors. In Table 5, we present the population-weighted annual average concentrations for all pollutants (taking the concentration at each receptor, multiplying by the affected population, and dividing by the total population across all receptors). In the actual emissions scenario, the average concentrations associated with Brayton Point are 0.20 $\mu g/m^3$ for SO_2, 0.036 $\mu g/m^3$ for NO_2, and 0.063 $\mu g/m^3$ for total PM_{10}. Under the hypothesis that the power plants increase their production to reach the maximum allowable emission rates, these concentrations would increase to 0.41 $\mu g/m^3$ for SO_2, 0.045 $\mu g/m^3$ for NO_2, and 0.11 $\mu g/m^3$ for PM_{10}. If the target emission rates were achieved, this would reduce population-weighted average concentrations to 0.029 $\mu g/m^3$ for SO_2, 0.013 $\mu g/m^3$ for NO_2, and 0.012 $\mu g/m^3$ for PM_{10}. Patterns are similar for the Salem Harbor plant (Table 5).

To demonstrate the geographic patterns of impacts, we present contour plots and three-dimensional plots of pollutant concentrations in Figures 3-4 (primary PM_{10} concentrations) and Figures 5-6 (secondary particulate matter concentrations). These concentration plots demonstrate that primary PM_{10} (and other primary pollutants) peak close to the power plant source and decrease with distance, while secondary particles peak at longer range and generally have a more uniform concentration profile. As an example of this phenomenon, the peak SO_2 concentration from Salem Harbor emissions occurs within 3 km (2 miles) of the plant, while the peak sulfate concentration is approximately 31 km (19 miles) from the source. Although only the actual emissions scenario is presented in these figures for the sake of brevity, the patterns are similar for the maximum potential and the lower target emissions profiles.

21

Given the marginal concentration increases predicted in the dispersion models, we predict the annual health impacts from these power plants under the three emissions scenarios (Table 6). We estimate 106 premature deaths per year from current Brayton Point emissions (95% CI: 60, 150), with 210 premature deaths at the maximum allowable emission rates (95% CI: 120, 310) and 25 premature deaths at the target emission rates (95% CI: 14, 36). For Salem Harbor, current emissions are estimated to cause 53 premature deaths per year (95% CI: 29, 76), a quantity which would increase to 97 given maximum allowable emissions (95% CI: 54, 140) and would decrease to 10 given target emissions (95% CI: 6, 15). The health benefits of moving from the actual to target emissions, both for mortality and morbidity outcomes, are presented in Table 7. It should be noted that the estimates are generally rounded off to two significant figures, with greater precision provided where necessary to maintain comparability between tables.

For both Salem and Brayton, a significant fraction of the mortality and morbidity effects is associated with secondary sulfate particles. In total, 81% of estimated premature deaths from Salem and 77% from Brayton are associated with secondary particles (the combination of sulfates and nitrates). Most of these secondary particle impacts are related to sulfates, which are responsible for 85% of secondary particle deaths from Salem and 80% from Brayton. For both plants, condensable particles make a significant contribution to mortality and morbidity effects as well, with primary filterable PM_{10} associated with only 3-4% of premature deaths.

Examining the geographic distribution of impacts can help determine the proportional impacts on local communities. Using the actual emission rates, 15% of premature deaths are estimated to occur within 50 km (31 miles) of the Brayton Point power plant, with a slightly greater figure of 32% for Salem Harbor (Table 8). The difference between the two plants is largely related to the population located within 50 km of each plant. The fact that a majority of

impacts occurs outside of 50 km can be attributed to the long-range transport of secondary particles and the significant fraction of the affected population that lives beyond 50 km of the plants. As mentioned above, 1.9 million of the 32 million people (6%) in our study area live within 50 km of Brayton Point, with 3.3 million living within 50 km of Salem Harbor (10%).

Although the local population makes a relatively small contribution to aggregate impacts, on a per capita basis, the impacts are greater closer to the power plant. The per capita mortality risks are 8 in a million within 50 km (31 miles) of Brayton Point and 3 in a million beyond 50 km, with risks of 5 in a million within 50 km of Salem Harbor and 1 in a million beyond 50 km. In general, the per capita mortality risks decrease with distance from the power plants. The geographic distribution of per capita mortality risks is presented in Figures 7 and 8.

Monetary Valuation

To make comparisons with the economic costs of controls, we can place monetary values on the mortality and morbidity effects detailed above. For morbidity outcomes, these values are meant to represent the productivity and utility losses that people face, along with medical and associated economic costs. We value morbidity using willingness-to-pay (WTP) studies whenever possible, and otherwise apply a scaling factor of 2 to cost-of-illness (COI) studies or medical cost databases, to reflect additional losses (Rowe *et al.*, 1995).

For mortality, the economic value used is that of a "statistical life". This represents the amount of money that people would be willing to pay to reduce a mortality risk divided by the magnitude of that risk, not simply an abstract value assigned to human life. In other words, if an individual were willing to pay $500 to reduce their risk of death by 1/10,000, their value of a statistical life would be $5,000,000. All mortality and morbidity cost estimates are converted to

1997 US dollars, assuming health care or CPI inflation rates when appropriate.

We use a recent EPA benefit-cost analysis (EPA, 1997) to estimate the value of a statistical life. This analysis pooled contingent valuation and wage-risk studies to yield a mean estimate of $6.1M, with a standard deviation of $4.1M under a Weibull distribution. The EPA acknowledged that there is some uncertainty in using this estimate, but we adopt their baseline value for this illustrative calculation.

For morbidity, the highest values are associated with chronic bronchitis ($260,000), respiratory hospital admissions ($25,000), cardiovascular hospital admissions ($24,000), and emergency room visits ($1,000). Additional values include $220 per restricted activity day, $70 per minor restricted activity day, $61 per asthma attack day, and $18 per respiratory symptom day. The derivation of these values is presented in full in Levy *et al.* (1999).

Given these values, we estimate that reducing emissions from actual to target emission rates would reduce aggregate health damages by $280,000,000 per year for Salem Harbor and $530,000,000 per year for Brayton Point (Table 9). For both plants, 93% of this total is related to reduced premature mortality, with most of the remainder related to chronic bronchitis (4%).

Clearly, there are a number of uncertainties surrounding these value estimates. Given the significant contribution of premature mortality to the aggregate value, the monetary value of damages is strongly dependent on the value placed on a statistical life. The value we selected is an average from a number of earlier studies, and is likely representative of the literature on value of statistical life. It has been argued that lower values based on the years of life lost would be more appropriate given the populations affected by air pollution increases, but a definitive value has not been established in the literature. Our monetary estimates should therefore be considered as upper bounds on the potential social benefits of emission controls.

<u>Uncertainty/Sensitivity Analysis</u>

Clearly, our damage function model contains a number of fundamental assumptions that could potentially influence the aggregate health estimates. As indicated in Table 10, uncertainty can arise in a number of sections of the analysis. For the purpose of this assessment, we will conduct many of the sensitivity analyses on the ISC-SLIM3 model runs rather than the CALPUFF model runs, given the relative difficulty of constructing CALMET data sets and constructing CALPUFF models. In addition, we will not consider uncertainty in the monetary valuation, since the above calculation was meant to be illustrative and the primary endpoint of our analysis is the health effect estimation.

Dispersion Model Selection

Clearly, prior to using ISC-SLIM3 as our model to test the sensitivity of the findings, we need to determine whether the overall model findings are sensitive to the choice of dispersion model. Applying ISC-SLIM3 to Brayton and Salem, we find considerably lower estimated concentrations and health estimates. For example, premature deaths under the actual emissions scenario are reduced from 106 to 29 for Brayton and 53 to 9 for Salem. This significant difference can largely be attributed to differences in estimated secondary sulfate and nitrate particle concentrations.

To compare CALPUFF and ISC-SLIM3, we calculated the population-weighted average concentrations. As indicated in Table 11, the CALPUFF model yields population-weighted average concentrations that are approximately twice that of ISC-SLIM3 for both Brayton and Salem, with the exception of sulfate and nitrate particles. The average sulfate particle

concentration is 4-5 times greater for CALPUFF, while the nitrate particle concentration is over 10 times greater for both power plants. Due to the complexity of nitrogen and sulfur chemistry, particularly in a geographic area with significant sources of both pollutants and variable weather patterns, the secondary particle estimates would be expected to differ more substantially between models than would primary pollutants.

Examining the concentration patterns more closely, the difference in sulfate estimates is relatively consistent across the receptor grid. For Brayton, the mean ratio between ISC-SLIM3 and CALPUFF is 0.28 with a standard deviation of 0.08, while the corresponding values are 0.24 and 0.12 for Salem. For both plants, the largest differences between the models tend to be found closer to the source. This could be attributable to the fact that SLIM3 is often applied only beyond 50 km from the source, where the assumption of uniform vertical mixing is well supported (Rowe *et al.*, 1995). Similarly, for nitrates, we find mean ratios between ISC-SLIM3 and CALPUFF of 0.08 for Brayton (standard deviation of 0.03) and 0.07 for Brayton (standard deviation of 0.04), with larger differences near the source. The systematic differences between the models would be expected to be a function of the way in which the formation of secondary pollutants from NO_2 and SO_2 emissions is modeled. Since CALPUFF allows for varying conversion rates based on factors such as solar intensity, relative humidity, and precipitation (versus a constant value assumed in SLIM3), there is substantial reason to believe that the CALPUFF estimates are more accurate.

However, an implication of the significant contribution of secondary particles to the aggregate health impacts is that perturbations in the sulfate and nitrate formation rates within CALPUFF could significantly affect our estimates. We have not quantified this effect in our sensitivity analysis. Although we believe that the chemical conversion mechanism within

CALPUFF represents a well-supported central estimate, further analysis with plausible alternative mechanisms could be warranted.

To provide another data point comparing the two dispersion modeling regimes, we applied ISC-SLIM3 to the Centralia power plant and compared the estimated health impacts with the impacts calculated using CALPUFF. We obtained power plant characteristics and emissions information from the researchers (Kirk Winges and Jonathan Samet, personal communication). For meteorological data, we used surface meteorological conditions from Spokane and upper air conditions from Quillayute (the nearest stations in both cases) between January 6 and November 30, 1990. As in the Centralia report, we estimate health impacts on all counties within 150 miles of the plant, with individuals beyond 150 miles counted if the geographic centroid of their county falls within the 150-mile radius. For the ISC-SLIM3 model, we place receptors at the geographic centroid of each census block group within these counties, for a total of 5,010 receptors.

Looking at the dispersion modeling results, the population-weighted annual average pollutant concentrations have relatively smaller differences than for Salem and Brayton, with ISC-SLIM3 estimating higher concentrations than CALPUFF (Table 11). Particulate matter concentrations are 56% higher using ISC-SLIM3, while nitrates and sulfates are more substantially overpredicted by ISC-SLIM3 (135% and 111% higher, respectively). Thus, the finding of more substantial model differences associated with secondary particles versus primary pollutants is consistent, although the direction and magnitude differs between the two studies. In total, using the ISC-SLIM3 framework within the Centralia study would result in an estimate of 59 premature deaths per year, nearly two times the baseline CALPUFF estimate of 34.

The systematic differences between the ratios for Centralia and the ratios for Brayton and Salem could potentially be a function of differences in secondary particulate formation

associated with differences in atmospheric chemistry and meteorology in Washington State and Massachusetts. The differences might also be a function of the different chemical conversion schemes used in the studies, with MESOPUFF II used for Centralia and RIVAD/ARM3 used for Brayton and Salem.

One way to test whether the different chemical conversion mechanisms result in fundamentally different findings is to compare the impacts of a ton of emissions of SO_2 on public health in the two studies, normalized by population size. For Centralia, the estimate of premature deaths per capita per ton of SO_2 emissions is 1×10^{-10}, compared with 5×10^{-11} for Brayton and 4×10^{-11} for Salem. If we increase the comparability between studies by restricting the boundary of analysis to a 150-mile radius for Brayton and Salem and using the same dose-response coefficient for mortality, the estimates for Brayton and Salem both become 1×10^{-10}, identical to the Centralia estimate. Although this is a simple comparison, it provides some indication that the dispersion models had similar findings despite the use of different chemical conversion mechanisms.

Site Selection

One area of uncertainty arises from our choice of receptors, which consisted of most of the total population of New England as well as portions of New York and New Jersey. Clearly, this decision to choose an area beyond which no impacts would be counted is somewhat arbitrary, based somewhat on the geographic region of interest for the study. Although some might argue that concentration estimates would be less accurate at greater distances from the source, the use of three-dimensional windfields in CALPUFF reduces the potential errors at long range. Furthermore, there is evidence from a past analysis using CALPUFF that long-range

transport of pollutants could potentially influence populations as much as 1000 km (over 600 miles) from the source (Evans *et al.*, 1999). In addition, the preliminary analysis in the article by Evans found that less than half of the total estimated particulate matter impacts occurred within 500 km (310 miles) of a source, indicating that the aggregate impacts associated with Brayton Point and Salem Harbor may be greater than estimated.

One way to evaluate whether the choice of geographic boundary is reasonable is to consider the total health impacts with a tighter geographic constraint. One of the reasons for the difference between the premature deaths estimated from Centralia (34 per year) and Brayton Point (106 per year) is the different area of influence chosen. When we restrict the Brayton Point analysis to populations within 150 miles of the plant, our estimate for premature deaths is reduced to 75 per year.

Plant Characteristics

Another source of potential model sensitivity is the assumed power plant characteristics and emission rates. We obtained all power plant information from a Massachusetts Department of Environmental Protection study, but some parameters differed slightly between this study and information in public databases (FERC filings and EPA CEMS and AIRS databases). To examine whether our findings differed using the public data sources, we ran the ISC-SLIM3 model using these input parameters. Most of the parameters are similar, but there are some differences associated with stack parameters and emission rates. Specifically, stack exit temperatures are somewhat lower in the public data than in the data used in our primary analysis. For Unit 4 of both plants, the exit velocities are approximately 30% lower, and the SO_2 and NOx emission rates are somewhat higher. All other data are approximately equivalent. Since the PM

emission data are derived from actual measurements in our baseline model, we use the identical PM$_{10}$ emissions rate in this alternative assessment.

Using the public data, our estimates of actual health impacts are increased by 23-33% for Salem Harbor and 14-24% for Brayton Point, depending on the health effect. As a result, the health gains in moving from actual to target emissions are greater under this scenario. For example, 11 premature deaths from Salem and 33 premature deaths from Brayton are associated with actual emissions using public data and the ISC-SLIM3 model, compared with 9 and 29 with the baseline data set using the ISC-SLIM3 model. Thus, the set of input parameters used for our baseline model likely reflects a slightly conservative estimate of aggregate impacts.

Meteorological Assumptions

To test the potential sensitivity of the model findings to changes in the meteorological input data, we evaluated the health effects of both power plants using a different set of meteorological assumptions. Since this was not feasible to do for the CALPUFF analysis, given the time required to construct the meteorological data as well as the comprehensive meteorology considered, we again use the ISC-SLIM3 analysis. Although the use of alternative meteorological information is not directly applicable to our CALPUFF analysis, this sensitivity analysis can potentially illustrate the degree to which perturbations in meteorological inputs might influence aggregate health impacts.

For ISC-SLIM3, we assumed that meteorological data from the Boston area was applicable to our entire model domain. Since the Boston meteorological station is located on the coast, there might be some "sea breeze" effects present in this data set that would not occur inland. In addition, many of the power plant impacts occur to the west of Boston (given the

coastal setting), and the Boston meteorological regime may not represent conditions elsewhere. To test the impact of these assertions, we constructed a meteorological data set using surface meteorology from Hartford, CT and using upper air conditions from Albany, NY.

Using this meteorological data set, our ISC-SLIM3 model estimates an actual annual impact of 12 premature deaths from Salem Harbor and 27 from Brayton Point (compared with values of 9 and 29 using Boston meteorology). Depending on the health outcome, using the Hartford-Albany meteorological data results in a 1-25% increase in health impacts from Salem and a 3-6% decrease in health effects from Brayton. Thus, the assumed meteorological conditions appear to have a relatively small influence on the aggregate health impacts. This finding supports the assertion that a significant portion of the difference between CALPUFF and ISC-SLIM3 resides in the chemical conversion mechanism. However, it should be reiterated that this sensitivity analysis is not directly applicable to CALPUFF, since CALPUFF does not apply a single meteorological data set to the entire receptor region.

Pollutant Assumptions

One of the differences between this report and past investigations is the inclusion of condensable particulate matter along with primary particles and secondary sulfate and nitrate particles. Although the existence of this form of pollution is well-established, it is possible that our model of secondary PM_{10} is capturing some of this effect. If we assume that condensable particulate matter is completely accounted for by other categories of particulate matter, the annual premature mortality risk (using the baseline CALPUFF model) drops from 53 to 45 for Salem Harbor and from 106 to 85 for Brayton Point. Conversely, condensable particulate matter could theoretically account for a fraction of the secondary sulfate and nitrate particles, but this

would only be plausible close to the stack and would not account for a significant portion of aggregate sulfate or nitrate impacts.

Epidemiological Assumptions, Mortality

Given the significant contribution of premature mortality to the monetized health damages as well as the societal importance of this outcome, we closely investigate some of the underlying assumptions in our mortality estimates.

Our baseline estimate is based on the assumption that the chronic mortality literature has derived correct long-term impacts from particulate matter, and that this effect encompasses the entire daily mortality effect. We judged this to be a reasonable central estimate. A plausible lower bound on premature mortality would come from the assumption that the chronic mortality studies are fundamentally flawed, leaving the coefficients from the acute mortality studies. For this subset of studies, actual annual premature deaths would be estimated as 22 for Brayton Point and 11 for Salem Harbor, a significant reduction from our baseline estimates.

An upper bound on premature mortality would sum the acute and chronic mortality studies and assume that they account for completely different disease processes (one related to long-term exposure, the other to short-term changes in concentrations). Under this assumption, actual annual premature deaths would be increased to 128 for Brayton Point and 64 for Salem Harbor (versus 106 and 53 under baseline assumptions).

An additional sensitive assumption involves the allocation of mortality among pollutants. For the American Cancer Society chronic mortality study, no evidence has been collected regarding correlated gaseous pollutants that might explain some fraction of the particulate matter effect, so this effect cannot be quantitatively evaluated. For acute mortality studies, we have

assumed that the univariate coefficients from the epidemiological studies must be modified to account for correlated pollutants, and we conclude that there is some (statistically insignificant) mortality risk from SO_2 that is not accounted for by PM_{10}. Since this assumption can be disputed, we estimate acute mortality assuming that the univariate PM_{10} impact encompasses all pollutants. Under this assumption, the aggregate acute mortality damages under actual emissions change minimally for both power plants (identical values to two significant figures).

Additional Endpoints

In the aggregate, health effects may be underestimated due to the omission of key pollutants, health effects, and upstream emission sources. Because of the computational burden of ozone modeling, the marginal influence of power plant NOx emissions on ozone formation has not been included in this analysis. Including ozone could lead to significant added health effects, given epidemiological literature implicating ozone in outcomes such as premature mortality, respiratory hospital admissions, asthma attacks, and respiratory symptoms (Levy *et al.*, 1999).

To approximate the magnitude of the health effects associated with ozone, we use a simplified literature estimate correlating ozone concentrations with NO_2 concentrations. Using a smog chamber study to approximate ozone increases from NO_2 emissions (Kelly and Gunst, 1990), we estimate that including the ozone effect of NOx emissions would yield 1 additional premature fatality from Brayton Point and 0.6 additional premature fatalities from Salem Harbor. Ozone would also be anticipated to have numerous morbidity effects (including respiratory hospital admissions, asthma attacks, and respiratory symptoms), as well as impacts on visibility.

Health effects would also be increased by the inclusion of additional pollutants, such as carbon monoxide or air toxics.

In addition, including endpoints other than human health would add to the damages associated with the power plants. These damages might include outcomes that would influence human well-being (i.e., decreased visibility, damage to materials, increased soot deposition on homes) as well as outcomes that are secondarily linked to humans (ecosystem and wildlife damage). A comprehensive analysis should include multiple endpoints beyond human health. Also, although the relative importance would depend on the control strategies being implemented, climate change should be a part of any complete analysis of fuel combustion sources.

Finally, the complete assessment of the health effects of a power plant would not just consider the emissions from the power plant stack, but would also consider all of the upstream processes that are related to the fuel combustion. In other words, a reduction in the amount of coal burned will not just reduce air pollutants from the stack, but will also lessen the transportation of fuel to the power plant, the ecological damages of coal mining and the risks to coal miners, waste disposal, and a number of other processes. Of course, these upstream benefits would only be obtained through fuel switching or other reductions in electricity generation, rather than through end-use emissions controls.

Discussion and Conclusions

In summary, we have applied a state-of-the-art damage function model to two grandfathered power plants in Massachusetts, to evaluate the potential benefits from emission controls and the potential health damages associated with increased emissions. To place the

proper bounds around our aggregate estimates, we used Monte Carlo analysis to propagate uncertainties and we conducted an extensive sensitivity analysis.

One of the limitations of our analysis is the fact that the uncertainty bounds presented only consider the reported uncertainty bounds within epidemiological studies and the variability in study findings, as well as estimated uncertainties within a single dispersion model. However, factors such as model uncertainty (shown to be significant in the comparison between CALPUFF and ISC-SLIM3) were not included in the reported confidence intervals, and the uncertainty bounds should therefore be interpreted with care.

In addition, many of the sensitivity analyses were conducted on the ISC-SLIM3 model, given the difficulty in running CALPUFF across a number of scenarios. It is conceivable that differences in the model structure would imply that a factor that minimally affected ISC-SLIM3 findings might be significant within CALPUFF. However, factors such as emission rate and plant characteristics would likely have similar effects on both models, and the meteorological sensitivity analysis is relatively unimportant given the detailed CALMET file constructed for this analysis.

We were also limited by the hypothetical nature of the investigation. If specific control technologies were evaluated within our study, we could theoretically consider the risks or benefits associated with changes in production efficiency, emissions of pollutants other than PM_{10}, SO_2, and NO_2, and other impact pathways. Our findings largely reflect the (primary and secondary) particulate matter health effects under different emissions scenarios, but further investigation would be needed to conduct a comprehensive benefit-cost analysis of controls at these plants.

The hypothetical nature of the investigation, along with the relatively small number of comparable studies conducted in the past, make it difficult to validate the model findings. The Centralia investigation by Samet and colleagues (1997) is the most directly applicable comparison, given the similar model construct. As indicated above, the estimate of premature deaths per capita per ton of SO_2 emissions is identical in Centralia and in our analysis, when the radius of influence and epidemiological assumptions are equated. Since sulfates comprise a majority of particulate matter concentrations in both studies, this crude comparison provides some indication that the results of the two studies are comparable.

In addition, although many past damage studies used different dispersion models and did not incorporate chronic mortality, we can also compare our estimates of "acute" premature deaths per capita per ton of primary PM_{10} with estimates from four previous studies (ORNL/RFF, 1994; EC, 1995; Rowe *et al*., 1995; Levy *et al*., 1999). Within our study, this figure was estimated to be 2×10^{-11} for Salem and 3×10^{-11} for Brayton. In the four previous studies, the estimates ranged between 9×10^{-12} and 3×10^{-11}, with the highest estimate corresponding to the Levy study (which used the identical dose-response coefficient as our study). As above, this demonstrates relative agreement with past damage studies, despite the differences in dispersion model selection.

Through our analysis, it is apparent that many of the assumptions that had to be made to complete an investigation of this sort did not significantly affect the total health damages. The choice of air dispersion model appeared to cause the largest uncertainty, with estimates from CALPUFF anywhere from half as large at Centralia to ten times the estimates from ISC-SLIM3 at Brayton and Salem. However, it is worth recalling that the model uncertainty for primary pollutants was relatively small, indicating that aggregate health effects are likely less sensitive to

assumed meteorological patterns. The largest uncertainties arose with secondary sulfate and nitrate particles. Since the estimated effects were identical using two different CALPUFF chemical transformation models and SLIM3 treats chemical interactions in an extremely simple fashion (exponential decay with constant conversion), the CALPUFF estimates are likely more reliable.

Significant uncertainties also arise when we try to estimate the monetary damages associated with premature mortality, both because of the use of chronic mortality findings and a standard monetary value for premature mortality. Our monetary estimates are considered illustrative, and a more comprehensive consideration of life-years lost and the proper valuation of premature mortality would be needed for a thorough benefit-cost analysis.

One interesting finding from our study is that the majority of particulate matter health effects can be associated with the secondary sulfate particles rather than the primary particulate matter emissions. This finding can be supported by multiple past studies, including the Centralia investigation (Samet *et al.*, 1997). As another example, the ExternE externality analysis of coal-fired power plants found that secondary particles had 2-6 times the mortality effect of primary particles, in an analysis where the SO_2 emission rate was 4-30 times the PM_{10} emission rate (EC, 1995b).

This finding as well as the results of our analysis can be explained in part by the recent analysis of Evans and colleagues (1999). This study focused on a concept known as exposure efficiency, defined as the fraction of material released from a source that is eventually inhaled or ingested. Effectively, the health impact from a pollutant will be proportional to its exposure efficiency multiplied by its emission rate, since this represents the total amount of pollution exposure for a population. In a preliminary analysis using CALPUFF, the authors found that the

average exposure efficiency of primary particulate matter was approximately 10 times greater than that of sulfates (from SO_2 emissions) and 100 times greater than that of nitrates (from NO_2 emissions). In our study, the SO_2 emission rate of both Brayton Point and Salem Harbor is approximately 100 times greater than the primary PM_{10} emission rate, indicating that we would expect greater effects from sulfates than from primary PM_{10} in our analysis.

We conclude that decreasing the emission rate from the actual to target rates would result in estimated annual reductions of 43 premature deaths from Salem Harbor and 81 premature deaths from Brayton Point, along with reductions in numerous morbidity outcomes. Further study would be needed to quantify the complete range of costs and benefits associated with control policies, but this study provides a methodology to estimate the public health benefits if target emission rates were achieved at Salem Harbor or Brayton Point.

References

Bowers JF, Anderson AJ. *An Evaluation Study for the Industrial Source Complex (ISC) Dispersion Model*. EPA-450/4-81-002. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards: Research Triangle Park, NC, 1981.

E.H. Pecham and Associates, Inc. *Control Measure Analysis of Ozone and PM Alternatives: Methodology and Results*. EPA Contract #68-D3-0035. Prepared for the Innovative Strategies and Economics Group, Office of Air Quality and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC, July 1997.

European Commission. *ExternE: External Costs of Energy, Volume 4: Oil and Gas*. Directorate-Generale XII, Science, Research, and Development: Brussels, 1995a.

European Commission. *ExternE: External Costs of Energy, Volume 3: Coal and Lignite*. Directorate-Generale XII, Science, Research, and Development: Brussels, 1995b.

Evans J, Wolff SK, Phonboon K, Levy J, Smith K. "Exposure Efficiency: An Idea Whose Time Has Come?" Paper for the Scientific Group on Methodologies for the Safety Evaluation of Chemicals (SGOMSEC 14), November 7 to 12, 1999.

Kelly NA, Gunst RF. "Response of Ozone to Changes in Hydrocarbon and Nitrogen Oxide Concentrations in Outdoor Smog Chambers Filled with Los Angeles Air". *Atmos. Environ.* 24A: 2991-3005 (1990).

Levy JI**,** Hammitt JK, Yanagisawa Y, Spengler JD. "Development of a New Damage Function Model for Power Plants: Methodology and Applications." *Environmental Science and Technology* 33: 4364-4372 (1999).

Oak Ridge National Laboratory and Resources for the Future. *Estimating Fuel Cycle Externalities: Analytical Methods and Issues*. McGraw-Hill/Utility Data Institute: Washington, DC, 1994.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Health CW Jr. "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults." *Am. J. Respir. Crit. Care Med.* 151: 669-674 (1995).

Rowe RD, Lang CM, Chestnut LG, Latimer DA, Rae DA, Bernow SM, White DE. *The New York Electricity Externality Study Volume I: Introduction and Methods*. Empire State Electric Energy Research Corporation: New York, 1995.

Samet JM, Jodrey DJ, Xu J, Winges KD. *An Assessment of the Health Risks Due to Air Emissions from the Centralia Power Plant*. Submitted to Centralia Plant Collaborative Decision Making Group, August 1997.

U.S. Environmental Protection Agency. *The Benefits and Costs of the Clean Air Act: 1970 to 1990*. EPA 410-R-97-002. Office of Air and Radiation: Washington, D.C., 1997.

U.S. Environmental Protection Agency. *The Benefits and Costs of the Clean Air Act: 1990 to 2010*. EPA-410-R99-001. Office of Air and Radiation: Washington, D.C., 1999.

U.S. Environmental Protection Agency. *A Comparison of CALPUFF with ISC3*. EPA-454/R-98-020. Office of Air Quality Planning and Standards: Research Triangle Park, NC, 1998.

Figures are available on the Harvard School of Public Health web server as MS Word graphic files. In order to view the figures at the links below you must have MS Word 97 or later installed on your computer.

Figure 1.

Location of Brayton Point and Salem Harbor Power Plants.
(http://www.hsph.harvard.edu/papers/plant/plant_figure1.doc)

Figure 2.

Receptor Grid Used in Baseline Dispersion Model.
(http://www.hsph.harvard.edu/papers/plant/plant_figure2.doc)

Figure 3.

Primary PM10 Concentrations, Actual Emissions, Salem Harbor (Annual Average, ug/m3).
(http://www.hsph.harvard.edu/papers/plant/plant_figure3.doc)

Figure 4.

Primary PM10 Concentrations, Actual Emissions, Brayton Point (Annual Average, ug/m3).
(http://www.hsph.harvard.edu/papers/plant/plant_figure4.doc)

Figure 5.

Concentration Profile for Secondary Particulate Impacts (Ammonium Nitrate and Ammonium Sulfate Particles), Actual Emissions, Salem Harbor (Annual Average, ug/m3).
(http://www.hsph.harvard.edu/papers/plant/plant_figure5.doc)

Figure 6.

Concentration Profile for Secondary Particulate Impacts (Ammonium Nitrate and Ammonium Sulfate Particles), Actual Emissions, Brayton Point (Annual Average, ug/m3).
(http://www.hsph.harvard.edu/papers/plant/plant_figure6.doc)

Figure 7.

Geographic Distribution of Per Capita Annual Mortality Risks from Actual Salem Harbor Emissions.
(http://www.hsph.harvard.edu/papers/plant/plant_figure7.doc)

Figure 8.

Geographic Distribution of Per Capita Annual Mortality Risks from Actual Brayton Point Emissions.
http://www.hsph.harvard.edu/papers/plant/plant_figure8.doc

Table 1.
Characteristics of Salem Harbor and Brayton Point Power Plants.

	Salem	**Brayton**
Nameplate capacity (MW)	805	1611
Net generation (MWh, 1998)	3,900,083	8,936,579
Fuel consumption (1997)		
Bituminous coal	947,233 short tons	3,141,629 short tons
Heavy fuel oil (#6)	2,871,735 bbl.	405,622 bbl.
Light fuel oil (#2)	-	2,144 bbl.
Natural gas	-	3,941,322 MCF

Table 2.
Modeled Stack Parameters and SO_2, NO_x, and PM_{10} Emission Rates
for the Brayton Point Power Plant (Maximum Potential, Target, and Average).

Brayton Stacks	Potential Emission Rates (g/sec)			Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
	SO2	NOx	PM10				
UNIT1	687.0	108.0	15.2	107.2	398	25.06	4.42
UNIT2	687.0	108.0	15.2	107.2	407	26.56	4.42
UNIT3	1726.0	321.0	38.2	107.2	402	35.43	5.94
UNIT4	1465.0	163.0	12.2	152.4	469	33.47	5.64

Brayton Stacks	Target Emission Rates (g/sec)			Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
	SO2	NOx	PM10				
UNIT1	70.4	35.2	2.3	107.2	398	25.06	4.42
UNIT2	72.2	36.1	2.4	107.2	407	26.56	4.42
UNIT3	162.5	81.3	5.4	107.2	402	35.43	5.94
UNIT4	29.0	14.5	1.0	152.4	469	33.47	5.64

Brayton Stacks	Average Actual Emission Rates (g/sec)			Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
	SO2	NOx	PM10				
UNIT1	301.3	86.3	5.1	107.2	398	25.06	4.42
UNIT2	308.7	92.7	2.7	107.2	407	26.56	4.42
UNIT3	767.0	262.7	4.3	107.2	402	35.43	5.94
UNIT4	400.0	80.3	3.1	152.4	469	33.47	5.64

Brayton Stacks	Potential Condensable Emission Rates (g/sec)	Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
UNIT1	25.8	107.2	398	25.06	4.42
UNIT2	25.8	107.2	407	26.56	4.42
UNIT3	64.9	107.2	402	35.43	5.94
UNIT4	6.1	152.4	469	33.47	5.64

Brayton Stacks	Target Condensable Emission Rates (g/sec)	Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
UNIT1	4.7	107.2	398	25.06	4.42
UNIT2	4.8	107.2	407	26.56	4.42
UNIT3	10.8	107.2	402	35.43	5.94
UNIT4	1.0	152.4	469	33.47	5.64

Brayton Stacks	Actual Condensable Emission Rates (g/sec)	Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
UNIT1	25.6	107.2	398	25.06	4.42
UNIT2	25.3	107.2	407	26.56	4.42
UNIT3	62.8	107.2	402	35.43	5.94
UNIT4	5.5	152.4	469	33.47	5.64

Table 3.
Modeled Stack Parameters and SO$_2$, NO$_x$, and PM$_{10}$ Emission Rates
for the Salem Harbor Power Plant (Maximum Potential, Target, and Average).

Salem Stacks	Potential Emission Rates (g/sec)			Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
	SO2	NOx	PM10				
UNIT4	1465.0	169.0	16.2	152.4	446	34.65	5.64
UNIT5	1104.0	151.0	36.7	135.6	413	25.66	5.44

Salem Stacks	Target Emission Rates (g/sec)			Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
	SO2	NOx	PM10				
UNIT4	78.0	39.0	2.6	152.4	446	34.65	5.64
UNIT5	95.3	47.7	3.2	135.6	413	25.66	5.44

Salem Stacks	Average Actual Emission Rates (g/sec)			Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
	SO2	NOx	PM10				
UNIT4	866.7	123.7	7.3	152.4	446	34.65	5.64
UNIT5	450.7	128.0	5.3	135.6	413	25.66	5.44

Salem Stacks	Potential Condensable Emission Rates (g/sec)	Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
UNIT4	6.1	152.4	446	34.65	5.64
UNIT5	41.5	135.6	413	25.66	5.44

Salem Stacks	Target Condensable Emission Rates (g/sec)	Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
UNIT4	2.6	152.4	446	34.65	5.64
UNIT5	6.4	135.6	413	25.66	5.44

Salem Stacks	Actual Condensable Emission Rates (g/sec)	Source Height (m)	Exit Temperature (K)	Exit Velocity (m/sec)	Stack Inner Diameter (m)
UNIT4	5.2	152.4	446	34.65	5.64
UNIT5	39.5	135.6	413	25.66	5.44

Table 4.

Central Estimates for Increase in Morbidity Effects Per 10 μg/m^3 Increase in 24-Hour Average PM$_{10}$ Concentrations.

Health Outcome	% Increase
Chronic bronchitis, age 25+	8.2%
Respiratory hospital admissions, all ages	1.0%
Cardiovascular hospital admissions, age 65+	0.6%
Emergency room visits, asthma, all ages	4.3%
Emergency room visits, non-asthma, all ages	0.8%
Asthma attacks, all ages	3.1%
Restricted activity days, age 18+	2.9%
Minor restricted activity days, age 18+	4.4%
Upper respiratory symptoms, age 18+	6.8%
Upper respiratory symptoms, age < 18	1.6%
Lower respiratory symptoms, age < 18	3.1%

Table 5.

Population-Weighted Average Concentrations ($\mu g/m^3$) Estimated for Salem Harbor and Brayton Point – Maximum Potential, Actual, and Target Emission Rates.

	Salem Harbor			Brayton Point		
	Potential	Actual	Target	Potential	Actual	Target
Annual average SO_2	0.41	0.20	0.029	0.77	0.31	0.063
Annual average NO_2	0.045	0.036	0.013	0.11	0.082	0.029
Annual average PM_{10} – filterable	0.010	0.0022	0.0011	0.016	0.0033	0.0024
Annual average PM_{10} – condensable	0.0099	0.0093	0.0018	0.026	0.025	0.0046
Annual average particulate sulfate (as ammonium sulfate)	0.084	0.043	0.0063	0.18	0.075	0.016
Annual average particulate nitrate (as ammonium nitrate)	0.010	0.0080	0.0029	0.027	0.021	0.0069

Table 6.

Predicted Annual Health Impacts from Salem Harbor and Brayton Point Power Plants – Maximum Potential, Actual, and Target Emission Rates (Mean and 95% Confidence Intervals) [1].

	Salem Harbor			Brayton Point		
	Potential	Actual	Target	Potential	Actual	Target
Premature deaths	97 (54, 140)	53 (29, 76)	10 (6, 15)	210 (120, 310)	106 (60, 150)	25 (14, 36)
Chronic bronchitis	100 (12, 190)	57 (7, 100)	11 (1, 20)	230 (29, 420)	115 (15, 210)	27 (3, 49)
Respiratory hospital admissions	50 (9, 88)	27 (5, 48)	5 (1, 9)	110 (20, 200)	55 (10, 98)	13 (2, 23)
Cardiovascular hospital admissions	29 (21, 37)	16 (11, 20)	3 (2, 4)	64 (46, 82)	32 (23, 41)	8 (5, 10)
Net emergency room visits	1,000 (560, 1,400)	570 (300, 790)	110 (58, 150)	2,300 (1,300, 3,200)	1,140 (640, 1,600)	270 (150, 370)
Asthma attacks	27,000 (21, 52,000)	14,400 (12, 28,000)	2,800 (2, 5,400)	58,000 (NS, 120,000)	28,900 (NS, 57,000)	6,900 (NS, 14,000)
Net restricted activity days	31,000 (2,900, 57,000)	16,700 (1,600, 31,000)	3,200 (320, 6,000)	67,000 (4,400, 130,000)	33,500 (2,200, 63,000)	7,900 (530, 15,000)
Net minor restricted activity days	51,000 (21,000, 82,000)	28,100 (11,000, 45,000)	5,400 (2,200, 8,600)	110,000 (33,000, 170,000)	56,000 (16,000, 87,000)	13,000 (3,800, 21,000)
Net upper respiratory symptom days, adults	180,000 (30,000, 320,000)	99,000 (16,000, 180,000)	19,000 (3,100, 34,000)	400,000 (66,000, 710,000)	199,000 (33,000, 350,000)	47,000 (7,800, 84,000)
Lower respiratory symptom days, children	6,800 (NS, 18,000)	4,500 (NS, 10,000)	1,300 (3, 2,500)	15,000 (NS, 37,000)	9,700 (NS, 21,000)	2,900 (NS, 6,000)
Upper respiratory symptom days, children	7,900 (NS, 21,000)	4,300 (NS, 11,000)	830 (NS, 2,200)	18,000 (NS, 47,000)	8,800 (NS, 24,000)	2,100 (NS, 5,600)

[1] Estimated using CALPUFF with the RIVAD/ARM3 chemical conversion mechanism and using the American Cancer Society study (Pope *et al.*, 1995) for premature deaths. 95% confidence intervals are generated using Monte Carlo analysis on estimated CALPUFF model uncertainty and standard errors reported in epidemiological studies or estimated through random effects models. NS = non-significant.

Table 7.
Annual Health Benefits from Achieving Target Emission Rates at Salem Harbor and Brayton Point Power Plants (Actual Minus Target Emissions, Mean and 95% Confidence Intervals) [1].

	Salem Harbor	Brayton Point
Premature deaths	43 (24, 61)	81 (45, 120)
Chronic bronchitis	46 (5, 84)	88 (11, 160)
Respiratory hospital admissions	22 (4, 39)	41 (8, 75)
Cardiovascular hospital admissions	13 (9, 16)	25 (17, 31)
Net emergency room visits	460 (250, 640)	870 (490, 1,200)
Asthma attacks	11,600 (9, 23,000)	22,000 (NS, 44,000)
Net restricted activity days	13,500 (1,300, 25,000)	25,600 (1,700, 48,000)
Net minor restricted activity days	22,700 (9,200, 36,000)	43,000 (12,000, 66,000)
Net upper respiratory symptom days, adults	80,000 (13,000, 140,000)	152,000 (25,000, 270,000)
Lower respiratory symptom days, children	3,200 (NS, 8,000)	6,800 (NS, 15,000)
Upper respiratory symptom days, children	3,500 (NS, 9,200)	6,700 (NS, 18,000)

[1] Estimated using CALPUFF with the RIVAD/ARM3 chemical conversion mechanism and using the American Cancer Society study (Pope *et al.*, 1995) for premature deaths. 95% confidence intervals are generated using Monte Carlo analysis on estimated CALPUFF model uncertainty and standard errors reported in epidemiological studies or estimated through random effects models. NS = non-significant.

Table 8.
Geographic Distribution of Annual Health Impacts from Salem Harbor and Brayton Point Power Plants, Actual Emissions (Mean and 95% Confidence Intervals) [1].

	Salem Harbor		Brayton Point	
	< 50 km	> 50 km	< 50 km	> 50 km
Premature deaths	17 (9, 24)	36 (20, 52)	16 (9, 23)	91 (51, 130)
Chronic bronchitis	18 (2, 33)	39 (5, 71)	17 (2, 31)	98 (12, 180)
Respiratory hospital admissions	9 (2, 15)	19 (3, 33)	8 (1, 15)	47 (8, 83)
Cardiovascular hospital admissions	5 (4, 6)	11 (8, 14)	5 (4, 7)	27 (19, 34)
Net emergency room visits	180 (95, 250)	390 (210, 540)	170 (96, 240)	970 (550, 1,300)
Asthma attacks	4,500 (4, 8,900)	9,900 (8, 19,000)	4,400 (NS, 8,600)	24,500 (NS, 49,000)
Net restricted activity days	5,500 (700, 10,000)	11,200 (930, 21,000)	4,900 (260, 9,300)	28,600 (2,000, 53,000)
Net minor restricted activity days	9,100 (3,800, 15,000)	19,000 (7,500, 30,000)	8,300 (2,300, 13,000)	48,000 (14,000, 74,000)
Net upper respiratory symptom days, adults	32,000 (5,200, 57,000)	67,000 (11,000, 120,000)	30,000 (4,900, 53,000)	170,000 (28,000, 300,000)
Lower respiratory symptom days, children	2,000 (34, 3,900)	2,500 (NS, 6,700)	2,600 (NS, 4,900)	7,200 (NS, 16,000)
Upper respiratory symptom days, children	1,200 (NS, 3,300)	3,100 (NS, 8,100)	1,400 (NS, 3,700)	7,500 (NS, 20,000)

[1] Estimated using CALPUFF with the RIVAD/ARM3 chemical conversion mechanism and using the American Cancer Society study (Pope *et al.*, 1995) for premature deaths. 95% confidence intervals are generated using Monte Carlo analysis on estimated CALPUFF model uncertainty and standard errors reported in epidemiological studies or estimated through random effects models. NS = non-significant.

Table 9.
Monetary Valuation of Health Impacts Under Different Emission Scenarios [1].

	Salem	Brayton
Potential	$630M	$1400M
Actual	$350M	$700M
Target	$70M	$160M
Actual – Target	$280M	$530M

[1] Estimated using CALPUFF with the RIVAD/ARM3 chemical conversion mechanism and using the American Cancer Society study (Pope *et al.*, 1995) for premature deaths. The value of a statistical life was taken from a recent EPA benefit-cost analysis (EPA, 1997), and the values of morbidity outcomes were derived from the study by Levy and colleagues (1999).

Table 10.
Categories of Damage Function Uncertainty and Modeling Choices.

Uncertainty	Approach
Model construction	
Receptor selection (geographic range considered)	Qualitative discussion, estimation of aggregate impacts with different radii
Plant characteristics	Use two sources of plant information
Air pollution modeling	
Meteorological data	For ISC-SLIM3, use alternative (non-coastal) meteorological data
Model selection	Use both CALPUFF and ISC-SLIM3
Health modeling	
Mortality	Consider range of possibilities: chronic only, acute only, chronic + acute, with/without methodology to control for correlated pollutant confounding
Boundary decisions	
Additional endpoints	Estimate potential health impacts of ozone; discuss other potential outcomes (non-health) and pollutants (water pollution)

Table 11.

Comparison between CALPUFF and ISC-SLIM3 Model Results for Brayton Point, Salem Harbor, and Centralia (population-weighted annual average $\mu g/m^3$, actual emissions).

		CALPUFF	ISC-SLIM3	Ratio (ISC-SLIM3/ CALPUFF)
Salem	SO_2	0.20	0.09	0.5
	SO_4 particles	0.043	0.0076	0.2
	NO_2	0.036	0.016	0.4
	NO_3 particles	0.008	0.00039	0.05
	Filterable PM_{10}	0.0022	0.00084	0.4
	Condensable PM_{10}	0.0093	0.0034	0.4
	Total PM	0.062	0.012	0.2
Brayton	SO_2	0.31	0.20	0.6
	SO_4 particles	0.075	0.019	0.3
	NO_2	0.082	0.054	0.7
	NO_3 particles	0.021	0.0015	0.07
	Filterable PM_{10}	0.0033	0.0017	0.5
	Condensable PM_{10}	0.025	0.013	0.5
	Total PM	0.12	0.035	0.3
Centralia	SO_2	0.68	0.88	1.3
	SO_4 particles	0.06	0.13	2.1
	NO_2	0.15	0.20	1.4
	NO_3 particles	0.016	0.038	2.4
	Total PM	0.11	0.17	1.6

Appendix 1.

Summary of Meteorological Processing for CALMET

Derived from Report Provided by:

Dennis Moon, Ph.D.
Chief Scientist, SSESCO

Our system for production of meteorological data sets for air quality studies is based on utilization of the most comprehensive data sets available, combined with the latest assimilation techniques. Prognostic models are well known to have significant advantages over diagnostic windfield models. The chief advantage lies in the imposition of dynamic constraints to the system, in addition to the static constraints applied in diagnostic models, such as mass conservation and hydrostatic. Dynamic constraints are those resulting from the application of conservation laws which involve time derivatives, such as conservation of momentum. The chief drawback of prognostic models is the computational expense of running them. Computational stability considerations require that the models be stepped forward with a timestep that is proportional to the grid cell size. Thus high-resolution grids require an extremely large number of time steps to be computed to cover the needs of a long-term air quality study. For this reason high resolution prognostic models are most often applied to episodic case studies.

While the application of customized prognostic meteorological models to long-term air quality studies can be prohibitively expensive, data from NOAA prognostic model outputs and analyses can be combined with mesoscale data assimilation systems to produce high resolution data sets of long duration. NOAA runs a suite of models at varying initial times, resolutions, domains of coverage, and forecast duration. Each model run starts with results from a previous run, combined with all available observed data, including surface and upper air observations, satellite, and radar data. This process of combining the various data sources to yield a unified representation of the three-dimensional atmosphere is termed assimilation. This has been an area of active research over the years, as increasingly accurate analyses, combining more data types is one of the principal means for improving forecast quality. We can reap the benefits of this research by basing the production of our "digital atmosphere" on these NOAA analyses.

Of prime interest to us is NOAA's RUC2 model. RUC stands for Rapid Update Cycle. This is a short-term forecast model that is rerun very frequently. Of importance to us is the fact that the model is re-initialized each hour. The model grid is of 40 km spacing, with over 40 layers in the vertical. This resolution is sufficient to easily represent the upper air feature captured by the radiosonde network. For over two years, SSESCO has been archiving these RUC2 analyses for the purpose of applying the data to air quality studies.

One drawback in applying the RUC2 data directly to air quality studies is that a 40 km grid is typically not of high enough resolution to capture all of the relevant flow and thermal structures that arise near the earth's surface. For this purpose we need to introduce high-resolution terrain data and surface observations. This is done using a mesoscale assimilation system. While NOAA has been advancing the assimilation and modeling process as applied to synoptic scale weather systems, a parallel effort in mesoscale modeling systems has been proceeding at a number of governmental and educational research institutions. Foremost among those efforts has been the work done at the Center for the Analysis and Predictions of Storms (CAPS), at the University of Oklahoma. This group, founded by NSF and the FAA, is focused on research and the development of software tools related to small-scale weather phenomenon. We have chosen the ARPS Data Assimilation System (ADAS), as our primary mesoscale assimilation tool. The ADAS system starts with a first-guess field derived from NOAA model data and then reads in observational data (surface, upper air, satellite, radar) and performs climatalogical, spatial, and temporal continuity checking for invalid data. The key to the assimilation process is the blending of different data sources, each with their own error characteristics into a unified, "most probable" three-dimensional distribution of the target variable. Taking into account the error characteristics of the first-guess gridded data and each of

the observational sources, ADAS performs an objective analysis onto the target model grid. ADAS uses a highly efficient iterative approach to the widely used Statistical or Optimal Interpolation (OI) technique, known as the Bratseth technique. Mass conservation and boundary conditions are then applied to derive the vertical motion fields.

The datasets developed by this system can be input into CALMET using its ability to ingest MM5 fields and interpolate them to the CALMET grid. For this study a grid was developed to cover the domain of interest at a cell size of 15 km. The grid uses a Lambert Conformal projection. It is anticipated that the CALMET run will employ the same horizontal grid projection and spacing to minimize interpolation errors. The grid has fourteen vertical levels, going up to about 5100m AGL. The vertical grid spacing is stretched from about 20m near the ground to 600m near the top of the domain. While we did not anticipate that the CALMET grid performed used in the CALPUFF analysis would require this many levels, we considered it best for CALMET to vertically interpolate from a higher to lower resolution grid. The grid structure and terrain can be seen in the figure on the following page.
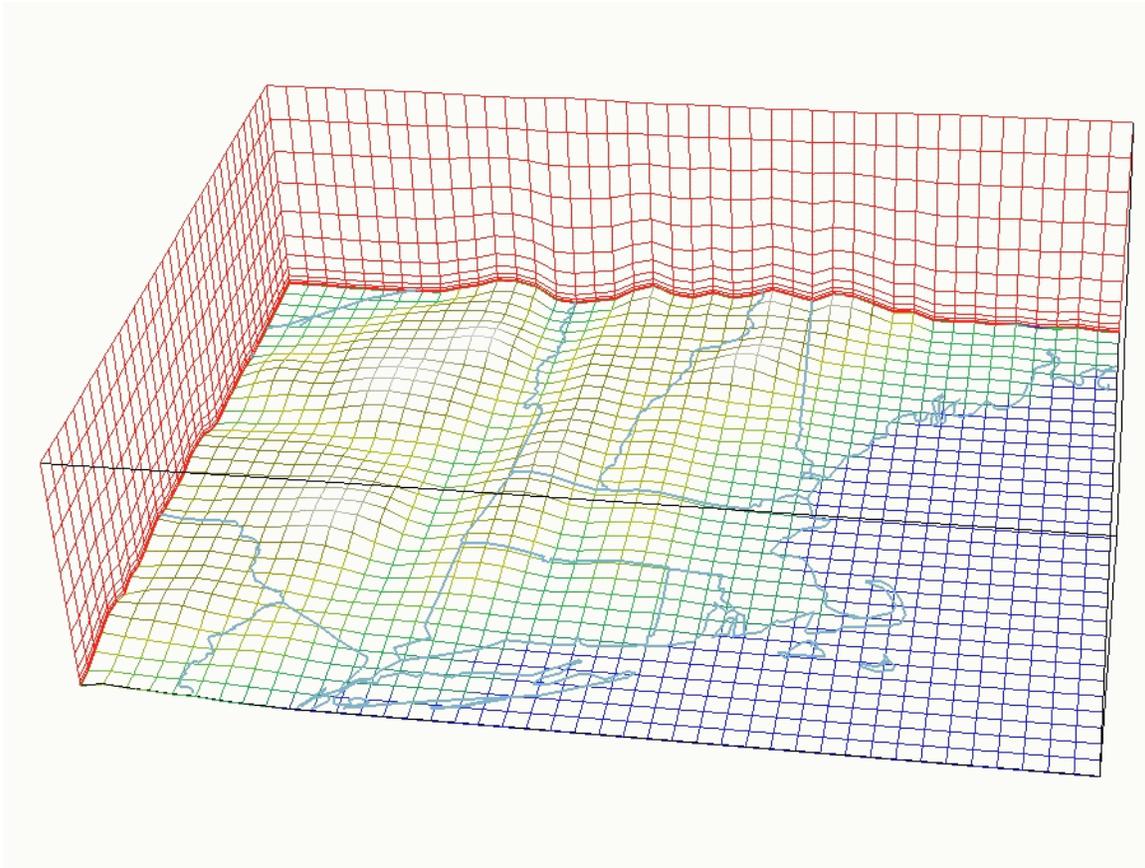
For each hour in the yearlong study, an ADAS analysis was performed using the RUC analyses for a first guess field and combining it with the metar surface observations. The RUC gridded analyses already capture the upper air information from the radiosondes, in addition to other, more recent, data sources such as upper level winds determined from satellite imagery analysis, VHF radio sounders, and ACARS aircraft reported wind and temperature data. The assimilation of the surface data allows us to recapture high resolution information lost to the 40 km grid, and to re-compute mass conservation in the presence of the higher resolution 15 km terrain. Temperature, pressure, wind, and humidity fields were computed using the Bratseth implementation of the Optimal Interpolation algorithm. In addition, metar reports of fractional cloud coverage were analyzed to create a gridded cloud coverage field. Since the output from ADAS already incorporates the observations at the scale of the CALMET grid, it will not be

necessary to re-introduce the same data in the CALMET processing. The role of CALMET will simply be to perform the CALMET terrain adjustment and to calculate the micrometeorological parameters used by CALPUFF. If a precipitation analysis is required, this will also be performed by CALMET. The ADAS output fields were written in the format used by CALMET for ingesting MM5 data, the so-called 'MM5.dat' format. This is an ASCII format, which is quite inefficient in terms of disk space. In order to keep the files sizes manageable, the year was broken up into 24 periods, each of which is covered by a data file. The cloud coverage data was written to separate ASCII files in the format used by CALMET for ingesting this data. In all cases the CALMET source code used to read the data was checked to ensure that the files were in the correct format. For hours with missing RUC2 analyses, time interpolation was performed on the preceding and following RUC2 data to generate a first guess field, and then the assimilation was performed using the metar data. The following images show outputs from the system including surface level wind streamlines and contoured fractional cloud coverage.

Wind Steamlines   01/16/2000   00:00:0.00GMT
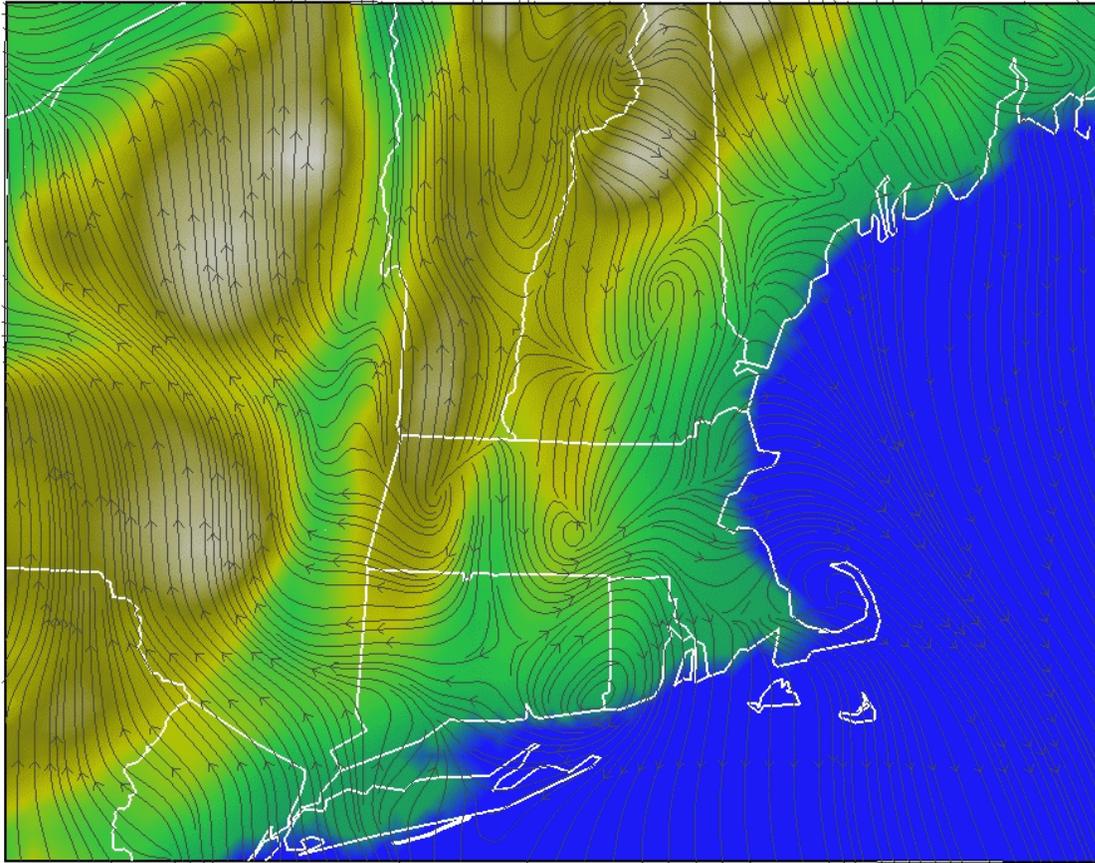
Cloud Coverage 01/16/2000 00:00:0.00GMT
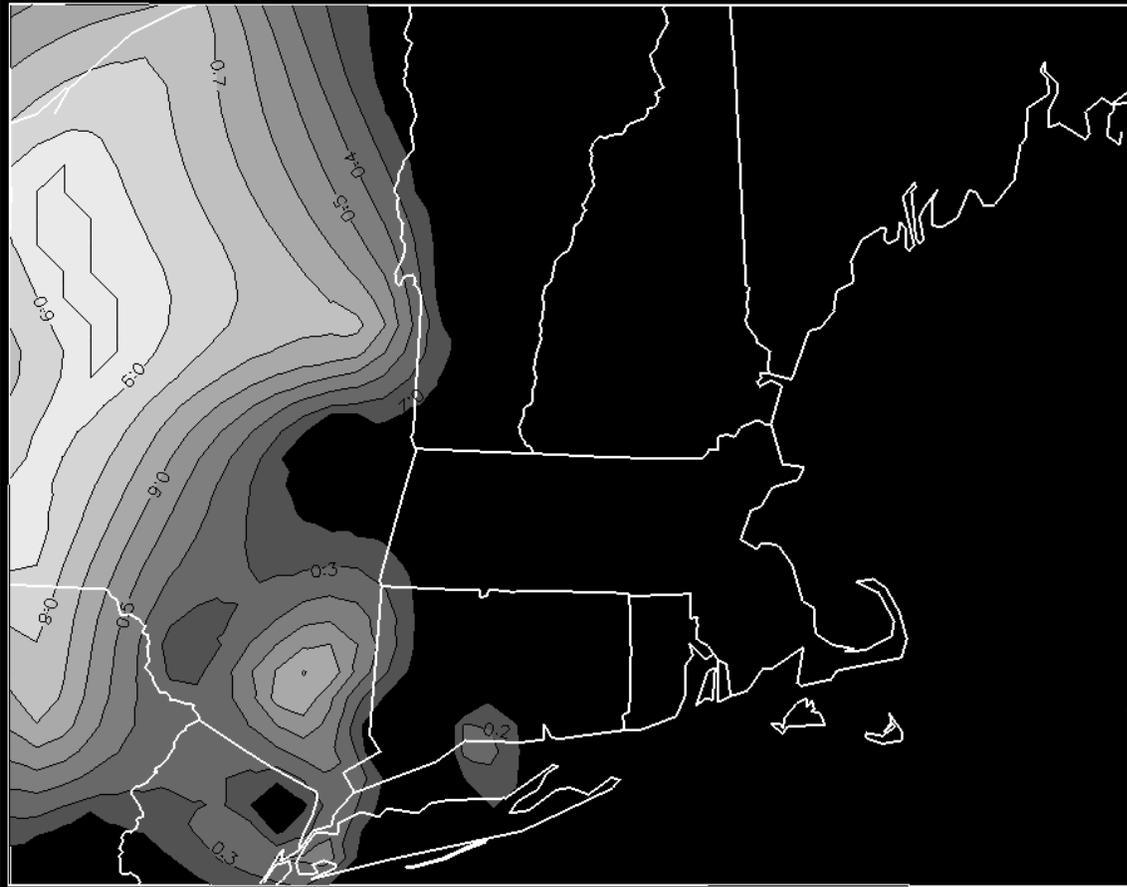
Appendix 2.

Morbidity Outcomes Evaluated in Damage Function Model

Adapted from Supporting Information for "Development of a New Damage Function Model for Power Plants: Methodology and Applications"

Published in Environmental Science and Technology, December 1999.

Chronic bronchitis

In a cohort of non-smoking adults, a 45 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a relative risk of 1.81 for chronic bronchitis (95% CI: 0.98, 3.25) [1]. In this study, $PM_{2.5}$ concentrations were estimated from visibility data by developing regression equations based on a subset of observations. Converting to PM_{10}, we estimate an 8.2% increase in chronic bronchitis for a 10 $\mu g/m^3$ increase (95% CI: -0.1%, 17.2%). No significant relationship is reported with O_3, NO_2, or SO_2. We assume a chronic bronchitis prevalence of 5.0% [2] and an incidence/ prevalence ratio of 1/9.3 [3], yielding an annual incidence of 0.53% and a corresponding change in additional cases due to daily air pollution exposure.

Respiratory hospital admissions

We focus on the effects of PM_{10} and O_3 on respiratory hospital admissions (RHA) for all ages, since the two studies evaluating NO_2 and SO_2 found no significant relationship [4,5]. For these two studies and four others [6-9], we calculate pooled single-pollutant estimates of 1.4% for PM_{10} and 0.6% for O_3. Among studies with multi-pollutant analyses, the pooled effect estimate is reduced from 1.3% to 0.9% for PM_{10}, and from 2.0% to 1.4% for O_3, indicating some confounding. Applying these reductions to the single-pollutant estimates yields final estimates of 1.0% (95% CI: 0.0%, 1.9%) for PM_{10} and 0.4% (95% CI: 0.0%, 0.8%) for O_3. We estimate the change in RHA using a baseline rate of 1,351 RHA/100,000 people/year for all respiratory diseases less tonsillitis [10].

Cardiovascular hospital admissions

For the four studies of cardiovascular hospital admissions (CHA) in elderly populations [9,11-13], there is evidence of a relationship with PM_{10}, NO_2, SO_2, and CO. For SO_2 and NO_2, the effects are completely removed in multi-pollutant models. For a 1 ppm increase in CO, the pooled effect estimate is 1.9% in single-pollutant models, reduced from 1.7% to 1.6% in multi-pollutant models controlling for PM_{10}. For PM_{10}, the pooled single-pollutant estimate is 0.8%, with a reduction from 1.1% to 0.8% in the multi-pollutant studies. Thus, our final effect estimates are 0.6% for a 10 $\mu g/m^3$ increase in PM_{10} (95% CI: 0.4%, 0.8%) and 1.8% for a 1 ppm increase in CO (95% CI: 0.4%, 3.3%). We estimate the change in CHA given a baseline rate of 9,905 CHA/100,000 people/year among people age 65 or older [10].

Emergency room visits

Studies of emergency room visits (ERV) have considered both asthma-related and all-cause visits. For asthma ERV, the two studies with effect estimates for multiple pollutants [14,15] find only PM_{10} to be significant, with a pooled estimate of 4.3% (95% CI: 1.3%, 7.3%). Other studies that find a relationship with O_3 [16,17] did not adequately consider confounding. For all-cause ERV, PM_{10} is associated with a 0.8% increase (95% CI: 0.2%, 1.4%), with no significance for gaseous pollutants [18]. Given 1.9 million asthma ERV and 88 million non-asthma ERV each year in the US [19] and a 5% prevalence of diagnosed asthma [20], we quantify the increases in ERV. As in previous studies [21,22], we make the conservative assumption that all hospital admissions involve an ERV. We prevent double-counting by subtracting hospital admissions from ERV, yielding net ERV.

Asthma attacks

We consider studies of either increased bronchodilator use or increased asthma attacks for both adults [23-26] and children [24,27,28]. Since the effects of PM_{10} are similar, we combine these populations to yield an overall estimate of 3.1% (95% CI: -0.6%, 6.9%). Only one study [23] measured O_3, finding a 0.8% increase (95% CI: 0.2%, 1.5%). We assume that asthmatics have a daily asthma attack frequency of 13% [22], yielding our estimated increased attack rates.

Restricted activity days

For restricted activity days (RAD), we pool multiple estimates from a study of the Health Interview Survey [29] to yield a 2.9% increase in RAD (95% CI: 1.7%, 4.0%) for PM_{10}. A follow-up study [30] found that minor restricted activity days (MRAD) were related to both PM_{10} (4.4%; 95% CI: 3.3%, 5.6%) and high-hour O_3 (0.3%; 95% CI: -0.5%, 1.2%). To calculate the overall changes, we use baseline rates of 6.6 RAD/person/year and 6.3 MRAD/person/year [2,30]. We estimate net RAD by subtracting all hospital days, net ERV, and asthma attacks, and we estimate net MRAD by subtracting asthma attacks [21,22].

Acute symptoms

For other acute symptoms, we consider aggregate upper or lower respiratory symptoms (URS or LRS) to be proxies for all endpoints. For adults, a study in California found no associations with LRS, but found a 6.8% increase in URS (95% CI: 2.3%, 11.5%) for PM_{10}, and a 1.0% increase in URS (95% CI: 0.5%, 1.5%) for high-hour O_3 [31]. We estimate net URS by subtracting asthma attacks and assuming a baseline incidence of 3.7%.

For children, the studies of URS and LRS find some relationship with SO_2, NO_2, O_3, and PM_{10} [24,32-35]. We assume from the estimates reported above and an analysis of cough [34] that SO_2

impacts are related to correlated PM_{10}. We assume causality for NO_2, since studies have linked indoor NO_2 with LRS [36] and little confounding was found for cough. For LRS, the pooled estimates are 3.1% for PM_{10} (95% CI: -7.4%, 13.6%), 3.1% for O_3 (95% CI: -9.7%, 15.9%), and 6.2% for NO_2 (95% CI: -0.7%, 13.0%). For URS, the pooled estimate for PM_{10} is 1.6% (95% CI: -2.8%, 6.0%), with other pollutants insignificant. Baseline incidence rates of 0.4% for LRS and 1.6% for URS [35] are used to yield the estimated health outcomes.

References, Appendix 3

1. Abbey, D.E.; Lebowitz, M.D.; Mills, P.K.; Petersen, F.F.; Beeson, W.L.; Burchette, R.J. *Inhalation Toxicol.* **1995**, *7*, 19-34.

2. Adams, P.F.; Marano, M.A. *Current estimates from the National Health Interview Survey, 1994*; National Center for Health Statistics; Vital Health Stat. 10 No. 193, 1995.

3. Steering Committee on Future Health Scenarios (SCFHS). *Chronic diseases in the year 2005: scenario report*; Translated by Arriens, J.W. Kluwer Academic Publishers: Boston, 1991.

4. Thurston, G.D.; Ito, K.; Hayes, C.G.; Bates, D.V.; Lippmann, M. *Environ. Res.* **1994**, *65*, 271-290.

5. Anderson, H.R.; Spix, C.; Medina, S.; Schouten, J.P.; Castellsague, J.; Rossi, G.; Zmirou, D.; Touloumi, G.; Wojtyniak, B.; Ponka, A.; Bacharova, L.; Schwartz, J.; Katsouyanni, K. *Eur. Respir. J.* **1997**, *10*, 1064-1071.

6. Pope, C.A. III. *Arch. Environ. Health.* **1991**, *46*, 87-97.

7. Thurston, G.D.; Ito, K.; Kinney, P.L.; Lippmann, M. *J. Expos. Anal. Environ. Epidemiol.* **1992**, *2*, 429-450.

8. Delfino, R.J.; Becklake, M.R.; Hanley, J.A. *Environ. Res.* **1994**, *67*, 1-19.

9. Burnett, R.T.; Dales, R.; Krewski, D.; Vincent, R.; Dann, T.; Brook, J.R. *Am. J. Epidemiol.* **1995**, *142*, 15-22.

10. Gillum, B.S.; Graves, E.J.; Wood, E. *National Hospital Discharge Survey: Annual summary, 1995*; National Center for Health Statistics, Vital Health Stat. 13 No. 133, 1998.

11. Morris, R.D.; Naumova, E.N.; Munasinghe, R.L. *Am. J. Pub. Health* **1995**, *85*, 1361-1365.

12. Schwartz, J.; Morris, R. *Am. J. Epidemiol.* **1995**, *142*, 23-35.

13. Schwartz, J. *Epidemiology* **1997**, *8*, 371-377.

14. Schwartz, J.; Slater, D.; Larson, T.V.; Pierson, W.E.; Koenig, J.Q. *Am. Rev. Respir. Dis.* **1993**, *147*, 826-831.

15. Lipsett, M.; Hurley, S.; Ostro, B. *Environ. Health Perspect*. **1997**, *105*, 216-222.

16. Weisel, C.P.; Cody, R.P.; Lioy, P.J. *Environ. Health Perspect*. **1995**, *103*, 97-102.

17. Yang, W.; Jennison, B.L.; Omaye, S.T. *Inhalation Toxicol.* **1997**, *9*, 15-29.

18. Samet, J.M.; Speizer, F.E.; Bishop, Y.; Spengler, J.D.; Ferris, B.G. Jr. *J. Air Pollut. Control Assoc.* **1981**, *31*, 236-240.

19. Schappert, S.M. *Ambulatory care visits to physician offices, hospital outpatient departments, and emergency departments: United States, 1995*; National Center for Health Statistics; Vital Health Stat. 13 No. 134, 1998.

20. Benson, V.; Marano, M.A. *Current estimates from the National Health Interview Survey, 1992*; National Center for Health Statistics; Vital Health Stat. 10 No. 189, 1994.

21. Oak Ridge National Laboratory and Resources for the Future. *Estimating Fuel Cycle Externalities: Analytical Methods and Issues*. McGraw-Hill/Utility Data Institute: Washington, D.C., 1994.

22. Rowe, R.D.; Lang, C.M.; Chestnut, L.G.; Latimer, D.A.; Rae, D.A.; Bernow, S.M.; White, D.E. *The New York Electricity Externality Study Volume I: Introduction and Methods*; Empire State Electric Energy Research Corporation: New York, 1995.

23. Whittemore, A.S.; Korn, E.L. *Am. J. Pub. Health* **1980**, *70*, 687-696.

24. Pope, C.A. III; Dockery, D.W.; Spengler, J.D.; Raizenne, M.E. *Am. Rev. Respir. Dis.* **1991**, *144*, 668-674.

25. Dusseldorp, A.; Kruize, H.; Brunekreef, B.; Hofschreuder, P.; de Meer, G.; van Oudvorst, A.B. *Am. J. Respir. Crit. Care Med.* **1995**, *152*, 1932-1939.

26. Delfino, R.J.; Zeiger, R.S.; Seltzer, J.M.; Street, D.H.; Matteucci, R.M.; Anderson, P.R.; Koutrakis, P. *Environ. Health Perspect*. **1997**, *105*, 622-634.

27. Roemer, W.; Hoek, G.; Brunekreef, B. *Am. Rev. Respir. Dis.* **1993**, *147*, 118-124.

28. Peters, A.; Dockery, D.W.; Heinrich, J.; Wichmann, H.E. *Eur. Respir. J.* **1997**, *10*, 872-879.

29. Ostro, B.D. *J. Environ. Econ. Manag.* **1987**, *14*, 87-98.

30. Ostro, B.D.; Rothschild, S. *Environ. Res.* **1989**, *50*, 238-247.

31. Ostro, B.D.; Lipsett, M.J.; Mann, J.K.; Krupnick, A.; Harrington, W. *Am. J. Epidemiol.* **1993**, *137*, 691-700.

32. Hoek, G.; Brunekreef, B. *Arch. Environ. Health* **1993**, *48*, 328-335.

33. Hoek, G.; Brunekreef, B. *Environ. Res.* **1994**, *64*, 136-150.

34. Schwartz, J.; Dockery, D.W.; Neas, L.M.; Wypij, D.; Ware, J.H.; Spengler, J.D.; Koutrakis, P.; Speizer, F.E.; Ferris, B.G. *Am. J. Respir. Crit. Care Med.* **1994**, *150*, 1234-1242.

35. Hoek, G.; Brunekreef, B. *Am. J. Respir. Crit. Care Med.* **1995**, *151*, 27-32.

36. Neas, L.M.; Dockery, D.W.; Ware, J.H.; Spengler, J.D.; Speizer, F.E.; Ferris, B.G. Jr. *Am. J. Epidemiol.* **1991**, *134*, 204-219.