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Maximizing Health Benefits and Minimizing Inequality: Incorporating Local-Scale Data in the Design and Evaluation of Air Quality Policies

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The U.S. Environmental Protection Agency undertook a case study in the Detroit metropolitan area to test the viability of a new multipollutant risk-based (MP/RB) approach to air quality management, informed by spatially resolved air quality, population, and baseline health data. The case study demonstrated that the MP/RB approach approximately doubled the human health benefits achieved by the traditional approach while increasing cost less than 20%—moving closer to the objective of Executive Order 12866 to maximize net benefits. Less well understood is how the distribution of health benefits from the MP/RB and traditional strategies affect the existing inequalities in air-pollution-related risks in Detroit. In this article, we identify Detroit populations that may be both most susceptible to air pollution health impacts (based on local-scale baseline health data) and most vulnerable to air pollution (based on fine-scale $PM_{2.5}$ air quality modeling and socioeconomic characteristics). Using these susceptible/vulnerable subpopulation profiles, we assess the relative impacts of each control strategy on risk inequality, applying the Atkinson Index (AI) to quantify health risk inequality at baseline and with either risk management approach. We find that the MP/RB approach delivers greater air quality improvements among these subpopulations while also generating substantial benefits among lower-risk populations. Applying the AI, we confirm that the MP/RB strategy yields less $PM_{2.5}$ mortality and asthma hospitalization risk inequality than the traditional approach. We demonstrate the value of this approach to policymakers as they develop cost-effective air quality management plans that maximize risk reduction while minimizing health inequality.

KEY WORDS: BenMAP; inequality; $PM_{2.5}$; susceptible; vulnerable

1. INTRODUCTION

The U.S. Environmental Protection Agency (EPA) recently undertook a case study focused on the Detroit metropolitan area to test the viability of a new multipollutant risk-based (MP/RB) approach to air quality management, informed by spatially resolved air quality concentrations, population, and baseline health data. This analysis demonstrated that, as compared to a traditional pollutant-by-pollutant approach to air quality management (i.e., the status-quo [SQ] scenario), utilizing local data and emphasizing multipollutant emission reductions aimed at reducing overall air-pollution-related risk (i.e., a multipollutant, risk-based strategy) produces a strategy that yields approximately

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double the total monetized benefits at a modestly higher cost.[1–3] Were this analysis to support a federal regulation, it would move closer to the objective of Executive Order 12866 (requiring agencies to perform a cost-benefit analysis of regulations) and OMB Circular A-4 (specifying the content of regulatory impact analysis) to maximize net benefits, by satisfying the stipulation to consider economic, environmental, and public health benefits.[4,5] However, less clear was whether the MP/RB approach would also meet the directives of this same Executive Order to include considerations of distributive impacts and equity in maximizing net benefits—or the requirements of Executive Order 12898 to address "disproportionately high and adverse human health . . . effects . . . ."[6]

Though information regarding the distribution of benefits—and not just the size—are critical to the complete evaluation of regulatory policy, Farrow (2010) argues that such considerations are ". . . seldom incorporated into regulatory impact analyses in general and into benefit–cost analysis in particular."[7] The tendency to not explicitly consider equity implications in such analyses may be due to what Adler (2008) observes is the lack of a "clear paradigm for equity analysis [in] governmental practice."[8] Neither the Executive Orders nor OMB Circular A-4 indicate how regulatory agencies might meet both equity and efficiency goals, and what constitutes a population that is "disproportionately affected" by environmental risk.[4] However, a rich literature indicates that certain populations may have a greater likelihood of experiencing adverse health effects due to a variety of characteristics, including race, gender, lifestyle, life stage, exposure to ambient air pollution, socioeconomic factors affecting access to health care, and residential location, among other factors.[9,10] Yet, few studies have adequately incorporated such information into health benefits analyses or formally considered the implications of differential health effects from an equity standpoint.

Addressing these issues is further complicated by the various ways in which the aforementioned factors can influence health, and a lack of consistent definitions for population susceptibility and vulnerability, two key dimensions of differential health effects. For example, U.S. EPA finds that susceptibility describes the degree to which a given population experiences a "greater or lesser biological response to exposure."[9] The American Lung Association defines susceptibility as the "greater likelihood of an adverse outcome given a specific exposure . . . [that] includes both host and environmental factors (e.g., genetics, diet, physiological state, age, gender, social, economic, and geographic attributes."[11] Pope and Dockery[12] find that susceptibility describes those "characteristics that contribute to increased risk of PM-related health effects (e.g., genetics, preexisting disease, age, gender, race, socioeconomic status, healthcare availability, educational attainment, and housing characteristics)."

This same literature produces a variety of definitions of population vulnerability, including the propensity for "harm or neglect . . . [including] acts of commission or omission on the part of others that can cause wound."[13] U.S. EPA defines vulnerability as "differential exposure and differential preparedness (e.g., immunization)."[14] This Agency has also described vulnerability as "PM-related effects due to factors including socioeconomic status . . . ." [15] Thus, there are numerous factors that describe populations that are vulnerable and/or susceptible to air pollution health impacts—though there remains ambiguity in the literature as to which of these factors describe vulnerability, susceptibility, or both. However, the high-resolution air quality, population, baseline health, and socioeconomic data available through the Detroit case study provide a unique opportunity to develop a spatially resolved representation of many of the above attributes. And, as we discuss in detail below, to the extent that these attributes can be measured among the population and such individuals are clustered in particular locations of the domain, local emission controls could be targeted to affect these groups. Such an approach would advance EPA's goal of reducing the public health burden borne by these populations[16] and make progress toward the directives stipulated in the Executive Orders and Circular A-4 guidance.[5]

In addition to the ex ante value of considering local data when designing air quality strategies, local data can inform metrics that evaluate the expected ex post changes in the distribution of air-pollution-related health risks associated with each strategy. Recent research has successfully applied indicators commonly used for income inequality to human health risk data to compare the distribution of risks across competing air quality management plans.

[4] The Office of Information and Regulatory Affairs recently released an "Agency Checklist" for Regulatory Impact Analyses in which it asks whether the RIA "analyze[s] relevant effects on disadvantaged or vulnerable populations (e.g., disabled or poor)," referring readers to Circular A-4 for more information.

In a 2006 paper, Levy *et al*. identified the Atkinson Index (AI), an inequality indicator of the generalized entropy family that is subgroup decomposable, as the most suitable, based on a set of criteria discussed below, for assessing human health inequality impacts that could complement assessments of the efficiency (or net benefits) of pollution control strategies.[17] Levy *et al*. applied the AI in two subsequent papers, one focused on efficiency and inequality impacts of alternative strategies to reduce multipollutant emissions from power plants nationwide, and the second focused on alternative fine particulate matter ($PM_{2.5}$) control scenarios for urban transportation bus fleets in the Boston area.[18,19] As part of our analysis we build on this literature base, using the AI to compare the effects of the SQ and MP/RB air quality management strategies on the distribution of health risks in the Detroit population, as well as its effects on sensitive and vulnerable subgroups.

This analysis leverages the spatially resolved air quality, population, and baseline health data for the Detroit area to determine: (1) how the air-quality-related health benefits of the two strategies were distributed across the population; (2) the extent to which those segments of the population with the highest baseline risk of air pollution health impacts would benefit; and (3) how effective either strategy was in reducing the overall level of health risk inequality, measured by the AI, across the population. In the absence of a generally accepted analytical approach, we utilized multiple measures of vulnerability and susceptibility as indicators of populations that may be at disproportionate risk of air pollution health impacts, before assessing the extent to which either risk management scenario affected the distribution of health risk among these populations. We also consider how the ex ante procedure for identifying vulnerable and susceptible populations and the ex post application of the AI can be used jointly to design air quality management strategies that satisfy both Executive Order 12866 and 12898.

2. METHODS

The Detroit case study simulated the annual mean $PM_{2.5}$ air quality changes resulting from two alternate emission control scenarios, each designed to meet contrasting policy goals. We first describe the key attributes of these two strategies before summarizing the input parameters for the health impact function used to quantify the change in the incidence of PM-related premature mortalities and asthma hospital visits. Finally, we describe how we adapted the health impact function data inputs to identify vulnerable and susceptible populations.

2.1. Designing the Air Quality Control Scenarios

Wesson *et al*. constructed two emission control scenarios for the Detroit metropolitan area.[1] The first was meant to emulate an approach that air pollution control officials might follow when developing a "State Implementation Plan" to attain the National Ambient Air Quality Standards (NAAQS) for $PM_{2.5}$ and ozone.[1] This strategy aimed to maximize decreases in $PM_{2.5}$ and ozone air quality levels at monitors projected to violate each of the two standards, aiming to attain the standards at minimum total cost. The controls for this strategy were based on the "Ozone Attainment Strategy for Southeast Michigan" submitted to EPA in June 2005 by the Michigan Department of Environmental Quality and the U.S. EPA Regulatory Impact Analysis (RIA) of the revised NAAQS for $PM_{2.5}$.[20,21] We term this strategy the SQ.

By contrast, the second strategy sought to achieve at least the same air quality improvements at violating monitors while also maximizing overall $PM_{2.5}$ and ozone-related human health benefits across the Detroit population. Drawing upon information including census population data, monitor speciation profiles, and detailed local emission inventories, EPA constructed a control strategy that would meet or exceed the $PM_{2.5}$ and ozone reductions at violating monitors achieved by the SQ strategy, while also maximizing air quality improvements in highly populated areas (which would tend to maximize population health benefits). We refer to this strategy as "multi-pollutant/risk-based" (MP/RB). Fig. 1 provides a conceptual overview of this multipollutant risk-based modeling framework, describing how estimates of air quality changes and health benefits were each used to inform the selection of emission controls for the MP/RB strategy.

In general, EPA maintained the same overall level of emission reductions between the two strategies to make the comparison of benefits and costs more straightforward, recognizing that fully maximizing net benefits could lead to larger or smaller overall emissions decreases. Relative to the SQ, the MP/RB strategy included more reductions of directly emitted $PM_{2.5}$ but lower emission reductions of SO_2, resulting in a control strategy more heavily weighted towards direct $PM_{2.5}$ reductions. Compared to the SQ, the MP/RB strategy applied more emission controls to the metals processing

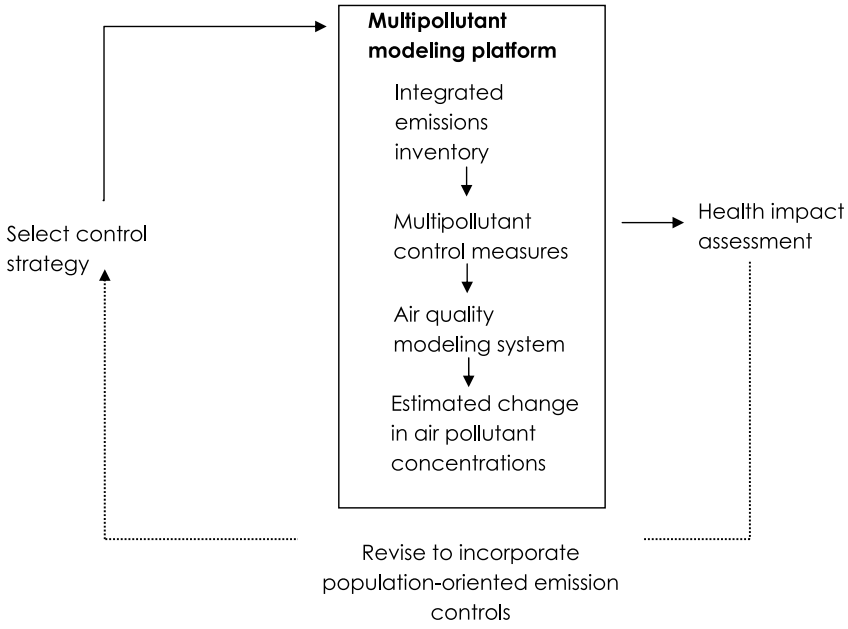

Fig. 1. Risk-based, multipollutant modeling framework.

industry and mobile source sector and fewer controls to the electrical generating unit sector. For this analysis, we modified the MP/RB control strategy described by Wesson *et al.*[1] by omitting 142 tons of $PM_{2.5}$ reductions from two steel mills, a 4% change in the overall reduction in directly emitted $PM_{2.5}$. This adjustment was made to ensure that the cost of the SQ and MP/RB control strategies were equal for this analysis, further facilitating comparisons across the two scenarios.

2.2. Estimating Air Pollution Health Impacts

This analysis uses health impact functions to quantify the change in the number of cases of $PM_{2.5}$-related premature mortality and asthma hospitalizations, which we use as sample outcomes for which data exist for differential relative risks and/or baseline incidence rates. A standard health impact function has four components: the affected population, the change in air quality, the baseline incidence rate, and the effect estimate drawn from the epidemiological studies.[22,23] We utilize the environmental Benefits Mapping and Analysis Program (BenMAP) to perform this calculation.[24] We summarize the data inputs below.

2.2.1. Selection of Baseline Incidence Rates

Because epidemiological studies assess changes in risk relative to some baseline rate, it is necessary to specify a baseline incidence rate for each health endpoint. The Michigan Department of Community Health (MDCH) provided ZIP code level all-cause mortality rates by 10-year age range for the time period 2006–2008. Rates at this spatial resolution are not commonly available from publicly available databases due to privacy concerns, necessitating a special data query by MDCH that masked personally identifiable information. MDCH also provided ZIP code level hospital discharge rates for asthma (ICD-9493) for 2006 for ages <20, 20–64, and >65. Compared to national or regional rates, location-specific and spatially resolved baseline incidence rates provide a more accurate characterization of the baseline health status of the population of interest.[23] As described below, these Detroit-specific rates are a key input to the procedure for identifying populations susceptible to air pollution health impacts.

2.2.2. Selection of Concentration-Response Relationships

For this analysis, we focus on premature mortality and asthma-related hospital admissions in relation to $PM_{2.5}$ exposure. We assess changes in the incidence of these endpoints using concentration-response functions that best match the finely resolved mortality and hospital discharge data we use as an input to the health impact function (see later).

We draw our concentration-response function for $PM_{2.5}$ mortality from the American Cancer

Society (ACS) cohort.[25] The ACS cohort is large and geographically diverse—covering over 116 urban areas throughout the United States—and previous assessments of $PM_{2.5}$ mortality impacts using risk estimates drawn from studies of this cohort have been vetted thoroughly.[26] Perhaps as importantly in this context, the ACS cohort study also includes mortality concentration-response estimates stratified according to the highest level of education achieved by the exposed population. Krewski *et al*. find that educational attainment is inversely related to all-cause PM mortality risk, noting that "[a]lthough the reasons for this finding are unknown . . . level of education attainment may likely indicate the effects of complex and multifactorial socioeconomic processes on mortality or may reflect disproportionate pollution exposures."[27]

Thus, we elected to draw risk coefficients from the recent extended analysis of the ACS cohort.[27] We applied the adult (age > 29) all-cause mortality risk estimates from Krewski *et al*. based on the random effects Cox proportional hazard model with 44 individual-level covariates and seven ecological covariates for the change in $PM_{2.5}$ occurring 1999–2000 among 116 U.S. cities (RR = 1.06, 95% confidence intervals 1.04–1.08 per 10 $\mu g/m^3$ increase in $PM_{2.5}$). The authors also specify a model in which they estimate education-modified risk for this same time period, reporting all-cause mortality risk stratified by educational attainment: less than grade 12 (RR = 1.082, 95% confidence intervals 1.024–1.144 per 10 $\mu g/m^3$ change), grade 12 (RR = 1.072, 95% confidence intervals 1.020–1.127 per 10 $\mu g/m^3$ change), and greater than grade 12 (RR = 1.055, 95% confidence intervals 1.018–1.094 per 10 $\mu g/m^3$ change). Using these latter risk estimates allows us to more accurately estimate mortality impacts among populations vulnerable to $PM_{2.5}$-related premature mortality.

When estimating the change in $PM_{2.5}$-related asthma hospital visits, we selected a concentration-response estimate from a study in Seattle examining nonelderly (<65) hospital admissions for asthma from 1987 to 1994.[28] While there are multiple other such publications, in this article we elected to select a risk estimate that has been used in previous EPA Regulatory Impact Analyses.[20] From this article we draw a risk estimate based on the 1-day lag GAM stringent model (RR = 1.04, 95% confidence intervals 1.01–1.06 per 10 $\mu g/m^3$ change).

When quantifying the incidence of $PM_{2.5}$-related impacts using these studies we estimate confidence intervals using a Monte Carlo analysis based on the standard error reported in each epidemiological study. While this does not fully capture all uncertainties associated with study selection and geographic extrapolation, as transferring concentration-response relationships from one context to another introduces some uncertainty to the analysis,[23] our risk management conclusions are not influenced by this limitation. Differences in population exposure and susceptibility between the study site and the location of the health impact assessment can introduce error. However, in the absence of a locally developed concentration-response function for both premature mortality and asthma hospitalizations, we believe it was appropriate to apply these estimates to Detroit.

2.2.3. Air Quality Modeling Estimates

Our analytical approach to estimating $PM_{2.5}$ air quality levels in Detroit followed three key steps: (1) the development of an air quality modeling "platform" containing emissions inventory and air quality monitoring data; (2) running the Community Multi-Scale Air Quality model (CMAQ) to estimate the $PM_{2.5}$ levels associated with each scenario; and (3) running the EPA Model Attainment Test Software (MATS) to combine the air quality modeling estimates with monitored air quality data (Fig. 1).[29,30]

This analysis applied the 2002 Modeling Platform[31] that contained emission inventory updates making it more reflective of the Detroit urban area[1] and subsequently processed with the Sparse Matrix Operator Kernel Emissions (SMOKE) model that allocated these emissions to each CMAQ grid cell. We applied the CMAQ model to estimate both initial condition and boundary conditions (ICBCs) and air quality changes for each of the scenarios. CMAQ v.4.6.1i was run for January and July 2002 for three domains, centered on Detroit, Michigan, each with a different grid resolution: 12 km × 12 km, a 4 km × 4 km, and 1 km × 1km (Supplementary Fig. S1). The CMAQ model applies a generalized chemistry mechanism, general numerical solver, and comprehensive description of gaseous and aqueous chemistry and modal aerosol dynamics. ICBCs were supplied from a previously completed CMAQ run for the continental United State at a 36 km × 36 km grid resolution to run CMAQ at a 12 km × 12 km resolution for the Midwest domain.[32] ICBCs were supplied to the 4 km × 4 km CMAQ model run from a CMAQ 12 km × 12 km grid resolution run, and to

the 1 km × 1 km CMAQ model run from a CMAQ 4 km × 4 km grid resolution run. The meteorological inputs for the 12 km CMAQ domain were derived from MM5 data that were processed to create model-ready inputs using the Meteorology-Chemistry Interface Processor (MCIP), version 3.4.[32] For the 4 km and 1 km domains, meteorological data were provided by disaggregating the 12 km MCIP output meteorology to the 4 km and 1 km domains. Model performance was evaluated for all three domains for $PM_{2.5}$ and shown to be adequate.[33] Based on the available measurements, CMAQ performed as well at the finer scales (i.e., 1 km and 4 km resolution) as for the 12 km resolution domain.

The 2002 base year inventory was then projected to create a 2020 future year emissions inventory, taking into account any national rules or "on the books" controls and any growth or decline of an emissions source group. These data were modeled with CMAQ for January and July for the same 12 km, 4 km, and 1km domains using the same meteorological inputs. We also modeled the two control strategies, SQ and MP/RB, where emissions reductions were applied to the 2020 base year emissions inventory. Using the EPA MATS,[30] modeled concentrations were combined with ambient monitoring data from 2002 to 2004 to simulate the response of the atmosphere to changes in emissions between 2002 and 2020 and those resulting from the SQ and MP/RB control strategies. Three sets of 2020 $PM_{2.5}$ air quality estimates were created for the 1 km domain: a 2020 base case (i.e., the absence of a policy change), the SQ strategy, and the MP/RB strategy. While the Detroit case study modeled changes in $PM_{2.5,}$ ozone, and air toxics, this article focuses on the distributional implications of $PM_{2.5}$ air quality changes. $PM_{2.5}$ was the pollutant most affected by the two control strategies in the case study. Ozone displayed more spatially homogeneous changes in population exposure, and air toxics risk calculations are not compatible with the procedure for identifying vulnerable and susceptible populations.

2.2.4. Estimation of Air Quality Changes Across the Population

We use U.S. Census block-level population data[34] aggregated to each 1 km cell used by CMAQ. Population for the year 2000 is stratified by age, sex, race, and ethnicity categories corresponding to the demographic classifications considered in the health impact functions and is projected to 2020 using an economic forecasting model that accounts for the relationship between economic growth and population size and distribution.[35] We next allocated the modeled incremental change in $PM_{2.5}$ concentrations between the baseline scenario and each of two policy cases with the population projected in each 1 km grid cell, assuming that the modeled air quality value in each cell is the best measure of population exposure.

2.3. Procedure for Identifying Vulnerable and Susceptible Populations

2.3.1. Defining Susceptibility and Vulnerability

To the extent that certain attributes contributing to either susceptibility or vulnerability can be detected among the population, then a profile of susceptible and vulnerable individuals can be constructed and the geographic distribution of these populations determined. However, many of the factors described above—in particular genetics and preexisting disease—are very difficult to measure or model using publicly available data. Moreover, the literature does not provide a consistent definition of vulnerable and susceptible and certain factors (e.g., socioeconomic status) are referenced in the definition of both terms, though potentially proxying for different causal pathways in each case.

Given these ambiguities, we test a variety of approaches for constructing profiles of susceptible and vulnerable individuals, using combinations of available baseline health, demographic, education, poverty, and air quality data. In general, we use baseline health and demographic data to construct profiles of susceptible populations; we use a combination of demographic, education, poverty, and air quality data to develop profiles of vulnerable populations. Finally, we evaluate the extent to which these alternate approaches identify the same populations at greatest risk of air pollution health impacts.

2.3.2. Identifying Populations Susceptible to Air Pollution Health Impacts

While the literature points to a variety of attributes as indicators of susceptibility, many of these are difficult to measure based on readily available data, making them difficult to employ in this analysis. We allocate age-standardized rates of all-cause mortality and asthma hospitalizations from the ZIP code level to the 1 km CMAQ grid, and use these

as the basis for our susceptibility analysis because these sets of data are spatially resolved and are a reasonably good surrogate for the baseline health status, which we use as a proxy for indicating physical susceptibility of the population of the Detroit metropolitan area.[5] Moreover, these data are a key input to the health impact function we use to predict the excess cases of air-pollution-related health effects. All else equal, we expect to find a larger number of excess cases of asthma hospitalizations and premature mortality due to air pollution where the baseline incidence rates for these two endpoints are elevated. This is largely due to the fact that the health impacts are based on $PM_{2.5}$ risks that are proportional to baseline risk.

While we calculated excess cases of mortality and asthma hospitalizations using crude rates, for the purposes of identifying susceptible populations we use age-standardized rates. As noted by Levy *et al.* (2009),[18] using age-standardized rates for this calculation allows us to capture distributions considered to be more concerning from an equity standpoint (e.g., populations with a high mortality rate who are more susceptible to air-pollution-related mortality) rather than populations that are simply older.[18] Plotting these incidence rates in ArcGIS® version 9.3 (ESRI, Redlands, CA, USA) reveals the location of susceptible populations. By calculating various percentile cutoffs, we can identify the portions of the Detroit metropolitan area that contain populations at the 50th, 75th, 90th, and 95th percentile of the distribution of susceptible populations.

2.3.3. *Identifying Populations Vulnerable to Air Pollution Health Impacts*

We constructed profiles of vulnerable populations using two alternate approaches that are supported within the epidemiological literature. The first is consistent with the EPA definition of vulnerability that is based on "differential exposure" to air pollution.[14] We first plot the spatial distribution of the annual mean $PM_{2.5}$ concentration for each 1 km CMAQ grid cell using the 2020 CMAQ-modeled base case projections. The results of this calculation indicate the location of those 1 km grid cells containing populations most exposed to $PM_{2.5}$ in the baseline scenario. We then identified those 1 km cells at the 50th, 75th, 90th, and 95th percentiles of exposure.

An alternate approach relies on a combination of data on educational attainment and poverty rates. As noted above, multiple studies[25,27] point to the role of socioeconomic status (for which educational attainment and income are indicators) as modifying PM mortality risk. The recent HEI extended analysis of the ACS cohort found educational attainment to be inversely related to all-cause PM mortality risk[27] (though this relationship was reversed for ischemic-heart-disease-related PM mortality risk). For these reasons we construct a profile of vulnerable populations based on a combination of educational attainment and poverty status. We allocate census block group-level data on educational attainment and poverty status to the 1 km grid (the same spatial scale as the air quality modeling estimates), and plot the spatial distribution of populations with lower educational attainment (<grade 12) and those below the poverty line, identifying those at the 50th, 75th, 90th, and 95th percentiles. When allocating the census block group-level data to the 1 km grid we assumed that the population within the block group was distributed uniformly, which is unlikely to add an appreciable amount of uncertainty to the analysis given their relatively small size. Populations whose education attainment was below grade 12 or whose income was below the poverty line were allocated from the block group to the 1 km cell according to a weighting algorithm. The weights are the percentage of the block group's total area overlapped by the grid cell.

2.3.4. *Locating Susceptible and Vulnerable Populations*

Intersecting each of the vulnerability and susceptibility layers in ArcGIS provides the location of those populations that fall within both categories as defined above. The large number of alternate approaches used to identify these populations yields a substantial number of combinations, only some of which we present in this article. In the absence of a universally agreed upon definition of vulnerable and susceptible, we focus on six combinations of the susceptibility and vulnerability data layers, and we test the sensitivity of our conclusions across these six combinations.

[5] To allocate the incidence rates, the following convention was employed. If a 1 km grid cell falls completely within a ZIP code, then it is assigned the rates for that ZIP code. If a 1 km grid cell overlaps multiple ZIP codes, then the grid cell is assigned an area-weighted average of the incidence rates for those ZIP codes.

2.4. Calculating the Change in Risk Inequality

The identification of susceptible and vulnerable subpopulations provides one approach to identify inequalities in air-pollution-related health risks faced by the study population. To more formally investigate changes in health inequality, we also assessed how the two air quality management strategies affect a quantitative measure of inequality in the distribution of risk across the Detroit population. We applied the AI as our primary measure of inequality, as discussed above, based on an analysis by Levy *et al*. (2006) that explored a variety of indicators and assessed their performance against a set of axioms the authors established for an inequality index in the context of health benefits analysis.[17] In addition to meeting basic criteria such as satisfying the Pigou-Dalton transfer principle (i.e., it does not decrease when risk is transferred from a low-risk to high-risk person, but does decrease when risk is transferred from a high-risk person to a low-risk person), the AI is subgroup decomposable, meaning the inequality can be broken down to address inequality within and between separate population subgroups. In addition, the AI allows for assessment of inequality using a range of different assumptions about the degree of societal concern about different portions of the risk distribution, allowing the risk assessor to avoid imposing implicit or explicit preferences that are better found within the domain of risk management. The AI ranges from zero to one, with zero being the most equal (i.e., every individual has equal risk) and one representing complete inequality (i.e., one individual has all of the population risk). All else equal, an air quality management approach with a lower AI compared to competing options is preferable. Policy analyses would typically use health benefits and the change in the AI associated with a management strategy as two separate endpoints, where policies with greater health benefits and greater reduction in the AI would be preferred, and policies involving tradeoffs between health benefits and the AI would require further evaluation.

The AI is defined as follows:

$$\mathrm{AI} = 1 - \left[\frac{1}{n} \sum_{i=1}^{n} \left[\frac{x_i}{\bar{x}} \right]^{1-\varepsilon} \right]^{\frac{1}{1-\varepsilon}},$$

where x_i is the risk for individual i, n is the number of individuals in the population, and ε is a parameter representing the degree of weight applied to the lower end of the distribution. The value of ε can range from zero to infinity, where zero represents no societal concern about inequality and higher values represent increasing concern about the low end of the distribution. It should be noted that, whereas having lower income is detrimental, having lower risk is beneficial; as such, the interpretation of ε is less straightforward for health risk than for income. As reported elsewhere,[17] typical values of ε in the literature range from 0.25 to 2, though more extreme values have been considered. We assign ε a value of 0.75 as our central estimate, as done previously,[17,18] but test the sensitivity of our conclusions by applying a lower-bound value of 0.25 and an upper bound value of 3.

In our primary model, we calculated the risk of mortality and asthma hospitalization attributable to $PM_{2.5}$ for each individual in the study area (x_i) using the age-stratified baseline incidence rates and health impact functions described above. For baseline risk, we considered risk attributable to $PM_{2.5}$ rather than overall risk as the more policy-relevant measure, and we estimated the change in incidence associated with $PM_{2.5}$ reductions to policy-relevant background. For the control scenarios, we adjusted the baseline risk according to the BenMAP results for the incremental change in incidence associated with the modeled change in $PM_{2.5}$ concentrations in each grid cell. We then calculated the AI for the baseline, SQ, and MP/RB scenarios for both asthma hospitalization and mortality risk for the entire study population. In addition, we separated the population into vulnerable and susceptible subgroups using the overlap of the 75th percentile of defined vulnerable and susceptible areas as described in Section 2.3.4, and we decomposed the AI to compare the degree to which inequality between the vulnerable/susceptible subpopulation and the rest of the study population contributes to the overall inequality in the study area. We calculated these between-group and within-group inequalities using the factorial approach to decomposing the AI, as described in Lasso de la Vega and Urrutia.[36] We conducted our calculations within a relational database in Microsoft Access using the risk estimates and population data generated in BenMAP.

In addition to our primary model, we completed several sensitivity analyses to test the robustness of our calculations. In our first sensitivity analysis, we used age-standardized rates to represent a single risk estimate for the population in each grid cell instead of rates stratified by age group. Second, we

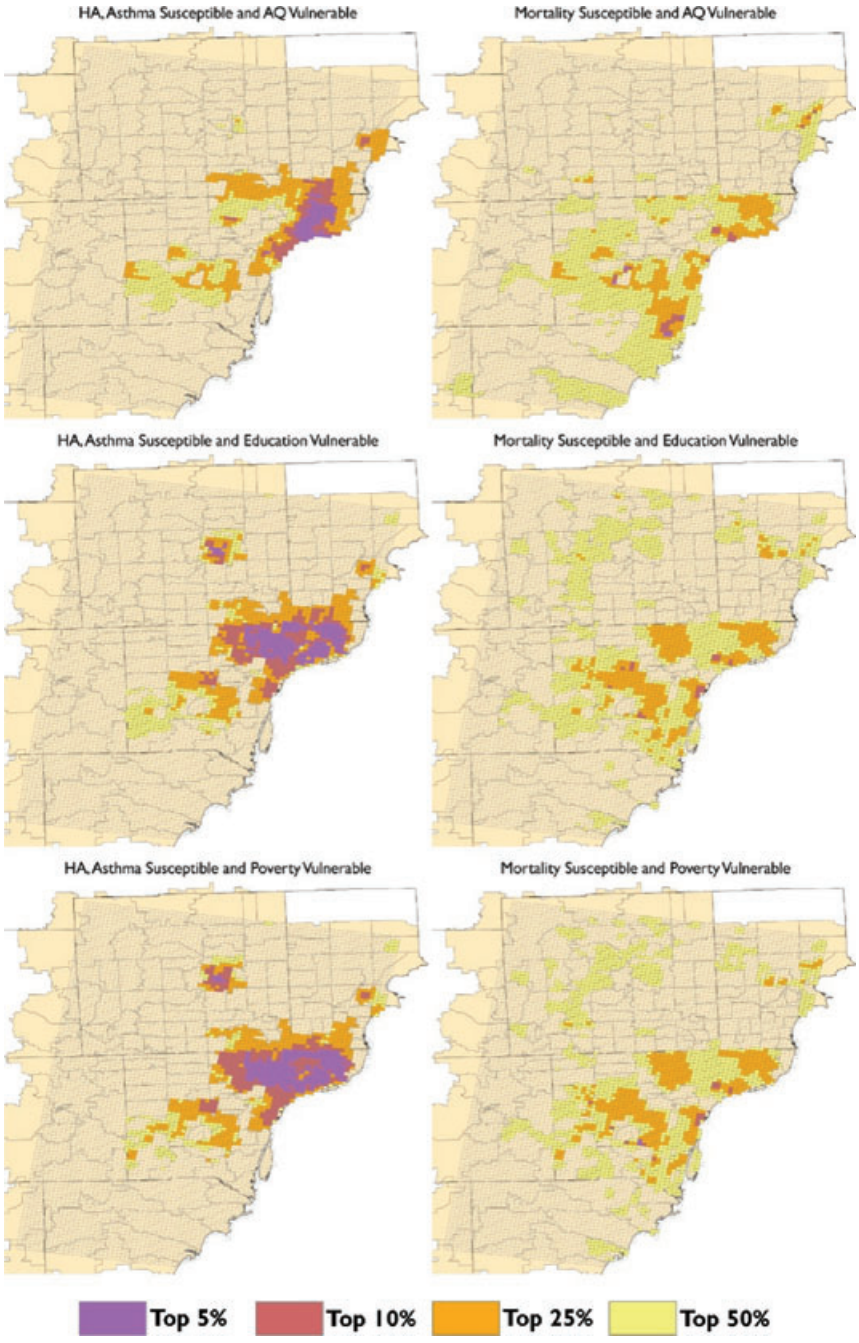

Fig. 2. Location of populations identified as vulnerable and susceptible to air pollution health impacts.

calculated AIs for the distribution in mortality risk derived using concentration-response coefficients stratified by educational attainment level. For this analysis, we decomposed the AI by education level. As mentioned above, we also tested the sensitivity to the value of ε using values of 0.25 and 3. The former value reflects reduced concern about inequality, and the latter value increases the AI's sensitivity to values at the lower tail of the distribution. In addition, we ran our calculations using inverse risk, to see if the results are robust when looking at a data set where larger values represent a better outcome. Finally, we calculated an alternative inequality metric, the Gini coefficient, to see if our results are robust to the choice of inequality measure. The Gini coefficient is an alternative metric explored and selected as an indicator useful for sensitivity analysis by Levy *et al.*[19]

Table I. Avoided Health Impacts for Status-Quo and Multipollutant/Risk-Based Scenarios (90% Confidence Interval)[a]

	Air Quality Scenario			
Health Endpoint	Status-Quo		Multipollutant/Risk-Based	
Mortality (populations 30–99)[b]	71 (51–91)		130 (95–170)	
Asthma hospitalization (populations <65)	6.8 (3.3–10)		16 (7.6–24)	
Education-Stratified Estimates[c]	Incidence Estimate	Percent of Total[a]	Incidence Estimate	Percent of Total[a]
Mortality (populations 30–99, <grade 12)	18 (7.3–28)	24%	40 (16–63)	28%
Mortality (populations 30–99, = grade 12)	24 (9.6–38)	32%	46 (19–74)	32%
Mortality (populations 30–99, >grade 12)	34 (15–53)	45%	58 (25–90)	40%

[a]Percentage may not sum to 100 due to rounding.
[b]Calculated using all-cause mortality risk estimate.
[c]Calculated using all-cause mortality risk estimate stratified by educational attainment.

3. RESULTS

3.1. Estimated Change in Exposure and Health Impacts Among Susceptible and Vulnerable Populations

When comparing each of the six maps of vulnerability and susceptibility (Fig. 2), certain patterns emerge. First, when paired with either asthma or mortality as a measure of susceptibility, both education and poverty appear to indicate that a fairly common spatial pattern of the Detroit population is vulnerable to air pollution. In particular, the rates for both mortality and asthma hospitalizations are at or above the 90th percentile in the grid cells in the northeastern corner of Wayne County, which also corresponds closely with an area of low socioeconomic status. Second, the use of asthma and mortality measures of susceptibility result in somewhat different spatial distributions of both susceptibility and vulnerability—suggesting that the populations affected by asthma and premature mortality are located in different portions of the city.

Table I summarizes the estimated change in asthma hospitalizations among populations <65 and all-cause premature mortality among populations ages 30–99 and stratified according to educational attainment for this same age range. Consistent with the findings of Wesson *et al.*[1] the MP/RB air quality management strategy provides greater health benefits than the SQ approach on an absolute basis for each health endpoint quantified. In addition, the MP/RB strategy delivers a larger percentage of mortality benefits among populations with a lower level of educational attainment (28% for the MP/RB vs. 24% for the SQ), which the literature suggests to be more vulnerable to PM mortality. This result is likely related to the spatial correlation in this setting between low-education populations and population density (Supplementary Fig. S2).

Table II reports the per-person change in annual mean $PM_{2.5}$ levels among populations identified as being both vulnerable and susceptible to air pollution impacts according to each of the different approaches described above. We find that, regardless of which of the six definitions of vulnerable and susceptible applied, the MP/RB strategy provides the greatest population-weighted $PM_{2.5}$ change among these subpopulations. The SQ approach delivers roughly equal reductions in exposure among the vulnerable and susceptible populations and the populations at lower risk. By comparison, the MP/RB strategy provides between 1.6 and 2.2 times the $PM_{2.5}$ exposure reduction in these vulnerable and susceptible populations when using the classifications based on the mortality rate combined with educational attainment and the asthma hospitalization rate and baseline $PM_{2.5}$ exposure, respectively.

While the MP/RB is superior to the SQ in its ability to reduce $PM_{2.5}$ exposure to these key subpopulations, the magnitude of the population-weighted air quality change is somewhat sensitive to the definition applied. For example, when defining vulnerable and susceptible populations according to baseline incidence rates and air quality exposure, the selected portion of the population sees a substantial exposure reduction under the MP/RB strategy—much more so than the alternative two definitions that rely on educational attainment or poverty status as a measure of vulnerability. This is logical given the construction of MP/RB as targeting directly emitted $PM_{2.5,}$ which will tend to correlate with areas of elevated baseline $PM_{2.5}$ exposure.

Table II. Reduction in Annual Mean $PM_{2.5}$ Exposure per Person, According to Air Quality Scenario and Approach to Identifying Vulnerable & Susceptible Populations ($\mu g/m^3$/person)[a]

	PM Changes Among Vulnerable & Susceptible Populations		PM Changes Among Rest of Population		Ratio of PM Changes Among Vulnerable & Susceptible Populations to PM Changes Among Rest of Population	
Approach to Identifying Vulnerable & Susceptible Populations	Status-quo	Multipollutant, Risk-Based	Status-Quo	Multipollutant, Risk-Based	Status-Quo	Multipollutant, Risk-Based
Rate of asthma hospitalizations and level of baseline $PM_{2.5}$ exposure	0.3	1.04	0.28	0.48	1.1	2.2
Rate of asthma hospitalizations and educational attainment < grade 12	0.29	0.79	0.28	0.45	1	1.8
Rate of asthma hospitalizations and poverty rate	0.28	0.77	0.28	0.44	1	1.8
Mortality rate and level of baseline $PM_{2.5}$ exposure	0.29	0.96	0.28	0.53	1	1.8
Mortality rate and educational attainment <grade 12	0.26	0.85	0.28	0.53	0.9	1.6
Mortality rate and poverty rate	0.24	0.87	0.28	0.53	0.8	1.7

[a]Estimates rounded to two significant figures.

Table III. Primary Atkinson Results and Sensitivity Analysis

	Atkinson Indices						Gini Coefficient	
	Mortality Risk, $\varepsilon = 0.75$	Asthma Risk, $\varepsilon = 0.75$	Mortality Risk, Age-Standardized, $\varepsilon = 0.75$	Mortality Risk, $\varepsilon = 3$	Inverse Mortality Risk, $\varepsilon = 0.75$	Mortality Risk, Edu-Stratified, $\varepsilon = 0.75$	Mortality Risk	Asthma Risk
Baseline	0.437776	0.300454	0.024662	0.776033	0.276517	0.458081	0.633641	0.517979
Status-quo	0.437661	0.300614	0.025006	0.775956	0.276487	0.457960	0.633557	0.518123
% Change from baseline	*−0.026%*	**0.053%**	**1.394%**	*−0.010%*	*−0.011%*	*−0.026%*	*−0.013%*	**0.028%**
Multipollutant risk-based	0.436940	0.293722	0.023517	0.773048	0.273343	0.457899	0.633231	0.512064
% Change from baseline	−0.191%	−2.241%	−4.643%	−0.385%	−1.148%	−0.040%	−0.065%	−1.142%

Percentages in italic indicate improvement from the baseline. Percentages in bold indicate greater inequality. Atkinson indices are presented with a large number of significant figures to allow for comparisons between scenarios.

3.2. Estimated Change in Inequality Indices

Table III shows the primary AI results for mortality risk and asthma hospitalization risk, respectively, noting that the table displays AI values with far more significant figures than would be warranted in order to illustrate small changes in values. For both asthma and mortality, the MP/RB approach does a better job than the SQ approach of reducing inequality in risk due to $PM_{2.5}$ in the study population as a whole. In our primary analysis of mortality risk attributable to $PM_{2.5}$ (using age-stratified rates), both the SQ and MP/RB approaches reduce inequality across the population, though the MP/RB approach yields greater reductions. Looking at the risk of asthma hospitalization attributable to $PM_{2.5}$, the SQ approach increases inequality across the population, while the MP/RB approach reduces the level of inequality across the population.

The decomposition of the primary AI results is shown in Tables IV and V. Decomposition of the AI for mortality risk shows that the within-group AI is more than two orders of magnitude greater than the between-group AI, and this result persists no matter how you define the susceptible/vulnerable subgroup (air quality, education, or poverty-based). Thus, for mortality risk, it is clear that the vast majority of inequality in risk is attributable to within-group differences (i.e., differences resulting from individual-level variation, including geographic location) and little of the inequality reflects differences in

Table IV. Age-Stratified PM-Related Mortality Risk Atkinson Inequality Indicator and Decomposition, $\varepsilon = 0.75$

		Vulnerable/Susceptible Decomposition, Mortality Susceptible and AQ Vulnerable		Vulnerable/Susceptible Decomposition, Mortality Susceptible and Poverty Vulnerable		Vulnerable/Susceptible Decomposition, Mortality Susceptible and Education Vulnerable	
	Atkinson	Between	Within	Between	Within	Between	Within
Baseline	0.437776	0.002749	0.436227	0.002823	0.436185	0.002761	0.436220
Status-quo	0.437661	0.002803	0.436081	0.002857	0.436050	0.002793	0.436086
Multipollutant risk-based	0.436940	0.002318	0.435632	0.002525	0.435514	0.002477	0.435542

Table V. Age-Stratified PM-Related Asthma Hospitalization Risk Atkinson Inequality Indicator and Decomposition, $\varepsilon = 0.75$

		Vulnerable/Susceptible Decomposition, Asthma Susceptible and AQ Vulnerable		Vulnerable/Susceptible Decomposition, Asthma Susceptible and Poverty Vulnerable		Vulnerable/Susceptible Decomposition, Asthma Susceptible and Education Vulnerable	
	Atkinson	Between	Within	Between	Within	Between	Within
Baseline	0.300454	0.062175	0.254076	0.178242	0.148720	0.176483	0.150538
Status-quo	0.300614	0.062191	0.254234	0.178054	0.149109	0.176275	0.150947
Multipollutant risk-based	0.293722	0.057493	0.250638	0.171416	0.147609	0.169620	0.149451

risk across population subgroups. For asthma, there is more substantial between-group variability in comparison with within-group variability, though less so when the vulnerable and susceptible group is defined by air quality vulnerability. When the vulnerable and susceptible group is defined by poverty or education, however, the within-group and between-group AIs are very similar, indicating that a significant amount of the overall inequality is attributable to differences in risk between the vulnerable and susceptible group and the rest of the population.

To test the robustness of our results, we completed several sensitivity analyses as described in Section 2.4. Table III shows the AI for the baseline and two air quality management approaches using age-standardized baseline mortality rates. When standardizing rates (i.e., a single population weighted risk estimate for each grid cell), the AI increases from the baseline for the SQ approach, but decreases when looking at the MP/RB approach, again indicating that the MP/RB approach performs better than the SQ approach at reducing inequality in risk. We do note that overall inequality is much lower using standardized rates, resulting from using a single risk estimate per grid cell rather than multiple stratified estimates.

Other sensitivity analyses also indicate that the MP/RB approach outperforms the SQ approach with regards to reducing inequality. When we used bounding values for ε to represent greater or lesser societal concern about individuals at the lower tail of the distribution, the MP/RB approach reduced inequality to a greater extent than the SQ approach. For example, with an ε of 3, the AI was increased in all cases relative to an ε of 0.75, but the MP/RB approach led to a 0.385% reduction in the AI, versus a 0.01% reduction for the SQ approach. We also calculated the AI using inverse risk to create a scenario where increasing values represent an improved situation, and saw similar results.

When we use the education-stratified risks, overall inequality across the population increases slightly, as more variation in risk is introduced. However, when comparing air quality management approaches the trends remain consistent, with the MP/RB approach outperforming the SQ approach. When we decomposed the AI by education level, we saw that the majority of the inequality in the population is attributable to within-group differences.

As a final sensitivity analysis, we calculated inequality in health risks in the study population as a whole using the Gini coefficient. For mortality risk,

the results are consistent with our findings using the AI; both air quality management approaches reduce inequality in the distribution of risk, with the MP/RB approach performing better. For asthma, the results are also consistent with our AI calculations, with inequality increasing under the SQ approach, and decreasing under the MP/RB approach.

Across all calculations and sensitivity analyses, the MP/RB approach outperformed the SQ approach in reducing inequality across the entire population. The decomposition of the index by vulnerable and susceptible subgroups indicated that most of the inequality in the population occurs at the individual level and not between groups for mortality. However, when between-group variation is a significant contributor to overall inequality (i.e., for asthma risk in the susceptible and vulnerable group defined by education or poverty), the MP/RB approach did a better job of reducing the between-group inequalities, indicating that it reduces asthma hospitalization risks to a greater degree for the susceptible and vulnerable group.

4. DISCUSSION

When considered jointly, information regarding population density, baseline health status, air quality exposure, and socioeconomic status can help inform emission control strategies that meet the equity and efficiency goals outlined in Executive Orders 12866 and 12898. These disparate sources of local data can be used to construct a profile of individuals who are at greatest risk of air pollution impacts so that air quality management policies can (to the extent possible) target emission controls to affect these populations. We describe several alternate approaches to building such profiles, allowing a measure of flexibility to local air pollution control officials as they consider how best to apply scarce resources to reduce exposure to certain subpopulations.

The methods described above are most useful in situations where: (1) there is some spatial clustering in the location of vulnerable and susceptible populations; and (2) there is a strong gradient in air quality due to local sources that are affecting these populations and can be controlled. We believe this to be true in most urban areas in the United States, where socioeconomic gradients can be significant and industrial and mobile sources contribute appreciably to ambient $PM_{2.5}$ concentrations. However, in more rural sections of the country, where air pollution concentrations are more uniform due to the absence of local emissions sources, these methods may not be as useful. We would expect air pollution in these areas to be primarily attributable to regional emission sources such that any controls affecting such sources would be likely to affect populations in the study area fairly equally. A similar situation would arise where the distribution of vulnerable and susceptible populations is found to be spatially homogeneous. In these situations the methods presented here may not be as informative to air pollution control officials in control strategy development.

Though the scenarios developed in Wesson *et al.* (2010) and applied in this analysis did not utilize this approach fully, we demonstrate that this MP/RB strategy succeeded in generating substantial human health benefits—particularly among vulnerable and susceptible populations—while also lowering the overall level of air pollution risk inequality as measured by the AI. These results demonstrate the utility of an ex-ante/ex-post approach to air quality management: first we construct vulnerable and susceptible population profiles to devise emission control strategies that minimize air pollution risk to these populations; and next we quantify the change in the AI after implementation of the strategy to assess the change in the distribution of risk.

The two air quality management policies considered in this article each sought to attain new air quality standards while meeting certain criteria—minimizing total costs (the SQ) or maximizing benefits and achieving multipollutant emission reductions (the MP/RB). However, the ex-ante/ex-post approach described in this article would clearly be useful for analyzing other policy alternatives that aimed to achieve both health benefits while reducing risk inequality. For example, policymakers might wish to design an air quality management plan that: maximizes overall population-level monetized benefits; reduces the level of risk for a specific health endpoint, such as asthma hospitalizations; and/or controls emissions from specific industrial activities.

There are some clear limitations to our findings that may influence the generalizability of our work. First, the identification of susceptible populations depends in part on information not publicly available for most health outcomes (highly spatially resolved baseline rates). That said, when working within a single geographic area, local health departments and other entities can often provide such information in a manner that would inform

efficiency and equity analyses. Similarly, we selected risk estimates from the epidemiological literature that were generally consistent with those used in previous EPA analyses as a means of facilitating comparisons with previous work. However, the use of alternate health endpoints and risk estimates would be entirely compatible with the ex-post/ex-ante analytical approach described above. In addition, we were only able to investigate a limited number of health outcomes for only one pollutant, given limitations in available information that could capture differential relative or absolute risks. In this case, the selected outcomes did contribute a significant portion of the benefits within the efficiency analysis, but in other cases, there may be a discrepancy between the drivers of monetized health benefits and the outcomes for which inequality can be most readily quantified. Finally, interpretation of the AI changes is complicated somewhat by the relatively small changes for either risk management scenario. However, this is consistent with prior work[12,13] and would be expected given a relatively small change in air pollution compared with ambient concentrations, as well as the fact that air pollution contributes a relatively small fraction to total baseline disease rates. The relative comparisons and directionality of the indicators remain interpretable and meaningful for policy analyses and decision making.

In spite of these and other limitations, our analysis provides multiple generalizable insights and lessons for future analyses. While our study focused on demonstrating the applicability of these methods with respect to $PM_{2.5}$ control strategy development, these methods are relevant to determining reductions for other pollutants of interest. In general, as described above, if the pollutant has a strong local gradient and affects a heterogeneous population of vulnerable and susceptible individuals, then these methods are applicable. We have also demonstrated that a risk-based approach that tends to target local sources in areas of high population density may not only provide greater health benefits, but may also reduce health inequality in multiple settings. This offers a "win-win" scenario for decisionmakers concerned with both efficiency and equality. Finally, we have shown that the combination of statistical and visualization methods applied to susceptible and vulnerable populations can provide valuable insight that allows air pollution control officials to make cost-effective decisions that maximize risk reduction and minimize health inequality.

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SUPPORTING INFORMATION

The following supporting information is available via a link in the online table of contents for this journal.

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Figure S.3: Annual mean $PM_{2.5}$ levels in 2020
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Figure S.6: Number of individuals below the poverty line
Figure S.7: White population in 2020 allocated to 1km domain (all ages)
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