The Importance of Population Susceptibility for Air Pollution Risk Assessment: A Case Study of Power Plants Near Washington, DC

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Abbreviations

ACS: American Cancer Society

BACT: Best Available Control Technology

CHA: Cardiovascular hospital admissions

ERV: Emergency room visits

MMBTU: Million British Thermal Units

NIDDM: Non-insulin dependent diabetes mellitus

NOx: Nitrogen oxides

$PM_{2.5}$: Particulate matter less than 2.5 μm in aerodynamic diameter

PM_{10}: Particulate matter less than 10 μm in aerodynamic diameter

SO_2: Sulfur dioxide

Abstract

In evaluating risks from air pollution, health impact assessments often focus on the magnitude of the impacts without explicitly considering the distribution of impacts across subpopulations. In this study, we construct a model to estimate the magnitude and distribution of health benefits associated with emission controls at five older power plants in the Washington, DC area. We use CALPUFF to determine the primary and secondary fine particulate matter ($PM_{2.5}$) concentration reductions associated with the hypothetical application of Best Available Control Technology to the selected power plants. We combine these concentration reductions with concentration-response functions for mortality and selected morbidity outcomes, using a conventional approach as well as considering susceptible subpopulations. Incorporating susceptibility has a minimal effect on total benefits, with central estimates of approximately 240 fewer deaths, 60 fewer cardiovascular hospital admissions (CHA), and 160 fewer pediatric asthma emergency room visits (ERV) per year. However, since individuals with lower education appear to have both higher background mortality rates and higher relative risks for air pollution-related mortality, stratifying by educational attainment implies that 51% of the mortality benefits accrue among the 25% of the population with less than high school education. Similarly, diabetics and African-Americans bear disproportionate shares of the CHA and ERV benefits, respectively. Although our ability to characterize subpopulations is constrained by the available information, our analysis demonstrates that incorporation of susceptibility information significantly affects demographic and geographic patterns of health benefits and enhances our understanding of individuals likely to benefit from emission controls.

Introduction

The issue of subpopulation susceptibility to fine particulate matter ($PM_{2.5}$) has been given increased attention by researchers in recent years, motivated in part by the research priorities articulated by the National Academy of Science *(1)*. Understanding patterns of susceptibility would not only help identify and protect sensitive subpopulations, but it would also contribute to the understanding of mechanisms by which $PM_{2.5}$ might influence human health.

Often, air pollution policies are informed by risk assessments or benefit-cost analyses, which generally focus on the total health benefits of alternative emission control strategies *(2-5)*. Because limited relevant susceptibility evidence exists, differential effects on susceptible subpopulations are rarely incorporated. Typically, the same relative risks are applied to all individuals in an "at-risk" age group, and baseline disease or health care utilization rates are assumed to be uniform across large geographic areas (often national averages).

However, it is likely that the effects of air pollution vary widely across subpopulations, depending on demographics, behavior patterns, income, access to health care, and other factors. Differences could exist either in relative risks (if an increment of air pollution yields a different percentage increase in effect in different populations) or in absolute risks (if there are differences in baseline disease patterns by subpopulation, independent of air pollution). For a benefits assessment, if policy makers were concerned about distributional issues or if the ultimate valuation of benefits depended on population characteristics, the incorporation of susceptibility could potentially influence the conclusions.

One current policy issue for which information on susceptibility could be influential is the regulation of emissions from older power plants. To date, older power plants have not been required to meet the same control requirements as new sources, helping to extend the useful

lifetime of older facilities *(6-8)*. These facilities contribute a substantial fraction of national power sector emissions. In 1999, coal-fired power plants contributed approximately 86% of nitrogen oxide (NOx) emissions and 93% of sulfur dioxide (SO_2) emissions from the utility sector, largely from facilities exempted from new source standards *(9)*.

At the time this article was written, multiple states (including Massachusetts, Connecticut, and Texas) had introduced multipollutant regulations or legislation to require older power plants to meet emission levels commensurate with the application of Best Available Control Technology (BACT). Pollutants considered typically included NOx and SO_2, as well as mercury and carbon dioxide. Multipollutant power plant legislation was also being debated at the federal level, but no bills or regulations existed at the time of our analysis.

From both a state and federal perspective, the question of how the benefits of emission controls would be distributed could be important. Policy makers may be concerned about providing benefits to high-risk communities, communities near power plants, or other subpopulations. If these questions were important, population susceptibility could influence the policy choices (e.g., emission trading versus mandatory on-site controls).

In this paper, we develop a model to estimate the health benefits associated with emission reductions at older fossil-fueled power plants. We focus on both primary $PM_{2.5}$ and secondary sulfate and nitrate particles formed through emissions of SO_2 and NOx, respectively. We consider a case study of all older power plants located within a 50 mile (80 km) radius of Washington, DC. We calculate three health endpoints – premature mortality, cardiovascular hospital admissions in the elderly, and pediatric asthma emergency room visits – both using conventional assumptions and then considering available evidence for differential effects on susceptible subpopulations. Our goal is both to quantify the health benefits associated with the

implementation of BACT at the selected power plants and to consider whether introduction of susceptibility models might affect the interpretation of our findings.

Case Study Setting

For this analysis, our goal was to select a geographic area that had multiple older power plants nearby and geographic heterogeneity in factors that might influence relative risks, baseline health status, or health care utilization (such as socioeconomic status). Washington, DC and its surrounding suburbs provide an example of such a region. According to 1990 US Census data, median household income in Washington, DC ranged from under $10,000 to over $150,000 across census tracts *(10)*. Washington, DC is also quite racially divided, with few African-Americans residing in the western half of the city and mostly African-Americans residing in the eastern half of the city.

In addition, within a 50 mile (80 km) radius of Washington, there are five fossil-fueled power plants grandfathered under the Clean Air Act - Benning, Chalk Point, Dickerson, Possum Point, and Potomac River (Table 1). The choice of these five power plants is somewhat artificial, since any single regulation would not affect only these plants. However, our analysis is meant to be illustrative, and these five plants are likely the greatest contributors to heterogeneity in power plant-related exposures in the area. Inclusion of additional power plants would increase the total benefits but decrease the relative concentration gradient across the Washington area.

Methods

General

To quantify the magnitude and distribution of health benefits, we estimate the emission reductions of key pollutants, apply an atmospheric dispersion model to determine incremental concentration reductions, and derive concentration-response functions. Any such analysis involves numerous boundary decisions and contains substantial uncertainties. In this paper, we focus largely on issues related to susceptible subpopulations and the resulting implications. We do not extensively address the complexities of other elements of the model, nor do we provide a formal analysis of uncertainties. We also do not consider the economic valuation dimension of a benefits assessment. Additional information about parametric uncertainties in our atmospheric model *(4,11)* and issues related to differential particle toxicity or alternative interpretations of the health evidence *(4)* can be found elsewhere.

Quantification of emissions

We estimate emissions of $PM_{2.5}$ and its precursors (NOx and SO_2), following the model structure in our earlier analyses *(4,11)* and supported by the fact that $PM_{2.5}$ has dominated aggregate benefits in past air pollution risk assessments *(2,3)*. This omits any benefits associated with ozone, air toxics, or other impact pathways from the power sector. Of note, most proposed regulations consider NOx and SO_2 but do not directly require controls for primary $PM_{2.5}$ (although many NOx and SO_2 control strategies would affect primary $PM_{2.5}$).

We use 1999 as the base year for our analysis, evaluating the concentration and health benefits that would have been obtained had lower target emission rates been achieved. This is not

identical to the future benefits that might be obtained through pending regulation, since some facilities have ongoing or near-term plans for repowering or emission controls.

Emissions of SO_2 and NOx were taken from the US EPA Acid Rain Program Emissions Scorecard *(12)*. To capture seasonality in emissions, we incorporated quarterly average emission rates when reported. When no data on seasonal emissions were available, we assumed constant emissions per unit of heat input. For filterable $PM_{2.5}$, total plant emissions were taken from the US EPA National Emission Trends database *(13)*. We estimated condensable $PM_{2.5}$ emissions given fuel type and sulfur content, using AP-42 emission factors from US EPA.

We selected lower target emissions to correspond to the levels proposed in multiple regulations, which correspond to the application of Best Available Control Technology (BACT). This resulted in target emission rates of 0.3 lb/MMBTU of SO_2, 0.15 lb/MMBTU of NOx, and 0.01 lb/MMBTU of filterable PM. Lower target condensable PM emissions were taken from AP-42, given assumed application of control technologies. Since both Dickerson and Benning have actual filterable $PM_{2.5}$ emissions less than the lower target rate, we set the lower target filterable $PM_{2.5}$ emission rate equal to actual emissions for these plants.

Atmospheric modeling

We established a receptor grid covering a 400 km (250 mile) radius around Washington, DC (centered at 38.9°N, 77°W), to capture a significant fraction of total benefits without extending the dispersion modeling boundaries excessively (Figure 1). Because of our focus on spatial patterns, it was important to determine concentration reductions at small geographic scales close to the sources. Within 100 km of Washington, census tracts were selected, as they are relatively small (generally between 2,500 and 8,000 people) and were theoretically designed

to be socioeconomically homogeneous. Beyond 100 km, county-level resolution was used, resulting in a nested receptor grid with 1,908 receptors. Using 1990 Census data (the most recent data available at the time of our study), our receptor grid contained 47 million individuals, 7 million of whom live within 100 km of Washington.

We conducted our atmospheric modeling using CALPUFF (CALMET version 5.2 000602a, CALPUFF version 5.4-000602-1, CALPOST version 5.2 991104b; Earth Tech, Concord, MA). CALPUFF is a regional-scale Lagrangian puff model that has been recommended by US EPA for long-range transport modeling *(14)*, given that it has been shown to be relatively unbiased at distances out to 200 km *(15)*. In general, limitations in the atmospheric chemistry make the secondary pollutant estimates relatively more uncertain than the primary $PM_{2.5}$ estimates, given the nonlinearities associated with sulfate and nitrate formation.

Our methodology to generate meteorological files for CALMET was similar to the approach in our past applications and is described in depth elsewhere *(4,11)*. We combined NOAA prognostic model outputs with mesoscale data assimilation systems for each hour across our case study year (January 1999-January 2000). This involved combining lower-resolution upper air data (40 km grid spacing) generated through NOAA's Rapid Update Cycle (RUC2) model with METAR surface observations and cloud cover data available at 15 km resolution. These data sources were combined using the ARPS Data Assimilation System (ADAS) and provided hourly CALMET windfields 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. The primary difference from our previous applications was the inclusion of 50 evenly spaced "soundings" based on columns of the ADAS

data, to more accurately provide a reasonable high-resolution temperature field and subsequent planetary boundary layer depth estimates.

In CALPUFF, we adopted recommended modeling assumptions that were used in our past applications *(4,11)*. We used the MESOPUFF II chemical transformation mechanism, which is generally preferred in urban settings. Wet and dry deposition were incorporated using precipitation data and CALPUFF default deposition rates. Hourly background ozone concentrations were taken from five CASTNET stations spaced throughout our receptor region (Prince George's, MD; Mercer, NJ; Elk, PA; Prince Edward, VA; Gilmer, WV), and we assumed a background ammonia concentration of 1 ppb.

For brevity's sake, we do not provide sensitivity or uncertainty analyses for our atmospheric modeling in this article. In our past analyses *(4,11)*, we found total benefits to be reasonably stable given single parametric changes in CALPUFF, including the chemical conversion mechanism, background ammonia concentration, and treatment of wet and dry deposition. In addition, we concluded that any bias associated with either hypothetical CALPUFF overestimation beyond 200 km or exclusion of long-range exposures is relatively small in comparison with other model uncertainties. A comprehensive risk assessment would need to incorporate these uncertainties in an evaluation of overall model uncertainty.

Health evidence

Although numerous health outcomes have been incorporated into past analyses *(2)*, we focus on a subset for which some evidence exists for differential effects on susceptible subpopulations. The choice of outcomes as well as the subpopulations considered is therefore entirely dependent on the current literature and is not meant to be comprehensive. Furthermore,

we restrict the health evidence to epidemiological studies conducted in the US, since patterns of health care utilization and the relationship between demographics and health status likely vary across countries. Given these criteria, we evaluate premature mortality (stratified by education), cardiovascular hospital admissions for the elderly (stratified by diabetic status and age), and asthma emergency room visits for children (stratified by race and age). For each outcome, we describe both a conventional approach and construct a susceptibility model. Our goal is not to consider the complete array of susceptible subpopulations, but rather to select one example for each outcome for which epidemiological evidence and population data exist.

Premature mortality

For premature mortality, we derive a central estimate from the follow-up analysis of the American Cancer Society (ACS) cohort study *(16)*. Multiple other cohort studies are available *(17,18)*, but the ACS study has the largest and most geographically diverse population, with relative risks bounded by other studies and a statistical approach suggested by a detailed reanalysis *(19)*. For all-cause mortality, the authors calculated a relative risk of 1.04 (95% CI: 1.01, 1.08) for a 10 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations (using 1979-1983 concentrations). The relative risk was slightly higher (1.06) using more recent pollution data, but we use the lower figure to be conservative and since Pope and colleagues presented stratified estimates based on the 1979-1983 concentrations *(16)*.

Relative risks did not vary substantially across most demographic factors, with the exception of educational attainment. Educational attainment appeared to be a strong effect modifier across all causes of mortality. The relative risk for a 10 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations was 1.085 (95% CI: 1.031, 1.142) for individuals with less than high school

education, 1.045 (95% CI: 1.004, 1.087) for individuals with high school education, and 1.003 (95% CI: 0.967, 1.040) for individuals with more than high school education.

There are numerous uncertainties related to the application of this stratified relative risk. The ACS cohort is somewhat more educated than the population at large, and correlated terms such as race and poverty status have not been significant in time-series mortality or hospital admissions studies *(20-22)*. In addition, the statistical approach implies that we are modeling the effect of education controlling for smoking and other factors, which would ideally be included to model the influence of all risk factors correlated with educational attainment. Regardless, we use the education-stratified values to determine the implications of the reported relationship.

For background mortality rates, the standard approach is to apply county-level averages to individuals age 30 and older (the age range considered in the ACS study). We use this as our baseline approach, but for our susceptibility model, consider whether mortality rates vary as a function of education while still averaging to the reported county-level rates.

There is a strong and consistent negative relationship between socioeconomic status and all-cause mortality *(23)*. Socioeconomic status can be measured by occupation, income, education, or some combination of these terms. It is generally believed that both income *(24)* and educational attainment *(25)* are independent predictors of mortality, although the bases for these relationships are not well understood. Some argue that those in lower socioeconomic classes display high-risk behaviors, such as smoking, being overweight, and not exercising *(26)*, resulting in higher mortality rates. However, only a small fraction of the increased mortality can be explained by a higher prevalence of high-risk behaviors *(27)*, so there must be other contributing factors. In any case, it is clear that those in low education or income categories represent a susceptible subpopulation for all-cause mortality.

Educational attainment is a useful predictor of mortality since it typically does not change after adulthood. Additionally, this term is available for all segments of the adult population, even those not in the workforce. Although it may be a proxy for other factors, various hypotheses have been presented for why lower education might be a causal factor for mortality. Education may be a marker for factors (such as intelligence and good health in early childhood) that allow for both educational attainment and good health in adulthood, for acquired knowledge that can be used to obtain positive health outcomes, for relative status in society, or for the development of positive social networks *(28)*. The protective effect of higher education has been seen in the US *(28)* and worldwide *(29,30)*.

We select our baseline mortality risk ratios from a study that evaluated risks for all-cause mortality as a function of both education and annual income among a cohort aged 25-64, drawn from the National Longitudinal Mortality Study *(28)*. The relationship between education and mortality was best described by a trichotomy (less than high school education, high school diploma or greater but no college diploma, or a college diploma or greater). When compared with the highest education group, the annual mortality relative risk for men was 1.7 for less than high school education and 1.5 for high school diploma or greater but no college diploma. For women, the corresponding relative risks were 1.5 and 1.2. The attenuation in women has been documented previously and can be attributed largely to the married subpopulation of women *(31)*. We apply these relative risks to all individuals over age 30, although there is some evidence that socioeconomic differences play less of a role in determining mortality rates among the aged *(32)*.

Cardiovascular hospital admissions

A number of studies in the US have evaluated the relationship between particulate matter exposure and cardiovascular hospital admissions (CHA) among individuals age 65 and older *(21,22,33-40)*. Most central estimates from these studies fall in the range of a 0.5-1% increase in CHA for a 10 $\mu g/m^3$ increase in daily PM_{10} concentrations. Using a typical $PM_{2.5}/PM_{10}$ ratio of 60%, we would consider a central estimate of an approximate 1% increase in CHA per 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ concentrations appropriate. As a baseline, we apply this percentage to the average background rate of 0.084 CHA per year per individual age 65 and older *(41)*.

Although numerous factors might influence either the baseline risk or the relative risk of an air pollution-related CHA, we focus on diabetes to illustrate the influence of a risk factor that varies demographically and might influence both risks. To estimate the number of diabetic and non-diabetic CHA in a county or census tract, we consider two relationships – the risk factors for diabetes among the elderly and the differential risk for a CHA given the presence of diabetes.

In those over 65, non-insulin dependent diabetes mellitus (NIDDM) accounts for virtually all of the diabetic caseload. There are numerous risk factors for NIDDM, including age, obesity, family history, and sedentary lifestyle. Although lifestyle variables are the strongest predictors of diabetic status (accounting for as much as 90% of population attributable risk *(42)*), we cannot estimate these variables at the census tract level from publicly available data. In the absence of this information, we estimate NIDDM prevalence as a function of gender, age, and race. According to a national survey *(43)*, NIDDM prevalence in individuals over age 65 is higher among African-Americans and Mexican-Americans than in non-Hispanic whites, ranging from 10.9% for non-Hispanic white males aged 65-74 to 29% for Mexican-American females aged 65-74. We apply these estimates to our study populations, despite the limitations in applying

national relationships based on race to a specific geographic setting. The relationship between race and common risk factors likely varies widely across regions and within small geographic areas, a feature that is not captured by our model.

Regarding risks for a CHA, it has been well established that diabetics have an increased risk of heart disease. Several studies also indicate that diabetics are admitted to the hospital more frequently than non-diabetics *(44,45)*. Thus, it is unsurprising that CHA rates are elevated in diabetic populations. According to a national diabetes surveillance report *(46)*, as of 1996, the annual CHA rate was 0.20 admissions per year per diabetic age 65-74 and 0.27 for diabetics 75 and older. In contrast, the rates for the population as a whole are 0.06 (age 65-74) and 0.11 (75 and older) *(41)*. Using these two rates and the estimated diabetes prevalence across our study population, we can calculate the CHA rate for non-diabetics. Clearly, there are several appreciable assumptions underlying these estimates. Although we know that marked differences can exist in hospital utilization rates among states and communities, we assume that tract-specific rates vary only as a function of the estimated number of diabetics, with CHA rates invariant for non-diabetics. This likely underestimates the degree of spatial and demographic variability in CHA rates.

On the relative risk side, a time-series study in Chicago *(35)* found a 2% increase in CHA for diabetic individuals over age 65 for a 10 $\mu g/m^3$ increase in PM_{10}, versus a 0.9% increase for non-diabetics. In contrast, the studies that evaluated factors such as race, education, or poverty *(21,34,40)* found no significant effect modification for CHA relative risks. To ensure that our concentration-response function is in agreement with our non-stratified estimate, we assume that a factor of two difference exists between diabetics and non-diabetics and calculate the concentration-response function given the estimated number of CHA in diabetics and non-

diabetics in our study population. The result is a 0.7% increase in CHA per 10 $\mu g/m^3$ increase in $PM_{2.5}$ for non-diabetics, with a 1.5% increase for diabetics.

Pediatric asthma emergency room visits

Many studies have associated emergency room visits (ERV) for numerous respiratory and cardiovascular causes with particulate matter, but to date only two studies in the US have considered asthma-related visits among children (defined here as 18 years of age or younger). In Seattle *(47)*, an 11.6 $\mu g/m^3$ increase in PM_{10} was associated with a 14% increase in asthma ERV (95% CI: 5%, 23%), with a 9.5 $\mu g/m^3$ increase in $PM_{2.5}$ associated with a 15% increase. This study found the relative risk to be similar in high-utilization and low-utilization areas (a proxy for socioeconomic status). In Atlanta *(48)*, a 4% increase in pediatric asthma ERV was estimated for a 15 $\mu g/m^3$ increase in PM_{10} concentrations (95% CI: 0.4%, 7%). As in Seattle, there did not appear to be effect modification due to race or socioeconomic status. Simply pooling these two studies using a random effects model *(49)* provides a central estimate of a 0.7% increase in asthma ERV per $\mu g/m^3$ increase in PM_{10}, which we translate into an approximate 1% increase in asthma ERV per $\mu g/m^3$ increase in daily $PM_{2.5}$. This can be applied to a background asthma ERV rate of 0.012 for children age 0-4, 0.0081 for children age 5-14, and 0.0069 for children above age 15 *(50)*.

Although the published studies did not identify susceptible subpopulations from a relative risk perspective, the background rate of asthma ERV would be anticipated to differ widely across subpopulations. This would be a function both of trends in asthma prevalence and in patterns of health care utilization across populations.

Asthma prevalence has increased substantially in recent years *(50)*, with lower-income individuals and minorities disproportionately affected by the disease *(51-55)*. Many of the significant predictors of childhood asthma, such as cockroach presence in the home *(56)* or maternal education *(57)*, are related to socioeconomic status. Furthermore, patterns of health care utilization are strongly related to income. The ratio of anti-inflammatory to beta-agonist medication is lower in low-income communities and is inversely correlated with hospitalization rates *(58)*, and lower-income populations lacking health insurance often use emergency services as a means of primary care. Thus, it would be expected that low-income populations would have somewhat higher pediatric asthma ERV rates.

Data on pediatric asthma ERV rates as a function of income were limited, but substantial racial differences have been documented. According to data from the National Hospital Ambulatory Medical Care Survey *(50)*, across all ages, the asthma ERV rate for African-Americans is nearly five times greater than for whites (0.023 and 0.0049 per capita, respectively). No data were provided on asthma ERV rates stratified across both age and race, but a study of three-year olds in the US finds a racial differential of similar magnitude but with some independent effects of both race and income *(51)*.

Given available information, we estimate baseline pediatric asthma ERV rates as a function of age and race, assuming the racial disparity to exist in all age groups. This encompasses both differences in prevalence and in health care utilization. As with our diabetes estimates, there are some substantial limitations in using only race as a predictor, since the relationship between race and asthma ERV risk factors varies by income, urban/rural status, and other factors. Regardless, the consistent relationship between race and ERV and the ability to gather racial information at the census tract level make this the best available covariate.

Results

Concentration Reductions

Using our atmospheric dispersion model, the emission reductions at the five selected power plants lead to annual average $PM_{2.5}$ (primary plus secondary) concentration reductions ranging from 0.009-0.9 $\mu g/m^3$ in our receptor region (Figure 2). By way of comparison, according to EPA AIRS data, annual average $PM_{2.5}$ concentrations in Washington were approximately 14-18 $\mu g/m^3$ in 1999. The maximum annual average $PM_{2.5}$ concentration reduction is found within Washington, as might be anticipated by the power plant selection criteria and the inclusion of primary $PM_{2.5}$.

The geographic distribution of benefits varies somewhat across particle types, power plants, and seasons. Annual average primary $PM_{2.5}$ concentration reductions peak closer to the plants and decrease more rapidly with distance than secondary sulfates or nitrates (Figure 2). As a result, a greater fraction of total exposure reduction (defined as the sum across receptors of the product of concentration reduction and population assigned to the receptor) occurs closer to the power plants for primary than for secondary $PM_{2.5}$ (Figure 3). However, there is tremendous variability in the distribution of total exposure reduction, principally due to variations in source locations and pollutant type (primary versus secondary). In addition, total exposure reduction per unit emissions displayed expected seasonal patterns, with slightly higher values for primary $PM_{2.5}$ in the winter and fall (related in part to lower mixing heights) and higher values for sulfates and lower values for nitrates in the summer due to the effect of temperature on relative conversion rates.

Health Benefits

For premature mortality, using non-stratified relative risks and homogeneous baseline mortality rates within counties, our central estimate is that emission reductions from the five power plants would lead to 210 fewer deaths per year (Table 2). The estimated impact under the current emissions scenario is 270 deaths per year. Of the total mortality benefits, approximately 25% occur in individuals with less than high school education (identical to the proportion in the population). Approximately 16% of mortality benefits accrue within 50 km of the power plants, largely related to the substantial contribution of secondary sulfates (62%) and nitrates (19%) to total $PM_{2.5}$ exposures.

In our susceptibility model, with both baseline mortality rates and $PM_{2.5}$ relative risks stratified by educational attainment, our understanding of the affected subpopulations changes substantially (Table 2). The total mortality benefit is largely unaffected, with a slight increase associated with differences in educational attainment between the Washington area and the ACS cohort. However, 51% of the estimated mortality benefits now accrue among individuals with less than high school education, double the prediction in the homogenous risk model.

Although stratification by education does not significantly influence the broad geographic patterns of benefits (i.e., the fraction of benefits within 50 km), at the census tract level, benefits differ by as much as a factor of 13 between the models. Figure 4 depicts the geographic patterns of benefits under both the baseline and susceptibility models, focusing solely on census tracts in Washington, DC for simplicity. Using the baseline model, the mortality risk reductions in Washington are reasonably homogeneous, ranging from 36 to 67 fewer deaths per year per million individuals over age 30. Under the education-stratified model, the range broadens considerably and the distribution is more complex, with per capita benefits now ranging by more

than a factor of 10 across census tracts. The mortality benefits are generally increased in southeastern Washington, the lowest-income area of the city.

When we consider CHA among the elderly, our baseline model estimates 59 fewer CHA per year. Although it seems counterintuitive that the mortality numbers could exceed the morbidity numbers, this is related to the limited focus on cardiovascular admissions due to only short-term exposures among the elderly (versus all-cause mortality from long-term exposures among individuals age 30 and older). Using a conventional model that assumes diabetics not to differ in any way from non-diabetics, 13% of the CHA benefits are estimated to occur among diabetics, while 80% are found among non-Hispanic whites (Table 2). The geographic distribution of CHA benefits is similar to the exposure reduction and mortality benefits, with differences reflecting the relative number of individuals age 65-74 and above age 75 within census tracts.

As expected, incorporating the diabetes-based information has a minimal impact on aggregate benefits but dramatically alters the profile of the affected individuals (Table 2). Using this model, 54% of the CHA benefits are found among diabetics, with 76% among non-Hispanic whites. Since we have assumed that baseline CHA risk for non-diabetics does not differ as a function of race or income, the CHA estimates under the susceptibility model are closer to those from the baseline model than for mortality (Figure 4). However, even only considering diabetes-related susceptibility changes the census tract-level benefits by as much as 40%.

Finally, we estimate 140 fewer pediatric asthma ERV per year using our non-stratified model (38% in children age 0-4, with 46% in children age 5-14). Twenty-seven percent of benefits occur in African-American children (who represent 21% of the study population). When we stratify asthma ERV risk by race, the total benefits increase to 160 fewer visits per year, with

significant changes in the geographic and demographic distributions (Table 2). The census tract-level risk reduction varies by an order of magnitude across Washington, with the benefits increased by more than a factor of two in the eastern half of the city (Figure 4). The proportion of benefits among African-American children is increased to 64%, commensurate with the assumption of greater baseline asthma ERV rates.

Discussion

Our analytical approach demonstrates two important points. First, given an interpretation of the epidemiological evidence that assumes that ambient concentrations in the Washington, DC area exceed any potential population threshold for $PM_{2.5}$ health effects, emission controls at older fossil-fueled power plants would provide tangible and quantifiable health benefits. Second, when we take account of susceptible subpopulations and differences in both relative risk and baseline disease rates across these populations, the small-scale geographic and demographic distributions of those benefits are strongly affected. For the example of premature mortality, if educational attainment influences both the relative risk of air pollution and the baseline mortality risk, then more than half of the mortality benefits accrue among the 25% of our study population with less than high school education. Similarly, for pediatric asthma emergency room visits, the fact that background rates are substantially greater in African-Americans implies that a majority of the emergency room visit benefits accrue in 21% of the population, even given identical relative risks from air pollution. The relatively smaller differences found for cardiovascular hospital admissions when diabetes is considered illustrates that evidence for differential effects on a relatively small fraction of the population has a smaller effect than a population-wide model.

There are clearly some barriers in both interpretation of the study findings and application of our model to other settings. One important uncertainty is related to the stratified risk models we selected. For all health outcomes, we used stratification variables (such as race) that might have independent effects on baseline health but likely are proxies for numerous socioeconomic endpoints. If the stratification variables represent other factors, this adds to the uncertainty in a site-specific stratified analysis.

In general, we have applied susceptibility models based on national data to a small number of states, which has multiple inherent limitations. Clearly, it would be preferable to use local health data, but data at small geographic scales for a large region are difficult to obtain and are rarely stratified across all demographic variables of interest. In addition, the reliance on national data increases the generalizability of our findings. Despite these issues, our models demonstrate that simple assumptions about susceptibility can be influential in our understanding of health risks and benefits. The alternative is an assumption of homogeneity, which itself introduces implicit uncertainty and may contribute to biases in selected settings.

Another limitation of our study is the fact that we have devoted limited attention to uncertainty analysis, a crucial element in interpreting sensitive and complex findings. Drawing on the uncertainty analyses in our earlier work *(4,11)*, most parametric changes in CALPUFF led to changes to aggregate benefits of less than a factor of two, while variations in concentration-response assumptions (particularly for mortality) could influence estimates by as much as a factor of five. The influence of population susceptibility is generally at the lower end of this range, even for small geographic scales. However, susceptibility information has a greater influence on the relative distribution of benefits than other assumptions, many of which tend to affect all populations identically (e.g., the population average concentration-response).

Furthermore, a broader view of areas of heterogeneity or susceptibility (e.g., assumptions regarding particle size and chemical composition, time-activity data, or physiological factors *(59)*) could increase the importance of this evidence. Further analysis that considered the full array of uncertainties and evaluated which (if any) would be influential in policy decisions would be warranted.

In addition, although we have focused on power plants (in part due to pending regulatory decisions at the time of our analysis), the issue of susceptible subpopulations is likely more significant for motor vehicle pollution. Given that motor vehicles have low stack heights and have a strong presence in urban street canyons with high population density, it is likely that aggregate impacts would be spread over a smaller population than for power plants. If the exposed population had demographic differences from the US average, assumptions of homogeneity would bias the risk calculations.

Finally, any assessment of impacts from a limited number of sources is somewhat impaired by the relatively small reductions when compared with baseline concentrations. This makes field validation of model results difficult and implies that an ultimate comparison of the costs and benefits of taking action would be required to determine if action is warranted.

Despite these limitations, our analysis illustrates that emission controls at older fossil-fueled power plants could lead to quantifiable concentration and health benefits and that susceptibility information informs the interpretation of those benefits. Although the individual benefits represent a small increment over baseline risks, the number of people affected due to long-range pollution transport implies aggregate benefits that are relevant for policy evaluation. As the health literature develops additional information about differences in relative and absolute

risk across populations, risk assessments and benefit-cost analyses should take advantage of this information to provide more interpretable information to decision makers.

Conclusions

We have evaluated the health benefits of emission controls at five older fossil-fueled power plants in the Washington, DC area, using both conventional risk assessment assumptions and incorporating available information about susceptible subpopulations. We find that the geographic and demographic distribution of benefits differs substantially between the two approaches. If robust and causal, our susceptibility models identify subpopulations that bear a disproportionate air pollution burden and account for a substantial fraction of the benefits of emission controls (lower-educated individuals for mortality, diabetics for cardiovascular hospital admissions, and African-Americans for asthma emergency room visits). The characterization of high-risk subpopulations can help both in the interpretation of the risk assessment and in targeting future exposure assessment or epidemiological efforts.

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Table 1: Characteristics of five power plants in Washington, DC case study (1999 data)**.**

	Benning	Chalk Point	Dickerson	Possum Point	Potomac River
Initial year of commercial operation	1968	1964	1959	1948	1949
Nameplate capacity (MW)	580	2046	588	1373	514
Heat input (MMBTU)	3,304,107	85,352,274	33,592,811	28,930,805	32,100,184
Emissions (Tons, % per quarter)					
SO_2	1,432 (2,21,76,2)	57,630 (21,25,31,23)	30,637 (30,17,34,18)	19,497 (24,22,32,23)	17,627 (22,28,29,21)
NOx	447 (2,22,74,1)	25,222 (20,24,30,26)	10,709 (30,17,34,18)	5,116 (25,22,32,21)	6,893 (21,28,30,21)
$PM_{2.5}$	12 (2,22,74,2)	304 (21,27,33,20)	14 (30,17,34,18)	156 (23,20,37,20)	106 (21,28,29,22)

Table 2: Magnitude and distribution of health benefits associated with modeled emission reductions at five power plants near Washington, DC (rounded to two significant figures; sums may not add due to rounding).

	Baseline model (No stratification)	Full susceptibility model (Stratification by listed covariate)
Deaths/year		
Total	210	240
< HS education	52	120
≥ HS education	150	120
Cardiovascular hospital admissions/year		
Total	59	60
Diabetic	8	33
Non-diabetic	51	27
Asthma emergency room visits/year		
Total	140	160
African-American	38	100
Non-African-American	100	57

Figure 1: Receptor grid and power plant locations for Washington, DC case study.

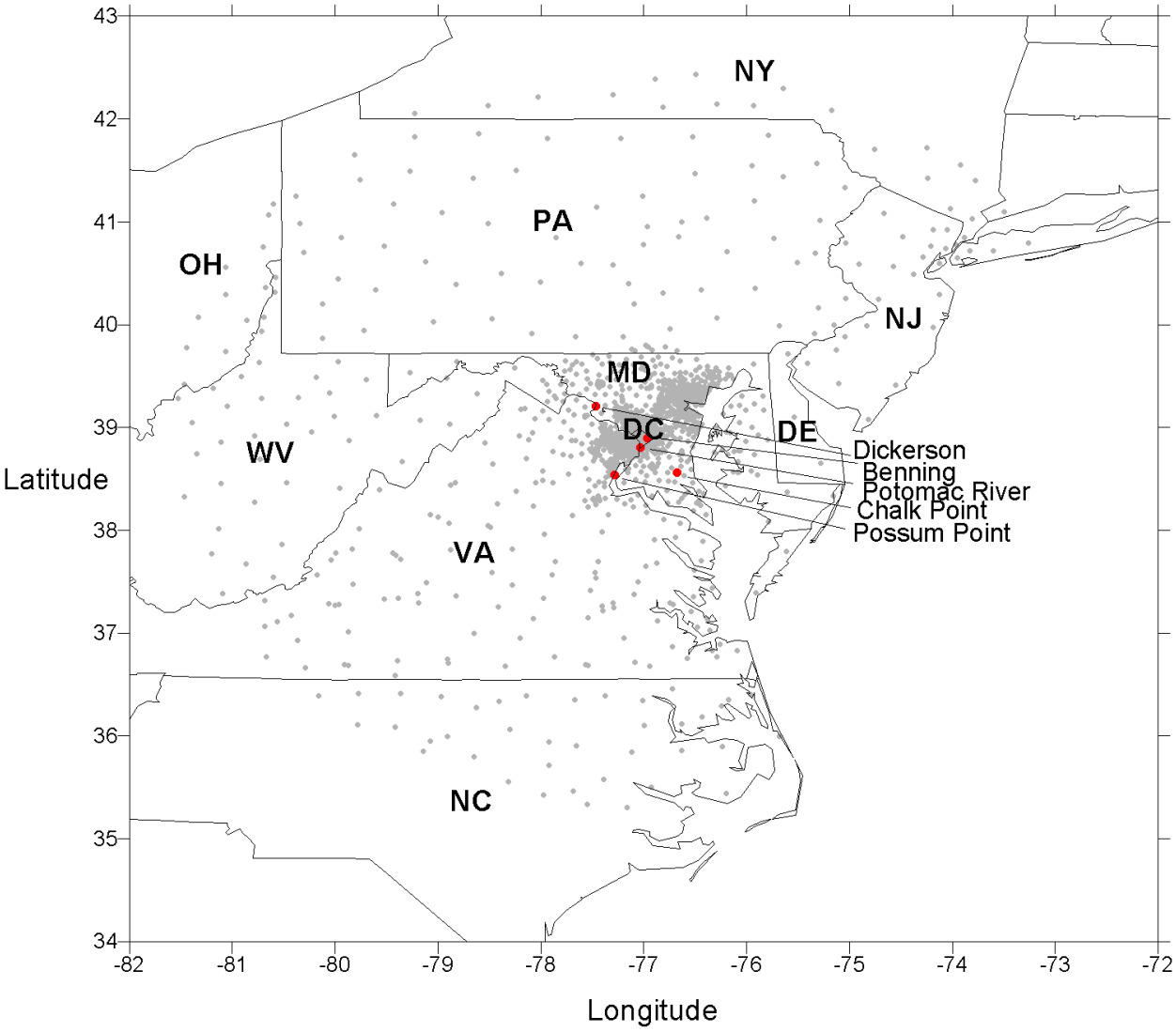
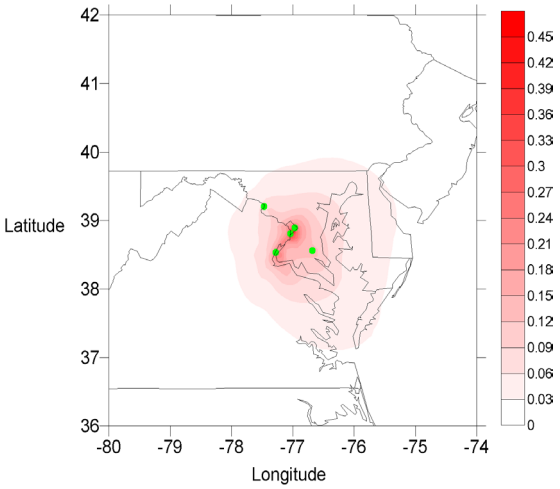
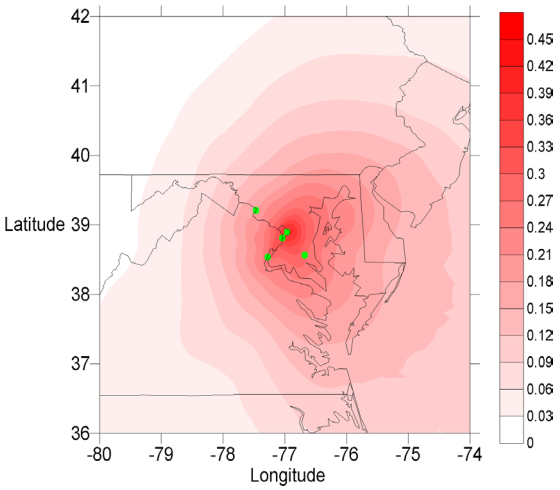


Figure 2: Combined concentration reductions (annual average, $\mu g/m^3$) from hypothetical emission controls at five power plants (primary $PM_{2.5}$, secondary $PM_{2.5}$, and total $PM_{2.5}$).

a) Primary $PM_{2.5}$



b) Secondary $PM_{2.5}$



c) Total $PM_{2.5}$

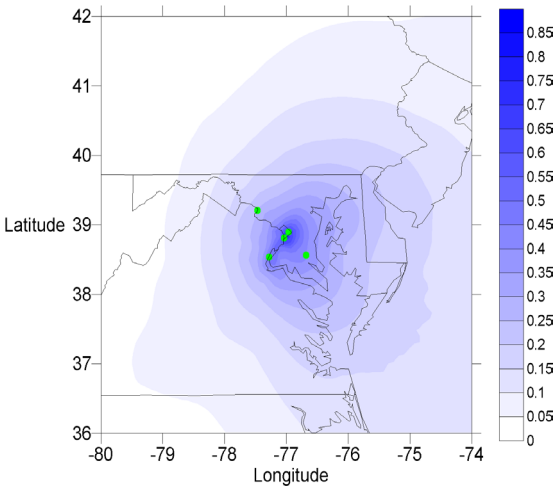


Figure 3: Cumulative distribution of total exposure reduction as a function of distance from the source, by power plant and pollutant type.

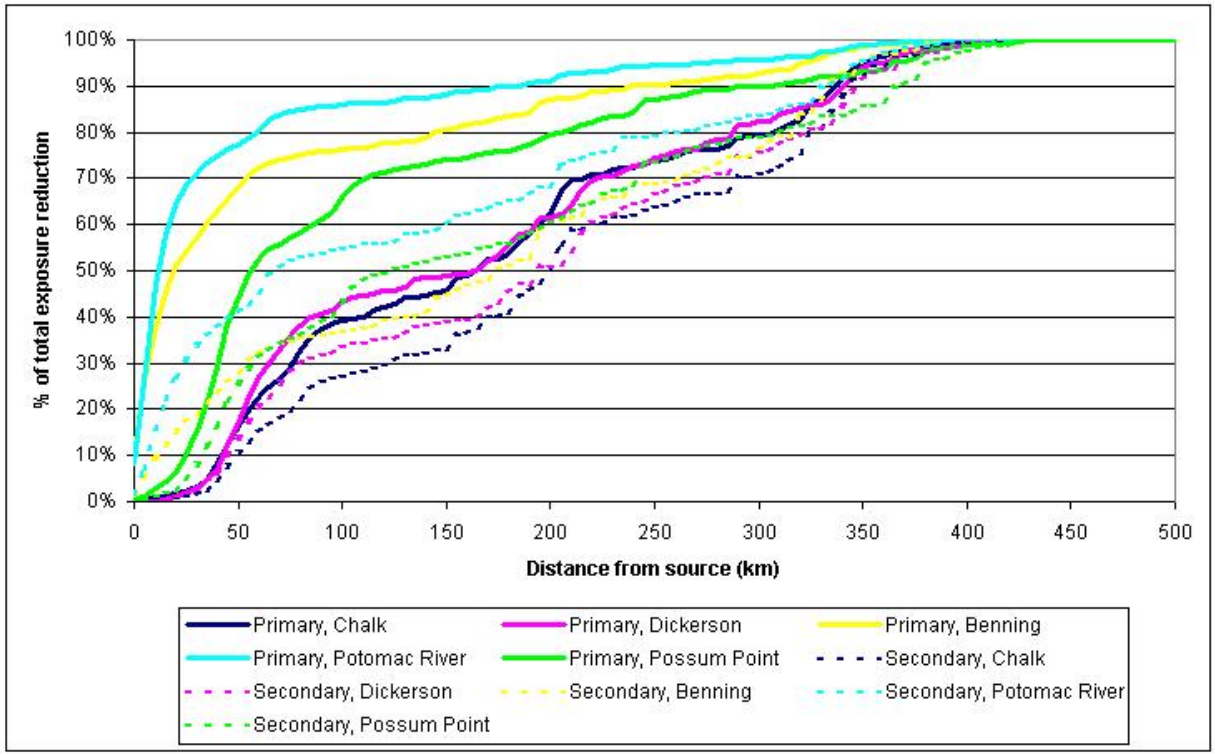


Figure 4: Distribution of health benefits by census tract in Washington, DC (no color indicates zero at-risk population).