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Modeling the Benefits of Power Plant Emission Controls in Massachusetts

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ABSTRACT

Older fossil-fueled power plants provide a significant portion of emissions of criteria air pollutants in the United States, in part because these facilities are not required to meet the same emission standards as new sources under the Clean Air Act. Pending regulations for older power plants need information about any potential public health benefits of emission reductions, which can be estimated by combining emissions information, dispersion modeling, and epidemiologic evidence. In this article, we develop an analytical modeling framework that can evaluate health benefits of emission controls, and we apply our model to two power plants in Massachusetts. Using the CALPUFF atmospheric dispersion model, we estimate that use of Best Available Control Technology (BACT) for NO_x and SO_2 would lead to maximum annual average secondary particulate matter (PM) concentration reductions of 0.2 $\mu g/m^3$. When we combine concentration reductions with current health evidence, our central estimate is that the secondary PM reductions from these two power plants would avert 70 deaths per year in a population of 33 million individuals. Although benefit estimates could differ substantially with different interpretations of the health literature, parametric perturbations within CALPUFF and other simple model changes have relatively small impacts from an aggregate risk perspective. While further analysis would be required to reduce uncertainties and expand on our analytical model, our framework can help decision-makers evaluate the magnitude and distribution of benefits under different control scenarios.

IMPLICATIONS

Older power plants are not required to meet the same emission standards as new facilities and contribute a substantial fraction of criteria pollutant emissions in the United States. We have developed a model to evaluate the health benefits associated with NO_x and SO_2 emission reductions. Our central estimate is that requiring two older coal-fired power plants in Massachusetts to use BACT would lead to ~70 fewer premature deaths per year due to reduced secondary PM exposure. Along with detailed information about uncertainties and costs, benefit estimates can be used to help guide policy for older power plants.

INTRODUCTION

Because of a "grandfathering" provision in the Clean Air Act, older power plants have not been required to meet the same control requirements as new sources. This has created economic incentives to continue the usage of older facilities and discouraged new entrants in the power sector.[1-3] As a result, a small number of older power plants are responsible for a significant fraction of national emissions of SO_2 and NO_x. According to U.S. Environmental Protection Agency (EPA) data, the power sector is responsible for ~67% of national SO_2 emissions and 28% of national NO_x emissions, of which pre-1980 coal-fired power plants are responsible for 97 and 85%, respectively.[4]

To remedy this situation, some states have proposed multipollutant regulations to require grandfathered power plants to meet Best Available Control Technology (BACT) requirements, with national legislation also introduced. The state-level debates regarding the proposed regulations have implicitly and explicitly focused on two major questions. First, will the potential benefits of emission reductions (e.g., human health, ecosystem health, climate change, leveling the economic playing field) justify the potential costs (e.g., increased electricity prices, reduced fuel diversity, decreased system reliability)? Second, should emissions limits be met by mandatory on-site reductions, through a national emissions trading system, or through a combination? In contrast, there has been only moderate debate about whether removing the grandfathering provision would lead to fewer violations of National Ambient Air Quality Standards (NAAQS) in the short-term or the long-term.

This focus can be related to two key issues. First, there has been growing emphasis on the need for a cost-effective means of improving ambient air quality. Second, and perhaps more critically, the scientific literature for many criteria pollutants and health effects has been unable to detect the existence of population thresholds. States that are above and below the NAAQS are evaluating these

control strategies in a similar framework. This implies that a risk-based "damage function" approach may be the most reasonable way to understand the benefits of control policies while addressing the critical questions listed above.

Adopting a risk-based framework for policy formulation is sensible on numerous fronts. For example, under a framework where the primary focus is on control of air pollution to avoid exceeding thresholds, sources with tall stacks that have more disperse concentration impacts but significant effects on aggregate exposure may be overlooked. Moving to a risk-based framework of analysis can allow for better prioritization of control from a public health standpoint, while incorporating concepts such as cost-effectiveness and environmental equity.

Multiple studies in recent years have used this framework to analyze the impacts associated with emissions from power plants[5-8] or from transportation sources.[9,10] All of these studies ultimately attributed a health and subsequent economic burden to the incremental emissions from specific sources or the equivalent calculation of the benefits of emission reductions from these sources. Lifecycle impact assessments can be viewed in a similar light, as they aim to estimate the range of impacts associated with a specific product or process. Although these studies and similar externality assessments have been conducted for decades, they have had limited regulatory application.

Although the methodologies differ widely across impact assessment types and practitioners, all of these studies follow a general framework that merits careful scrutiny. For any damage/benefit estimation, the primary components can be categorized generally as

- estimation/forecasting of the amount of goods produced,
- evaluation of the required inputs to produce these goods,
- emissions inventory/estimation,
- dispersion modeling/exposure assessment,
- estimation of impacts associated with pollutant exposures among at-risk subpopulations or systems, and
- valuation and aggregation of impacts.

A comprehensive evaluation of the benefits of controls requires each of these components to be modeled with some precision and with adequate characterization of uncertainty, and addressing all components in depth is beyond the scope of this paper. In this paper, we propose a model framework to quantify the human health benefits associated with emission reductions at fossil-fueled power plants, with limited quantitative and qualitative consideration of uncertainty in critical components. Because of the numerous elements embedded within a damage function model, we attempt to list the uncertainties and assumptions we consider critical for accurate benefit estimation for power plants (generalizable in part to other source categories). For each component listed above, we consider the uncertainties that might significantly affect model results. To demonstrate the implications of varying assumptions in a subset of model components and to quantify the general magnitude of benefits associated with controlling emissions from grandfathered power plants, we present the findings from a case study in Massachusetts.

ANALYTICAL MODEL

Case Study Framework

To help frame the presentation of our analytical model, we first provide some details about our case study. We focus on two power plants in Massachusetts—Brayton Point (Somerset) and Salem Harbor (Salem). Massachusetts was selected because the state and stakeholders were debating regulation of grandfathered power plants at the time of our analysis. In addition, the Brayton Point and Salem Harbor plants are two of the higher-emitting facilities among the six grandfathered power plants affected by pending regulation. In total, these six power plants contribute approximately half of total SO_2 emissions and point source NO_x emissions in Massachusetts (8% of total NO_x emissions).[11] The Brayton Point and Salem Harbor power plants provide more than half of the grandfathered power plant contribution.[12] Both power plants are largely coal-fired, and basic characteristics of both facilities are provided in Table 1.

The regulation finalized in Massachusetts in April 2001 (310 CMR 7.29) contained emission standards for NO_x, SO_2, Hg, and CO_2, along with a placeholder to add primary fine particulate matter ($PM_{2.5}$) control at a later date. To parallel this regulation, we focus primarily on the potential health benefits associated with NO_x and SO_2 controls, through reductions in secondary PM concentrations. This omits benefits associated with Hg and CO_2, as well as any benefits associated with reductions

Table 1. Characteristics of power plants for Massachusetts case study.

	Salem Harbor	Brayton Point
Initial Year of Commercial Operation	1952	1963
Nameplate Capacity (MW, 1998)	805	1611
Net Generation (MWh, average 1996–1998)	3,222,262	7,660,738
Heat Input (MMBTU, average 1996–1998)	44,139,484	84,210,445
Emissions (Tons, average 1996–1998)		
SO_2	30,100	46,500
NO_x	6300	14,400

in primary PM (filterable and condensable), ozone, gaseous pollutants, or air toxics. This implies that our primary benefit estimates should not be taken as total benefits of controls but rather as a subset of benefits anticipated to contribute significantly to the total.

To evaluate benefits in the context of this state regulation, we consider two emission scenarios: one representing current practice, and one representing lower target emissions achievable through the application of BACT. Actual emissions of SO_2 and NO_x were estimated as the 3-year average of emission rates between 1996 and 1998 (the most recent available data at the time of our analysis). For lower target emissions, emission rates under BACT for coal-fired power plants built in recent years were 0.30 lb/MMBTU for SO_2 and 0.15 lb/MMBTU for NO_x. These values closely parallel the target values in Massachusetts of 3 lb/MWh for SO_2 and 1.5 lb/MWh for NO_x. Finally, for this case study, we focus exclusively on premature mortality. Although multiple morbidity outcomes have been linked with $PM_{2.5}$ exposure and could have significant contributions to monetized benefits under some valuation approaches, premature mortality has been the largest contributor in past studies.[5-10,13,14]

Damage Function Framework

As mentioned above, numerous subanalyses are contained in models to estimate the benefits of source controls, and the precise elements in these subanalyses clearly depend on the project framework. In Table 2, we list the major components of a damage function model for estimating the human health impacts of power plants, including a comparison between the idealized model and the assumptions made for our case study. It should be noted that the focus on air emissions and human health is a boundary decision underlying this figure that omits multiple categories of pollutants and effects. In addition, we provide our qualitative assessment of the direction and potential magnitude of any biases (small, medium, or large) associated with our case study assumptions. The ranking of the magnitude of biases is entirely subjective and at this time cannot be translated into quantitative uncertainty bounds. In general, "large" uncertainties are those that might potentially invalidate the findings or alter estimates by as much as an order of magnitude, while "small" uncertainties would be unlikely to influence any policy decisions. In the following section, we briefly describe the major analytical issues and uncertainties for each component, focusing on the choices made in our case study and their possible implications. We focus most extensively on the health evidence and what can be assumed about the benefits of incremental decreases in $PM_{2.5}$ concentrations at current ambient levels.

Production Forecasting

A comprehensive assessment of benefits would need to evaluate the path of electricity production across time for the affected power plants, both under current regulatory and consumption trends and given the additional regulation. For a state-level regulation (the focus of our analysis), there are two plausible extreme scenarios. In the first case, the regulation would not greatly influence utilization patterns or encourage new entrants in the marketplace, while growing electricity demand over time would result in increased utilization at previously grandfathered power plants. This would limit the benefits of controls. At the other extreme, any required installation of control technology could increase costs and lead to reduced utilization, which would increase the benefits of requiring plants to meet the lower emission levels (provided that the replacement electricity had lower impacts per unit generation). Some of the critical uncertainties for production forecasting are related to the structure of the regulation; a regulation mandating on-site clean up would likely have different implications than a regulation with an emissions trading framework. Regardless, accurate long-term modeling of benefits requires the application of economic and energy consumption models to estimate individual plant utilization.

In our case study, we assume that all units at the facilities precisely meet the lower target levels achievable through the application of BACT, with no changes in utilization. It is likely that changes in the electricity market in the northeast coupled with pending regulations would have some influence on individual plant utilization, but plant-specific projections are unavailable, and no specific deviation from constant utilization is warranted. Given that utilization could move in either direction, this provides a reasonable central estimate of benefits, particularly in the near-term when substantial market changes are unlikely. This element of uncertainty is not incorporated into our quantitative analysis.

Input Estimation/Emissions Inventory

The emissions per unit electricity generation can potentially be the most straightforward component of the damage function model, if we do not adopt a life-cycle approach and if emissions of key pollutants have been directly measured during the relevant time period. In our case study, we have accurate estimates of both past emissions and future target emissions of SO_2 and NO_x. Substantial uncertainties can be related to the determination of system boundaries. If the goal of the analysis is only to estimate impacts from stack emissions, this problem is less crucial. However, even an impact assessment of stack emissions may omit important impacts by focusing on a limited number

Table 2. Components of a damage function model for health effects of air emissions from power plants.

Component	Description	Case Study Assumption	Likely Direction/ Magnitude of Bias from Assumption
Production forecasting	Evaluation of electricity production over time, under current regulatory/consumption trends, and given additional regulation	Current utilization, focus on near-term	?; unknown
Emissions inventory	Estimation of time-resolved emissions of key pollutants per unit of electricity generation under multiple scenarios, potentially including upstream sources	Only stack emissions of NO_x and SO_2 (primary PM, air toxics omitted)	↓; medium
Atmospheric modeling	Calculation of annual average concentration increment associated with power plants across receptor region	600×600 km modeling regime	↓; small
		Use of MESOPUFF II, default wet/dry deposition, default NH_3 concentrations	?; medium
		Use of CALMET/CALPUFF	?; unknown
		Concentrations correlated with exposures	?; small
Health effects	Determination of morbidity and mortality effects associated with modeled concentration increments	Estimation of only mortality	↓; medium
		Assumption that ACS represents correct $PM_{2.5}$–mortality relationship	?; large
		Equal toxicity of all particles	?; large
		Linear concentration-response with no threshold	↑; unknown
At-risk population	Within each census tract, determination of the population to evaluate for health impacts and the baseline rate of disease/death	Identical relative risk for all people >30, identical background mortality rates in all census tracts	?; medium
Valuation	For each health outcome, assign an economic or health-based value corresponding to the outcome for the relevant at-risk population	Not addressed; EPA VSL estimate used for illustrative purposes	↑; large

of pollutants or by omitting other exposure pathways (e.g., fly ash). We address this in part in our case study by incorporating primary PM (filterable and condensable) in our sensitivity analysis.

In addition, uncertainties can be found when the emissions inventory does not provide the information needed for accurate dispersion modeling, such as particle size distributions or time-resolved emissions. To address the potential importance of the latter point, we evaluate benefits assuming both uniform emissions across the year and using seasonally varying emissions. Because of seasonal patterns in electricity demand and planned outages, there will clearly be some variation in emissions (particularly if we were to model peaking rather than base load units). Often, only annual average tonnage data are available, so it is important to determine if the assumption of uniform emissions might lead to significant errors.

Atmospheric Modeling/Exposure Assessment

There are numerous uncertainties and methodological issues in evaluating exposures per unit emissions, including the relevant exposure period, the geographic area of concern, the estimation of meteorological patterns, the determination of chemical conversion and deposition, and the relationship between ambient concentrations and personal exposures. In this article, we briefly discuss the choices made for our case study and the potential implications. More detailed information about our atmospheric modeling choices and the sensitivity of model findings to these choices in a different geographic setting can be found elsewhere.[15]

As outlined in a subsequent section, cohort studies provide an association between premature mortality and annual average $PM_{2.5}$ concentrations, so we focus our atmospheric modeling on estimating annual average $PM_{2.5}$ reductions at each receptor point. Of note, assuming a linear concentration-response function at current ambient levels implies that this exposure measure would also be applicable for time-series studies (because the average of the daily impacts would equal the daily impacts of the average). We construct our dispersion model to evaluate a geographic region covering 40–45° N and 67–75° W (~600 km × 600 km), with receptors at each census tract (see Figure 1). In total, this results in a potentially affected population of ~33 million, including 6 million in Massachusetts and 13 million in New York. This modeling domain was selected to maintain reasonable accuracy for the dispersion model while incorporating a significant fraction of aggregate impacts. Because of the importance of long-range transport of secondary pollutants, our receptor region likely omits a portion of total impacts, and the magnitude of this omission is estimated in our sensitivity analysis.

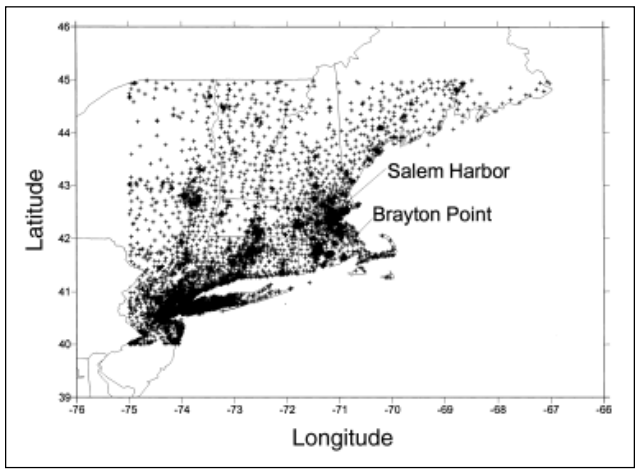

Figure 1. Receptor region for atmospheric dispersion model.

For the atmospheric modeling, we selected the CALMET/CALPUFF modeling framework.[16] CALPUFF is a Lagrangian puff model that can handle complex 3-dimensional windfields and has been shown to be unbiased for distances up to 200 km (with potential application at much longer range).[17] EPA has recommended CALPUFF for use in long-range transport modeling,[18] with the caveat that secondary particulate formation contains some uncertainties due to the first-order chemistry in CALPUFF and the complex nonlinearities in the sulfate-nitrate-ammonia-water system. We chose CALPUFF over other regional-scale models (e.g., UAM, Models-3, REMSAD) because of the relative ease of running the model for single sources under numerous parametric assumptions. Our modeling methodology using CALMET/CALPUFF is described at length in a separate publication.[15]

Briefly, we developed CALMET meteorological data by combining National Oceanic and Atmospheric Administration (NOAA) prognostic model outputs with meso-scale data assimilation systems for each hour across 1 year (January 1999–January 2000). NOAA's Rapid Update Cycle model provided upper air data at 40-km grid spacing. To provide the greater resolution needed to capture ground-level features, the Advanced Regional Prediction System (ARPS) Data Assimilation System was used in conjunction with METAR surface observations and reported cloud cover. The resulting hourly CALMET windfields had 15-km spacing within eight vertical layers. Precipitation data were taken from all National Climatic Data Center stations within the receptor region, with CALMET defaults used for interpolation between stations.

Within CALPUFF, we applied the MESOPUFF II chemical transformation mechanism and estimated wet and dry deposition using CALPUFF default parameters. We used hourly background ozone concentrations taken from CASTNET stations within the region (Woodstock, NH;

Connecticut Hill, NY; Washington Crossing, NJ). We assumed a constant NH_3 concentration of 1 ppb, which is an order of magnitude less than the CALPUFF default of 10 ppb and was selected to reflect the lower NH_3 levels in the northeast (to avoid overestimating nitrate formation). In this article, we address parametric uncertainty through the application of alternative assumptions for chemical conversion mechanism, implementation of wet and dry deposition, and assumed background NH_3 concentrations. However, it is difficult to evaluate whether the CALPUFF model is unbiased when compared with other atmospheric dispersion models, indicating that there remains significant model uncertainty that can only be evaluated indirectly in our analysis.

Finally, the issue of the relationship between concentration and exposure is not addressed in our analysis. Epidemiologic studies evaluate the relationship between health outcomes and ambient concentrations as recorded by central-site monitors, which correspond directly with the results from our dispersion models. These results are interpretable provided that ambient concentrations are correlated with population mean personal exposures to outdoor-generated pollutants. Because fine particles penetrate into the indoor environment with extremely high efficiency (particularly in well-ventilated settings),[19] this interpretation is likely appropriate. The critical question is whether any bias is induced due to differences between concentration and exposure. It has been argued that the difference between monitored and true ambient levels and the individual's deviation from risk-weighted average personal exposure would be Berksonian errors (which would not bias concentration-response functions), while the difference between average personal exposure and ambient levels could induce bias (most likely underestimating the effect).[20]

Concentration-Response Estimation

The key issue in determining appropriate concentration-response functions is to estimate the slope of the curve at current ambient concentrations. It should be noted that it is not necessary to determine whether a population threshold exists but, rather, whether there is evidence of a threshold above current ambient concentrations. In addition, because concentrations below NAAQS levels do not necessarily correspond to a zero risk level,[21] we must look to the health literature to determine if effects appear to be present at current ambient concentrations. In evaluating the health literature, we also must attempt to allocate effects to only the pollutants causally associated with the health outcomes. While misallocation could have limited influence on baseline risk calculations, it would have significant implications for the benefits of pollutant-specific control strategies. In this article, we focus on premature mortality associated with $PM_{2.5}$ exposure, motivated by its relative importance in past benefit-cost analyses.[13,14] In this section, we attempt to derive a reasonable central estimate of mortality impacts, and we discuss the substantial uncertainties in the Sensitivity Analysis section.

We consider the cohort mortality literature to capture any potential long-term exposure effects. To date, there have been three major published cohort mortality studies—the American Cancer Society study (ACS),[22] the Six Cities study (SC),[23] and the Adventist Health Study of Smog (AHSMOG).[24] The first two of these were recently reanalyzed by the Health Effects Institute (HEI).[25] In addition, preliminary findings from a national prospective cohort study of male veterans (VA) have been reported.[26] In general, the $PM_{2.5}$ effect is greatest in SC and smaller (and nonsignificant) in AHSMOG and VA. Because the ACS estimates are generally bounded by other studies, we derive our central estimate from ACS. In addition, more substantial concerns have been voiced about the other studies, including sample size and inability to discriminate among pollutants in SC, sample size and representativeness and lack of measured $PM_{2.5}$ in AHSMOG, and representativeness and inclusion of excessive covariates in VA.[27] Nevertheless, differences in findings among the studies should be included in a comprehensive uncertainty analysis and are considered to a limited extent in our sensitivity analysis.

The ACS study was a retrospective analysis of a cohort of more than 500,000 adults across the United States, followed from 1982 to 1989.[22] The cohort consisted of individuals at least 30 years of age at the time of enrollment who were generally acquaintances of volunteers for ACS (raising the question of population representativeness). To estimate air pollution exposures, individuals were matched to the nearest ambient monitors using concentrations from the start of the study period. In the original publication, the authors reported a relative risk of 1.17 for a 24.5 $\mu g/m^3$ increase in annual median $PM_{2.5}$ concentrations (95% CI: 1.09, 1.26), with a relative risk of 1.15 for a 19.9 $\mu g/m^3$ increase in annual mean SO_4^{2-} concentrations (95% CI: 1.09, 1.22).

In the HEI reanalysis,[25] numerous statistical models were tested to evaluate the robustness of this finding, with $PM_{2.5}$ relative risk ranging from insignificant to double the original estimate. Our central estimate is derived from a model using mean $PM_{2.5}$ concentrations from dichotomous samplers and including individual-level covariates for tobacco consumption, education, occupational exposure, body mass index, marital status, and alcohol consumption. Using this model, the authors calculated a relative risk of 1.12 (95% CI: 1.06, 1.19) for a 24.5 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations. This corresponds to an approximate 0.5% increase in premature mortality rates per $\mu g/m^3$ increase in annual mean $PM_{2.5}$

concentrations. Alternative models based on median $PM_{2.5}$ concentrations included methods to account for spatial autocorrelation, finding a similar relative risk to the original study (1.16) but a wider confidence interval (95% CI: 0.99, 1.37). No spatial autocorrelation model was applied to mean $PM_{2.5}$ concentrations.

By applying this concentration-response function in our case study, we assume that both SO_4^{2-} and NO_3^- particles have identical toxicity as "average" fine particles within the study regions. The ACS study found slightly higher risks per unit concentration for sulfates than $PM_{2.5}$ (as did the SC cohort study[23]), with no direct information available on nitrates. A recent time-series analysis based on SC data found that fine particles from coal combustion and mobile sources were associated with premature mortality but that crustal elements were not.[28] This finding agrees with studies that have associated daily mortality and morbidity more strongly with combustion particles than with noncombustion particles.[29,30] A baseline assumption of equal toxicity appears reasonable given current information. However, this is a source of significant uncertainty, and ongoing and future research may substantially alter this assumption.

Within the HEI reanalysis,[25] the only potential confounder that demonstrated a consistent effect was SO_2 (with educational attainment acting as a strong effect modifier). The authors did not infer causality for SO_2 but, rather, stated that it could be a marker for other correlated pollutants, that the findings could be related in part to spatial patterns in air pollution, and that the bundle of $PM_{2.5}$, SO_2, and sulfates appeared to be related to premature mortality. We test concentration-response functions corresponding to all three pollutants in our sensitivity analysis.

The HEI reanalysis found no evidence of a threshold at the annual average concentrations evaluated in the study (mean $PM_{2.5}$ concentrations of 10–38 $\mu g/m^3$, mean SO_4^{2-} concentrations of 1–27 $\mu g/m^3$). By way of comparison, annual average $PM_{2.5}$ concentrations were 8–17 $\mu g/m^3$ in Massachusetts in 2000, with total particulate sulfates of 8–9 $\mu g/m^3$.[31] Thus, the concentration range in the ACS study is relevant for evaluation of health benefits in Massachusetts and nearby states, although more uncertainty exists at the lower end of the concentration ranges. Although many areas in Massachusetts are below the pending $PM_{2.5}$ annual NAAQS of 15 $\mu g/m^3$, the health literature does not provide evidence of a population threshold. The nonthreshold assumption requires additional research and is clearly a significant source of uncertainty, which we address in our sensitivity analysis.

Although compelling evidence exists for the possibility of long-term exposure effects, there are numerous uncertainties related to the interpretation of this evidence. If a cohort effect did not exist, we could alternatively consider the evidence from time-series studies, for which there are relatively more studies and fewer analytical concerns (because only other air pollutants and weather can realistically act as confounders). For this sensitivity analysis scenario, we draw our estimate from the National Morbidity, Mortality, and Air Pollution Study of the 90 largest cities in the United States,[32] which found that mortality rates increase by 0.5% for every 10 $\mu g/m^3$ increase in daily PM_{10} concentrations. This is similar to the value derived in a recent meta-analysis of the PM_{10}-mortality literature, which found a pooled value of 0.6% when controlling for the effects of correlated gaseous pollutants.[33] Because we are focusing on $PM_{2.5}$, we can convert these estimates to an incremental mortality risk of ~0.1% per $\mu g/m^3$ increase in exposure to $PM_{2.5}$, assuming a standard $PM_{2.5}/PM_{10}$ ratio of 60% and assuming that only fine particles yield health effects. This risk is similar to values reported in past $PM_{2.5}$ time-series studies.[34] It should be noted that the deaths from time-series studies are not commensurate with deaths from cohort studies, in terms of life expectancy lost and other characteristics.

At-Risk Subpopulations

For premature mortality, the determination of at-risk subpopulations has multiple components. Within our case study, we assume that the at-risk group consists of all individuals above the age of 30 (as studied in the ACS cohort), with no differential relative risk by age, gender, health status, or other demographic characteristics. We also use the U.S. average mortality rate for this age group and apply it to all census tracts in the receptor region. Clearly, a more accurate analysis would incorporate tract-specific mortality rates stratified by demographic characteristics, along with differential relative risks across demographic strata. However, there is limited evidence indicating differential relative risks for cohort mortality. As mentioned earlier, educational attainment was found to be an effect modifier of mortality in the ACS study,[25] but ecological covariates such as income, poverty, and race showed little effect. Additional evidence can be taken from recent time-series studies, which found relative risks to be relatively homogeneous across all characteristics but baseline health status.[35-37] Despite the numerous assumptions underlying our at-risk population determination, it is unclear what the magnitude or direction of any potential bias would be. The bias would be anticipated to be greater for sources in close proximity to high-risk or low-risk communities for health outcomes known to have large geographic variability (e.g., power plants near urban areas for the evaluation of asthma exacerbation or emergency room visits).

Valuation

The final component of a damage function model is generally valuation of the array of quantified impacts. For human health, this can take the form of economic valuation, medical-based metrics (such as quality-adjusted or disability-adjusted life years), or indicator metrics as used in life-cycle impact assessment. Given the substantial uncertainty both within and between valuation categories, and given that we are only quantifying one health outcome in our case study, we do not consider valuation in a substantive way in this article.

For illustrative purposes, we provide a simple benefit estimate based on the EPA value of statistical life (VSL) central estimate of $5.8 million (in 1997 dollars),[13] based on an evaluation of past wage-risk and willingness-to-pay studies. Although a detailed evaluation of the literature is beyond the scope of this article, it is worth noting that the EPA value is based on study populations (largely healthy workers) that differ somewhat from the individuals at highest risk from air pollution (likely elderly individuals with pre-existing cardiovascular or respiratory disease). For older individuals, fewer life years are at risk from current-period mortality risks, but the opportunity cost of spending on risk reduction is also lower.[38] Thus, it is not theoretically obvious what the precise consequence of this difference would be. Alternatively, valuation can be placed in life-year terms, taking advantage of the Cox proportional hazards model that implicitly provides information on the loss of life expectancy. However, the notion that all life years would be valued equally is not supported by standard economic theory.

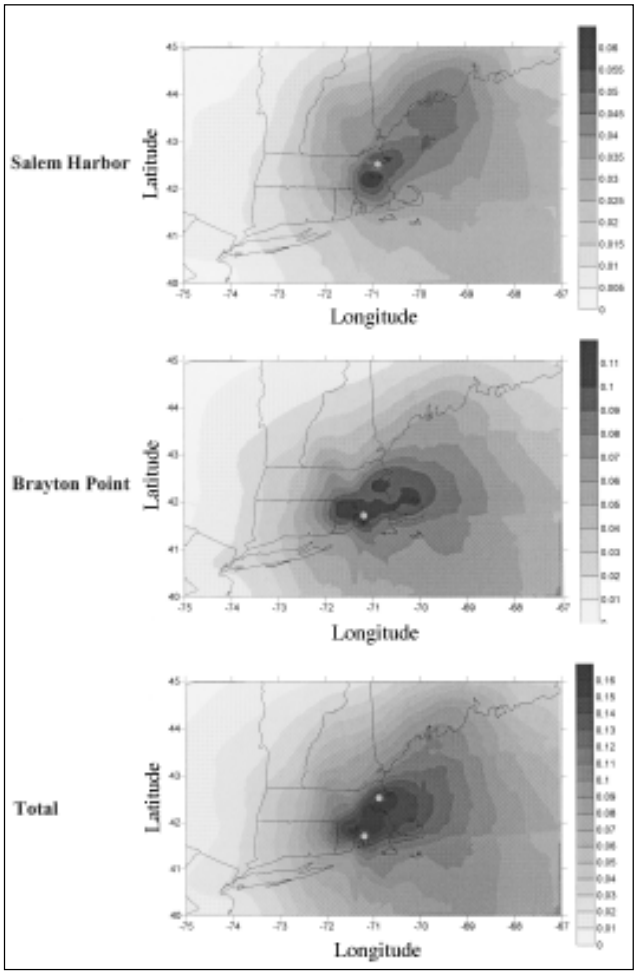

Figure 2. Geographic distribution of benefits from emission controls at the Salem Harbor and Brayton Point power plants (μg/m³ of secondary PM, annual average).

RESULTS

In this section, we provide the results of our case study analysis using our central estimates for all parameters. We evaluate the sensitivity of the estimated concentration reductions and health benefits to key assumptions in the following section. Using CALPUFF under baseline parametric assumptions, we estimate that SO_2 and NO_x emission controls at the Brayton Point and Salem Harbor power plants lead to annual average $PM_{2.5}$ reductions of 0.006–0.2 μg/m³, depending on the location in our modeling domain (see Figure 2). The benefits tend to peak in relatively close proximity to the power plants, although with some distance required for secondary particulate formation to occur. The maximum annual average benefit occurs ~40 km from the source for Salem Harbor and 25 km from the source for Brayton Point. Peaks occur at generally greater distances within seasons, but differences in wind patterns and mixing heights by season lead to annual average peaks relatively closer to the source. As would be expected, the geographic patterns of annual average concentration reductions generally follow the prevailing wind direction in New England. For both power plants, benefits 300 km toward the northeast are approximately 5–6 times greater than benefits 300 km toward the west.

Secondary sulfate particles provide a majority of benefits from both facilities. We can summarize the contribution of each pollutant by considering the population-weighted annual average concentration reduction (taking the benefit at each census tract, multiplying by the population within that tract, and dividing by the total population). Given our assumptions regarding concentration-response functions, the health benefits will be directly proportional to the population-weighted concentration reduction. Using this measure, sulfates contribute 83% of the benefits from Brayton Point and 88% of the benefits from Salem Harbor. This large SO_4^{2-} contribution is principally a function of the relative emissions of SO_2 and NO_x, as well as the fact that NH_3 preferentially reacts with SO_4^{2-} over NO_3^- and was assumed to be limited over our modeling domain. When we combine the modeled concentration reductions with our baseline $PM_{2.5}$ concentration-response function, we estimate ~70 fewer

Table 3. Implications of key assumptions on aggregate mortality risk reduction estimate for Brayton Point and Salem Harbor combined (holding other assumptions constant).

Modified Assumption	Total Mortality Risk Reduction[a]		
	Sulfates	Nitrates	Total
None (baseline)	59	11	70
Seasonally varying emissions	57	11	68
Addition of primary PM	59	11	79[b]
Dispersion modeling assumptions			
Use of RIVAD/ARM3 in CALPUFF	59	23	82
Exclusion of wet/dry deposition	83	15	97
Use of 10-ppb NH_3	59	17	76
Assumption of overestimation within 50 km, beyond 200 km of source	43	7	51
Assumption of overestimation beyond 200 km of source	48	9	58
Extension of model domain indefinitely	73	14	87
Concentration-response (C-R) assumptions			
Original ACS C-R for $PM_{2.5}$	82	15	97
HEI ACS C-R for sulfates, no NO_3^- impact	100	0	100
HEI ACS C-R for SO_2, no PM impact	–	–	290[b]
Original SC C-R for $PM_{2.5}$	160	30	190
Baseline ACS for $PM_{2.5}$, NAAQS violators only	14	2	16
Time-series $PM_{2.5}$ mortality only	14	3	16

[a]All figures are presented to two significant figures. Sums may not add due to rounding; [b]Including impacts other than sulfates and nitrates.

deaths per year (70% from Brayton Point) across a total population of 33 million and an "at-risk" population (age 30 and older) of 19 million (see Table 3). In our baseline model framework, the geographic distribution of individual health benefits is identical to the distribution of concentration reductions.

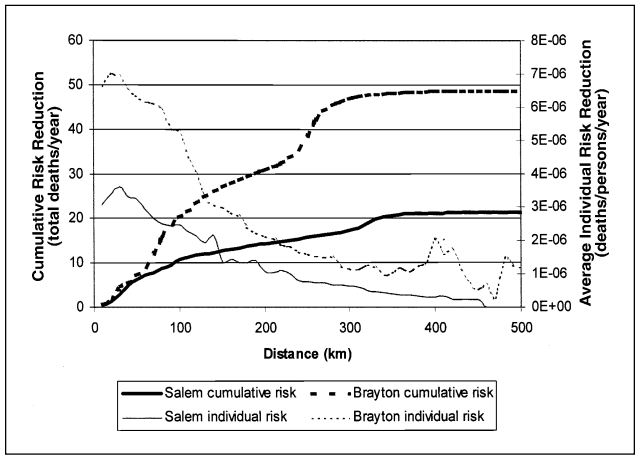

Figure 3. Distribution of individual and aggregate mortality risk reduction from emission reductions at Brayton Point and Salem Harbor power plants as a function of distance from the source.

We can plot both individual and aggregate risk reductions as a function of distance from the source to illustrate an important distributional difference (see Figure 3). In this figure, risks are averaged across all receptors in 10-km bins. Because concentration patterns and receptor locations are geographically skewed, the distribution of individual risk reductions is not always monotonic. For both power plants, individual benefits are greater closer to the facilities, given concentration patterns. However, for our modeling domain, only a small fraction of the population lives at close range (6% within 50 km of Brayton Point, 10% for Salem Harbor). Thus, a majority of the aggregate benefits accrues at long range, with more than half of the benefits found beyond 100 km of the source. This illustrates the importance of population patterns and density in determining aggregate benefits. Using the standard EPA valuation for premature mortality, the annual monetary benefits corresponding to our central health benefit estimate would be approximately $400 million. Given the substantial uncertainty in the VSL estimate, this calculation should be considered illustrative at best.

SENSITIVITY ANALYSIS

Although we presented the mortality benefits of emission controls as point estimates, as described above, the estimates are quite uncertain. While we cannot evaluate the full scope of uncertainties, we can determine the implications of a limited number of quantifiable assumptions to make a general determination of the relative magnitudes of uncertainties and the possible direction of any biases. We quantify the sensitivity of our findings to the incorporation of seasonal emissions, omission of primary PM, parametric assumptions in CALPUFF, the size of the modeling domain, and the concentration-response function for premature mortality for each of the particle constituents. We do not address monetary valuation, nor do we incorporate the assumptions listed in Table 2 that are currently unquantifiable (such as plant utilization or application of CALPUFF rather than other dispersion models). We present the implication of each assumption independently and consider discrete combinations of a limited number of assumptions. While an ideal analysis would combine estimated distributions for all parameters into a single probability density function, the accurate estimation of distributions or even probability weights to assign to different parametric assumptions is beyond the scope of our analysis.

Incorporation of Seasonal Emissions

As mentioned earlier, we modeled concentration and health benefits using both uniform and seasonally varying emissions. The findings for these analyses are essentially

identical (see Table 3). With the varying emissions model, aggregate SO_4^{2-} benefits decrease by 5% for Salem Harbor and 1% for Brayton Point, while NO_3^- benefits decrease by 3% for Salem Harbor and increase by 1% for Brayton Point. This is largely due to the similarity in emission rates and heat inputs across seasons. Our dispersion model estimated SO_4^{2-} impacts per unit emissions that were nearly an order of magnitude greater in the summer than in the winter, indicating that this factor could be important for sources with significant seasonality in emissions.

Omission of Primary Particulate Matter

Although we have omitted filterable and condensable PM from our baseline analysis due to the structure of proposed regulations in Massachusetts, both pollutants could be significant contributors to health benefits and would be affected by on-site control measures. To determine baseline filterable $PM_{2.5}$, we gathered data on 1996–1998 average PM_{10} emission rates and used EPA's Particle Calculator Version 2.0.2[39] to estimate the $PM_{2.5}/PM_{10}$ ratio. Lower target PM_{10} under BACT was determined to be 0.01 lb/MMBTU. For simplicity, we assumed that the $PM_{2.5}/PM_{10}$ ratio would be unchanged from current levels (which likely provides an upper bound on omitted benefits, because many control measures disproportionately decrease larger particles). For current condensable PM, we used AP-42 emission factors given coal sulfur content. Lower target levels were taken from AP-42 estimates for facilities with control technology in place, and all condensable PM was assumed to be in the fine fraction.

Under these assumptions, the mortality benefits associated with primary $PM_{2.5}$ emission reductions would be on the order of 10 fewer deaths per year, increasing our baseline estimate by 13% (see Table 3). As anticipated, the near-source contribution is more substantial than for secondary PM, with 32% of primary $PM_{2.5}$ benefits for Brayton Point and 60% for Salem Harbor occurring within 50 km of the source (versus 15 and 29% for secondary PM, respectively). Thus, our omission of primary $PM_{2.5}$ slightly underestimates the benefits of control and the proportion of near-source benefits.

Parametric Uncertainty in CALPUFF

In our sensitivity analysis, we test three major parametric assumptions in CALPUFF—the chemical conversion mechanism, the incorporation of wet/dry deposition, and the background NH_3 concentration. When we use the RIVAD/ARM3 chemical mechanism rather than MESOPUFF II (holding all else constant), SO_4^{2-} benefits are essentially unchanged (5% decrease at Brayton Point, 12% increase at Salem Harbor), but NO_3^- benefits are increased substantially (doubled at both power plants). The MESOPUFF II chemical mechanism is generally preferred (particularly in urban settings), but this demonstrates the model sensitivity of NO_3^- benefit estimates relative to SO_4^{2-} benefit estimates. Because a majority of total benefits are related to sulfates, the choice of chemical mechanism has a relatively small influence on total benefits (see Table 3).

We did not evaluate whether our incorporation of deposition might have overestimated benefits (insufficient deposition), but we can place a bound on any potential underestimation by removing all deposition terms from the model. This would clearly overestimate benefits, but given the numerous uncertainties associated with deposition rates and scavenging coefficients[15] and the omission of risks associated with the deposited materials, this may not be an inappropriate conservative estimate. Omitting deposition increases benefits for both sulfates (40% increase at Brayton Point and 39% increase at Salem Harbor) and nitrates (34% increase at Brayton Point and 29% increase at Salem Harbor). Finally, increasing the background NH_3 concentration to the CALPUFF default of 10 ppb has no effect on sulfates but increases NO_3^- benefits by 54% at Brayton Point and 45% at Salem Harbor. Thus, perturbing these three parametric assumptions tends to increase benefits, with atmospheric chemistry assumptions influencing nitrates more than sulfates (see Table 3).

Size of the Modeling Domain

Any uncertainty associated with our modeling domain is difficult to quantify, because we do not have the data to directly quantify long-range concentrations. Our modeling domain could overestimate benefits if CALPUFF were upwardly biased at longer range, or it could yield an underestimate if a significant fraction of exposure occurred beyond the boundaries of our receptor region. Tracer dispersion experiments found that CALPUFF was unbiased between 50 and 200 km, but EPA found that CALPUFF might overestimate at long range by as much as a factor of 2.[17] A reasonable lower bound would assume that all concentrations beyond 200 km were overestimated by a factor of 2 and, because near-source (<50 km) performance was not evaluated, that a similar bias exists there. This would reduce benefits by 26% for Brayton Point and 31% for Salem Harbor. If we only consider the possibility of overestimation beyond 200 km, these figures are reduced to 18 and 16% (see Table 3).

To place a bound on any potential underestimation due to our limited receptor region, we can fit a simple regression between concentration reductions and distance from the source. Beyond 50 km, total secondary PM concentration reductions can be well predicted as an exponential function of distance ($R^2 = 0.81$ for Brayton Point and 0.87 for Salem Harbor). If we assume for simplicity that population density is uniform at long range, we can

determine the degree of underestimation due to our abbreviated model domain. In total, we estimate that increasing our modeling bounds indefinitely would increase benefits by ~19% for Salem Harbor and 26% for Brayton Point (Table 3). Thus, it does not appear that we have significantly underestimated benefits, potentially related in part to the preponderance of long-range receptors at largely upwind locations.

Concentration-Response Function

There are clearly numerous plausible estimates of mortality concentration-response functions, based on reported confidence intervals, alternative statistical models within studies, use of different studies, and alternative assumptions about particle constituent toxicity. Although evaluating the complete range of uncertainties is beyond the scope of this paper, we can consider a set of discrete scenarios as alternatives to our baseline concentration-response function. These scenarios are listed next, with the figure in parentheses indicating the central estimate of the mortality increase per $\mu g/m^3$ of concentration increase.

- the original reported ACS concentration-response function for $PM_{2.5}$ (0.6%);[22]
- ACS concentration-response function for sulfates (0.8%, Table 31 of HEI reanalysis[25]), with no health impacts from nitrates;
- ACS concentration-response function for SO_2 in multivariate models (0.5%, Table 50 of HEI reanalysis[25]), assuming that SO_2 is a more appropriate marker than $PM_{2.5}$;
- our baseline ACS concentration-response function for $PM_{2.5}$ (0.5%), applied only to receptors in counties with any monitors exceeding the pending annual NAAQS (15 $\mu g/m^3$, 1999 data);
- the original reported SC concentration-response function for $PM_{2.5}$ (1.2%);[23] and
- zero $PM_{2.5}$ cohort mortality effect (as implied by VA[26] or selected models from other studies), with a time-series effect of 0.1%.

Depending on the concentration-response assumption, our total benefits can vary significantly from our baseline estimate. If we assume either that cohort mortality effects are not present or that they are only applicable for counties exceeding the pending annual $PM_{2.5}$ NAAQS, our benefit estimates are decreased substantially. On the other hand, using the SC concentration-response function or assuming that the SO_2 concentration-response function from the ACS reanalysis is appropriate increases our benefits significantly (see Table 3). More moderate differences are associated with using the original ACS concentration-response function or considering impacts to only be associated with SO_4^{2-} particles (39 and 44% increases in benefits, respectively).

Sensitivity Propagation

The above calculations illustrate that our total benefit estimates are sensitive to key parametric assumptions, with the largest quantifiable influence associated with the assumed concentration-response function. To provide a sense of the range of benefit estimates implied by quantifiable uncertainties, we consider discrete combinations of a subset of factors. While this should not be construed as a formal uncertainty analysis, it can help place some of the parametric uncertainties in context. If we combine the lower bound concentration-response functions with an assumption of dispersion modeling overestimation outside the 50–200-km range, our benefit estimate would be on the order of 10 deaths per year. An extreme upper bound would use the SO_2 concentration-response function with no deposition and an indefinite model domain, yielding a benefit of ~400 deaths per year. We consider both of these estimates to be unlikely and not representative of best modeling practice or literature interpretation. Excluding the SO_2 mortality scenario as well as the scenarios where no cohort effect exists or it only exists at more than 15 $\mu g/m^3$, we find benefit estimates that range between ~50 and 200 premature deaths per year.

As a comparison point, we can also calculate uncertainty bounds by conventional propagation of uncertainties as determined by 95% confidence intervals surrounding our baseline model estimate. A typical assumption would be that the population-weighted annual average concentrations have a 95% confidence interval between 50 and 150% of the central estimate (an interval generally applied to the Industrial Source Complex model[40] for shorter-term measurements in single locations). We combine this with the 95% confidence interval for our baseline premature mortality estimate, as reported in the HEI reanalysis (95% CI: 1.06, 1.19).[21] When we combine these two confidence intervals using Monte Carlo analysis, we find a mean mortality risk reduction of 70 deaths per year (95% CI: 30, 120). This clearly does not incorporate the range of issues within our sensitivity analysis but does encompass a number of the simple sensitivity estimates in Table 3.

DISCUSSION

Our case study has demonstrated that the magnitude and distribution of health benefits of power plant emission controls can be estimated reasonably. Through the application of an atmospheric dispersion model coupled with epidemiologic evidence regarding the health benefits of incremental concentration reductions, our central estimate is that requiring two Massachusetts power plants to apply BACT would lead to ~70 fewer premature deaths per year over a broad region. There are numerous obstacles in the interpretation of these findings. Although we likely

captured a significant contributor to benefits, without quantification of control costs or other benefits, it is difficult to interpret the importance of the findings. In addition, the individual risk reduction is relatively small, and the $PM_{2.5}$ concentration reductions are on the order of 2% of ambient concentrations. However, it is important to recognize that we have modeled only secondary PM from a subset of sources from one sector within one state. Any such analysis would find a relatively small contribution to ambient concentrations, but this does not imply that regulatory action would not be justified. This determination requires a comparison between the full array of benefits and the full array of control costs. The more difficult related issue is the potential increase in atmospheric modeling uncertainty for small concentration increments.

We have also only quantified a limited degree of uncertainty, and additional elements in the atmospheric modeling and health evidence (as well as plant utilization and other dimensions not addressed in our case study) could significantly affect our benefit estimates. For the dispersion modeling, our parametric sensitivity analysis lends support to the relative robustness of our estimate with respect to changes internal to CALPUFF. However, CALPUFF may be biased when compared with other models. For example, issues related to CALPUFF's aqueous-phase chemistry for SO_4^{2-} formation have been raised, which may imply significant underestimation of SO_4^{2-} formation and impacts.[17] We can validate our findings to a limited degree by comparing our analysis with other studies that used alternative dispersion models but made similar assumptions elsewhere.

For example, a recent study by Abt Associates[41] used REMSAD and a source-receptor (S-R) matrix to determine the benefits of a 75% emission reduction of SO_2 and NO_x from all power plants in the United States. Using the identical concentration-response function as in our baseline model, they determined annual benefits of ~19,000 fewer deaths per year using REMSAD and 12,000 fewer deaths per year using S-R from annual emission reductions of 7 million tons of SO_2 and 2 million tons of NO_x. The emission reductions in our study are ~0.8% of the national SO_2 reduction and 0.6% of the NO_x reduction. Applying these ratios to the above mortality benefits yields numbers on an order of magnitude of 100 fewer deaths per year, similar to our finding. While this is far from direct validation of the CALPUFF model and its application, the similarity of our results to those using other models lends plausibility to the order of magnitude of our findings.

Considering the health literature, our extreme assumptions demonstrated that there are some substantial uncertainties. For one, we have assumed that the cohort mortality evidence reflects a causal relationship for $PM_{2.5}$ that can be attributed solely to $PM_{2.5}$. We have also assumed that the slope of the concentration-response curve at current ambient levels is similar to the slope derived in the ACS cohort study. Both assumptions reflect scientific questions for which uncertainty may be reduced by future research. However, one of the advantages of the damage function framework is that it can transparently provide benefit estimates under a number of scenarios. Thus, decision-makers can take their beliefs for key parameters (e.g., the existence of health benefits below the NAAQS for $PM_{2.5}$) and determine the corresponding benefits, or they can use the range of values to determine if their policy decisions are influenced by selected assumptions.

Aside from the specific evidence we used, two broad critiques can be raised about the damage function/environmental externality approach. Related to some of the uncertainties listed previously, there is the argument that current knowledge about important model components (i.e., atmospheric chemistry, relative toxicity of particulate constituents, biological mechanisms supporting causality) is insufficient to construct damage function models. In other words, even if we could construct more detailed uncertainty analyses, we simply do not know enough about the behavior of air pollutants to begin to quantify the health benefits of air pollution control. While it is true that significant scientific uncertainties exist and will continue to exist for the foreseeable future, this should not act as a barrier to action or analysis. Rather, this implies that researchers should carefully analyze the range of uncertainties and determine whether the uncertainties might materially affect policy choices. In addition, the scientific uncertainties must be placed in context. For example, a dispersion model that does a poor job estimating the precise location of concentration peaks but accurately estimates population-weighted annual average concentrations is quite useful for our application.

A second concern is that the damage function approach (and the corresponding benefit-cost analyses that could be conducted) is contrary to the current regulatory structure for criteria pollutants in the United States, which focuses largely on the establishment of NAAQS and the development of plans to avoid violations. Thus, damage function modeling may be reasonably accurate, but it does not provide information relevant to policy-makers. While this is correct on its face, we would assert that the damage function approach has a number of applications within the existing regulatory framework. For example, as emission control plans are developed, damage function modeling can determine the magnitude and distribution of health benefits from an array of policies that might all achieve NAAQS compliance. Policy-makers could then select a portfolio of options that achieves the identical regulatory purpose at minimum cost with maximum aggregate benefits and reduced environmental inequities.

In addition, when multiple source categories and pollutants are modeled in a similar framework, damage function modeling can be used to help in overall prioritization of future research and regulatory agendas.

Future analyses should focus on incorporating the remaining elements necessary for an adequate benefit-cost analysis and on generalizing our findings to other settings. To have relevance for pending policy decisions, our damage function model should be applied to evaluate the benefits of specific proposed regulations (e.g., on-site emission reduction to BACT levels, unrestricted emission trading, mandatory partial on-site reductions coupled with regional emission trading). Ozone, Hg, CO_2, and any other pollutants associated with the control measures should be included if shown to be important, and the economic implications of the regulations should be ascertained. Regarding generalizability, while some meteorological and topographic characteristics are unique to Massachusetts, it is clear that our findings can be extrapolated to a limited extent to other settings (particularly those in close proximity to the modeled facilities). Furthermore, recent work has demonstrated that the exposure per unit emissions from a power plant is reasonably invariant across plants for secondary particles[42] and can be predicted well by a limited number of parameters (such as population density and climate).[43] Additional studies should confirm these relationships for different source types and settings.

CONCLUSIONS

We have constructed a model to quantify the concentration and health benefits associated with NO_x and SO_2 emission reductions from power plants. Application of our model to two power plants in Massachusetts finds a reduction of ~70 premature deaths per year associated with decreases in secondary PM concentrations. Although further research would be needed to incorporate additional elements in our model (including future plant utilization and life-cycle emissions) and to more comprehensively characterize uncertainties, our findings are relatively robust with respect to parametric changes in the dispersion model or moderate changes in assumed concentration-response functions. Given the potential magnitude of health benefits from large-scale regulation of power plants or other significant emission sources, the damage function modeling approach should be used in conjunction with cost information to inform future control strategies.

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