Health Impact Assessment of Coal-Fired Boiler Retirement at the Martin Drake and Comanche Power Plants

TECHNICAL REPORT

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Executive Summary

Health impact assessment (HIA) is a suite of tools used to characterize potential health effects of policies, projects, or regulations. The objective of this HIA was to understand the impact of decommissioning units at two large coal-fired power plants on mortality and morbidity in the Southern Front Range region of Colorado. Based on Community Multiscale Air Quality (CMAQ) chemical transport models of fine particulate matter with an aerodynamic diameter less than 2.5 μm (PM_{2.5}) and ozone (O₃), we modeled five potential emissions reductions scenarios and estimated the potential health benefits of reduced exposures to PM_{2.5} and ozone for premature deaths, cardiovascular and respiratory hospitalizations, and other health outcomes for ZIP codes in the Southern Front Range region, including the cities of Denver, Colorado Springs, and Pueblo. Health Benefits Scenarios 1 and 2 estimated the health benefits of shutting down most units at the Comanche plant in Pueblo, CO (one newer unit remained operational) relative to a baseline scenario using emissions from 2011 (Scenario 1) or a counterfactual baseline scenario that accounted for sulfur dioxide emissions controls (scrubbers) installed at the Martin Drake plant in Colorado Springs in 2016 (Scenario 2). Health Benefits Scenario 3 estimated the benefits of shutting down the Martin Drake plant relative to the 2011 baseline. Health Benefits Scenario 4 estimated the health benefits of shutting down the Martin Drake power plant and shutting down all but one boiler at the Comanche power plant relative to a 2011 emissions baseline. Health Benefits Scenario 5 estimated the marginal health benefits of decommissioning these plants (with one remaining coal-fired boiler at Comanche) relative to a counterfactual baseline year that considered emissions controls installed at the Martin Drake facility in 2016. In addition to estimating the number of deaths, hospitalizations, and other health outcomes that would potentially be avoided by reducing emissions at these facilities, we also estimated the monetary impact using outcome valuations typically used in US EPA health benefits analyses and examined the environmental justice implications of reduced emissions and exposures across the Southern Front Range.

• For Health Benefits Scenario 1 (Comanche Units 3 and 4 were "zeroed out" and compared to a baseline where all other emissions were at 2011 levels), we estimated that reducing

population exposures to $PM_{2.5}$ would result in 1 (95% CI: 0 - 1) fewer premature death each year. Reductions in $PM_{2.5}$ and O_3 exposures would also result in fewer restricted activity days among adults [5 (95% CI: -3 – 95)] and fewer missed school days for children [27 (95% CI: -19- 582)]. Benefits of retiring the Comanche units were similar when emissions controls at Martin Drake are taken into account (Health Benefits Scenario 2).

- For Health Benefits Scenario 3 (emissions at Martin Drake were "zeroed out"), we estimated that reducing population exposures to PM_{2.5} and O₃ would result in 4 (95% CI: 2 5) and < 1 (95% CI: 0 1) fewer premature deaths each year, respectively. Reductions in PM_{2.5} and O₃ exposures would also result in fewer restricted activity days among adults [10 (95% CI: 0 74)] and fewer missed school days for children [4 (95% CI: 2-5)].
- For Health Benefits Scenario 4, we estimated that reducing population exposures to PM_{2.5} and O₃ would result in 4 (95% CI: 2 6) and < 1 (95% CI: 0 1) fewer premature deaths each year, respectively. Among the largest annual health benefits are avoided asthma symptom days among children [16 (95% CI: -1 141) due to PM_{2.5} and 13 (95% CI: -348 972) due to O₃] and minor restricted activity days among adults [69 (95% CI: 0 488) due to PM_{2.5} and 71 (95% CI: -31 750) due to O₃]. We also estimated that, for Health Benefits Scenario 1, children in the study area would miss 77 (95% CI: -77 1180) fewer days of school each year due to lower O₃ exposures.
- Annual health benefits were lower for Health Benefits Scenario 5 compared to Scenario 4 due to the smaller change in exposure concentration after accounting for the control technologies installed at Martin Drake in 2016. For Health Benefits Scenario 5, we estimated that reducing population exposures to PM_{2.5} and O₃ would result in 2 (95% CI: 1 3) and < 1 (95% CI: 0 1) fewer premature deaths each year, respectively. Other annual benefits under Health Benefits Scenario 2 included 2 (95% CI: -17 44) and 9 (-242 678) avoided asthma symptom days due to PM_{2.5} and O₃ exposures, respectively; 28 (95%CI: -2 188) and 48 (95%CI: -16 513) minor restricted activity days due to PM_{2.5} and O₃ exposures; and 53 (95% CI: -48 833) avoided school absences among children due to O₃ exposures.

Monetized health benefits when both plants were "zeroed out" ranged from \$4.2 million (95% CI: \$2.1 million - \$7.2 million) for Health Benefits Scenario 4 to \$1.7 million (95% CI: \$0.8 million - 3.2 million) for Health Benefits Scenario 5. Benefits tended to be smaller when only one plant was considered. In all of the analyses, the monetized impacts were driven by the value of avoided premature mortality.

In addition, we found that ZIP codes with lower median incomes tended to receive a greater share of the health benefits of decreasing exposures to $PM_{2.5}$ and O_3 resulting from power plant shutdowns. This finding suggests that reducing emissions at the power plants could potentially alleviate some environmental justice concerns in the area.

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Introduction

Health Effects of Air Pollution from Coal Fired Power Plant Emissions

The World Health Organization's Global Burden of Disease (GBD) project has determined that air pollution is currently the world's largest single environmental health risk, estimated to result in 7 million deaths annually (Forouzanfar et al. 2016). Specifically, fine particulate air pollution (particulate matter with an aerodynamic diameter less than 2.5 µm; PM_{2.5}) has been identified as a major risk factor for cardiovascular, pulmonary, and cerebrovascular (i.e. stroke) morbidity and mortality (Brook et al. 2010; Health Effects Institute 2010; Krewski et al. 2009; Pope et al. 2009). Similarly, short term and long term exposures to ozone (O₃) have also been associated with morbidity and mortality, particularly for respiratory diseases (Bell et al. 2005, 2004; Jerrett et al. 2009; US Environmental Protection Agency 2013). Although recent declines in urban air pollution levels across the United States are associated with longer life expectancy, (Correia et al. 2013) current levels of PM_{2.5} and ozone are still high enough to pose a public health risk (Fann et al. 2012b). A large proportion of the adverse health impacts in the United States attributed to ambient air pollution are the result of emissions from industrial facilities and electricity-generating stations that burn coal (Fann et al. 2009, 2012a). Other important sources of air pollution that contribute to adverse health effects include emissions from transportation, other industrial emissions, and residential energy use (Caiazzo et al. 2013; Lelieveld et al. 2015).

Fossil fuel combustion for electricity generation and other industrial or residential uses is a significant source of air pollution in the United States (US Environmental Protection Agency 2016c). Coal is a fossil fuel, and burning coal results in several air pollutant emissions, including oxides of nitrogen (NO_x), sulfur dioxide (SO₂), mercury and other heavy metals, and fly ash, a type of particulate matter (US Energy Information Administration 2018a). Sometimes this fly ash is emitted directly into the atmosphere, but most power plants have control technologies in place, including electrostatic precipitators or large filtration systems called "baghouses", to filter this pollutant before it can be released through the smokestack. However, gases such as

 SO_2 and NO_x are harder or more expensive to control. When SO_2 and NO_x are released into the atmosphere, they can oxidize in the atmosphere and combine with ammonia to form sulfate and nitrate particles; these particles are a type of fine particulate matter. Additionally, NO_2 , which is a byproduct of any type of combustion, in the presence of sunlight and volatile organic compounds can react to produce ground-level ozone pollution (US Energy Information Administration 2018a).

Coal remains an important fuel source for electricity generation and is responsible for 30.1% (1,208 billion kWh) of the electricity used in the United States in 2017 (US Energy Information Administration 2018c). For comparison, natural gas, wind, and photovoltaic solar were responsible for 31.7% (1,273 billion kWh), 6.3% (254 billion kWh), and 1.2% (53 billion kWh) of electricity generated, respectively (US Energy Information Administration 2018c). As of 2017, coal-fired power plants in the US had a total generating capacity of 260 gigawatts; overall capacity is projected to drop by 65 GW between 2018 and 2030, but remain constant between 2030 and 2050 (US Energy Information Administration 2018b).

Air Quality in the Front Range Region of Colorado

Air quality in Colorado is measured using a network of fixed-site monitors distributed across the state; in total, there are 53 sites measuring seven different pollutants and meteorological conditions (Colorado Department of Public Health & Environment 2017). Under the Clean Air Act (CAA) of 1970, the US Environmental Protection Agency (US EPA) established National Ambient Air Quality Standards (NAAQS) for six pollutants, including particulate matter (PM) and ozone. When concentrations measured at these monitors meet these standards, the area is considered in "attainment" (National Research Council 2004). Currently, air quality across the state meets the NAAQS for PM_{2.5}, PM₁₀, carbon monoxide, sulfur dioxide, lead, and nitrogen dioxide. However, the Denver-Boulder-Greeley-Ft. Collins-Loveland metropolitan area is considered in non-attainment of the 2008 O₃ NAAQS (US Environmental Protection Agency 2018). Long term monitoring data of the Front Range region of Colorado suggests that, unlike the east coast of the United States where air quality is improving over time, ozone is a worsening problem in the Front Range area of Colorado as well as the entire western United States (Cooper

et al. 2010).

The NAAQS and associated regulations have resulted in significant reductions in criteria pollutant concentrations nationwide, despite the concurrent growth in population, gross domestic product, energy consumption, and vehicle miles traveled since 1970 (US Environmental Protection Agency 2014). However, air quality problems still persist. When concentrations measured at monitors exceed the levels prescribed in the NAAQS, the area is considered in nonattainment of the standard, which triggers several regulatory actions. Included in this regulatory process is the development of a State Implementation Plan (SIP), which outlines the state's plan to reduce ambient concentrations and meet the NAAQS (National Research Council [NRC] 2004). SIPs can include a number of different types of source controls, e.g., installing scrubbers on power plant emissions stacks to remove pollutants from the waste stream, or restrictions on certain activities such as painting or operating gas powered lawn mowers (National Research Council [NRC] 2004). For example, the Colorado SIP for attaining the ozone standard outline measures to reduce emissions of ozone precursors (volatile organic compounds and NO_x), including vehicle inspection and maintenance programs and additional regulations for oil and gas development (Colorado Air Quality Control Commission 2016). In addition to helping reduce ozone concentrations, reductions in NO_x resulting from implementation of the SIP will also reduce secondary PM_{2.5} concentrations in the region, leading to potentially greater health benefits.

Even though air quality in the United States has improved substantially since 1970, current AQM programs may not be fully protective of public health for several reasons. Importantly, the CAA Amendments of 1990—enacted to strengthen the CAA of 1970—require NAAQS to be protective of public health, even for sensitive subpopulations, but epidemiological research has not yet identified "safe" levels below which there are no adverse health effects (Bell et al. 2006; Cesaroni et al. 2013; Daniels et al. 2004; Schwartz et al. 2002). Any air quality management actions that lower air pollutant concentrations could potentially lead to public health benefits (Pope et al. 2015). This means that, even in Colorado where particulate matter concentrations meet the NAAQS, reducing concentrations could have a positive public health impact. In addition to the lack of known "safe" exposures, the AQS monitoring network is not

designed to fully capture variability in pollutant levels within an urban area (Hubbell 2012; Levy and Hanna 2011; Matte et al. 2013), meaning an area within a city might be designated as in attainment of the air quality standards even though some residents living near sources of pollution, e.g., large coal-fired power plants or major highways, experience exposures that exceed the NAAQS concentrations (Isakov et al. 2009).

Environment Justice and Air Pollution

In the United States, poor and poorly educated people die at higher rates compared to those of higher incomes and better education (Pappas et al. 1993), and recent evidence indicates that this disparity continues to grow (Masters et al. 2012). Exposure to environmental pollutants, such as lead, industrial waste, and ambient air pollution is unequally distributed by class and race (Brown 1995) and contributes to higher rates of illness and death among individuals and communities of lower socioeconomic status (SES) (Adler and Newman 2002). The movement to address disproportionate burden of hazardous environmental exposures in low SES communities is referred to as environmental justice; the unfair burden among these communities is known as environmental injustice (Brulle and Pellow 2006). EPA defines environmental justice as "the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, policies" (US Environmental Protection Agency 2016a). US EPA also goes on to define "fair treatment," stating:

Fair treatment is the principle that no group of people, including a racial, ethnic or a socioeconomic group, should bear a disproportionate share of the negative environmental consequences from industrial, municipal and commercial operations or the execution of federal, state, local and tribal programs and policies. In implementing its programs, EPA has expanded the concept of fair treatment to include not only consideration of how burdens are distributed across all populations, but the distribution of benefits as well (US Environmental Protection Agency 2016a).

This concept of fair treatment extending to the benefits of environmental policies is important for air quality management actions which have the potential to result in benefits that are not

equally distributed across an area.

There have been numerous studies that link increased exposure to ambient air pollution with residence in low SES communities (Jerrett et al. 2001; Miranda et al. 2011; Morello-Frosch et al. 2011; Pearce et al. 2006), though this relationship has not been found consistently (New York City Department of Health and Mental Hygiene 2013; Vrijheid et al. 2012). In addition to higher levels of pollution in low SES areas, low SES communities often have unequal access to resources to help mitigate the negative effects of comparatively higher pollution exposures (Schulz and Northridge 2004). However, through policy and regulatory mechanisms, ambient environmental pollution is considered a modifiable risk factor for disease. Environmental policies have resulted in decreased population exposure to ambient air pollution (Chan et al. 2012; National Research Council 2004; Popp 2001), which in turn, has been linked to improved population health outcomes (Gauderman et al. 2015; Laden et al. 2006). However, questions remain regarding the relative benefit of these policies to low versus high SES groups (Cesaroni et al. 2013; Levy et al. 2002).

Health Impact Assessment

Health impact assessment (HIA) is a decision-making methodology to systematically incorporate public health research into public policy with the goal of improving population health (Cole et al. 2005; National Research Council 2011). HIA is considered to be a suite of tools that provides decision makers with recommendations to promote positive health impacts and to mitigate adverse health impact of proposed policies or regulations (Bhatia et al. 2014). Activities involved in HIA may range from "broad," which includes holistic, sociological, and qualitative approaches, to "tight" which focus on limited scope questions that may have a more quantitative approach (Kemm 2000). One of the primary functions of HIA, particularly related to questions regarding environmental policy, is to provide health outcomes data that may be used in cost-benefit analyses. For example, epidemiologic studies have demonstrated consistently that traffic-related air pollution is related to excess illness and mortality (HEI Panel on the Health Effects of Traffic-Related Air Pollution 2009). Quantitative HIA incorporates epidemiologic evidence on the relation between exposures and disease and applies this exposure-response information (known as a concentration-response function) to background

disease incidence (i.e. cardiovascular death rate in study population) to derive population-level estimates of a health outcome that may attributable to the exposure (Bhatia and Seto 2011). These estimates can be used in scenarios to understand the expected health outcome given a proposed or hypothetical change in an environmental exposure.

Though HIA have been used in an array of public health and environmental policy proposals (Dannenberg 2016), there is growing use of the method for understanding the societal impacts of ambient air quality. In Europe, Kunzli *et al.* have estimated that outdoor air pollution causes 6% of total deaths, with half related to traffic-related air pollution, 25,000 new cases of chronic bronchitis in adults, and more than half a million asthma attacks (Künzli et al. 2000). Fann *et al.* estimated that exposure to ambient PM_{2.5} has resulted in 130,000 excess deaths in the US, and 1.1 million years of life lost among the population age 65 and older (Fann et al. 2012b). Further, Levy *et al.* have demonstrated that policies to reduce point source emissions from older power plants would provide greater benefits for susceptible subpopulations, including African Americans, individuals with less than a high school education, and people with diabetes (Levy et al. 2002). Hubbell *et al.* (2009) provide a useful schematic for the analytical framework of quantitative HIA (Figure 1).

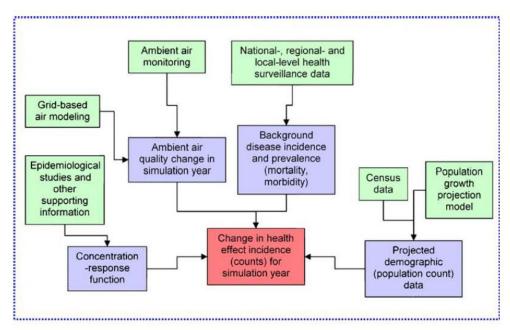


Figure 1: Schematic for conduct of analytical health impact assessments. Source: Hubbell et al., 2009

As these excess diseases and their related costs (referred to as externalities in the

economics literature) are not borne by the polluters (i.e. motorists, point sources), they are passed on to society, and often measured by the value of lost work days, restricted activities due to increased symptoms, health care events, or premature mortality (Delucchi 2003; Leigh and Geraghty 2008; Parry et al. 2007). For example, Levy et al. estimated that improved emission controls using the best available technology in five older power plants in the Washington, DC area would result in 240 fewer premature deaths, 60 fewer cardiovascular hospital admissions, and 160 fewer emergency departments for pediatric asthma per year for the underlying population (Levy et al. 2002). US EPA estimated that nationally, the implementation of the Clean Air Interstate Rule (2004) and the Nonroad Diesel Rule (2005) combined to result in 30,000 fewer premature deaths from air pollution annually (Hubbell et al. 2009). Based on economic theory, a value can be assigned to these excess health-related costs that can be used as a gauge to assess the potential costs (or savings) from a public project. For example, the Global Burden of Disease uses disability adjusted life years (DALYs) to account for excess morbidity and premature mortality (Murray and Lopez 1997), which may be assigned a dollar value to implement in benefit cost analyses or similar public policy evaluations (Morrow and Bryant 1995).

Quantitative HIA may be implemented in tandem with qualitative HIA approaches, in which comprehensive policy solutions are developed in partnership with key stakeholders including local government agencies, citizen groups, and community organizations (Cole et al. 2005). For example, regarding unconventional development of natural gas resources, McKenzie et al. implemented quantitative approaches to estimate cumulative cancer risk from proximity to well pads (McKenzie et al. 2012). In a companion analysis, Witter et al. conducted key stakeholder interviews with relevant community parties, including residents, the site operator, the surface rights owner, the industry association, the state health department, and the county environmental health manager to understand the potential positive and negative outcomes to inform decision making processes on unconventional natural gas drilling (Witter et al. 2013). In the best-case scenarios, these health outcomes are used to evaluate potential policy decisions. In Europe, a series of HIA on the health effects of ambient air pollution were used to estimate the public health impacts and associated costs under current levels of air pollution (O'Connell

and Hurley 2009). A suite of policy options to reduce ambient air pollution were evaluated, with benefits (included reductions in illness and death) were compared with the costs of several pollution reduction strategies (Holland et al. 2005). These estimates then informed the European Commission's new policy directives for control of ambient PM_{2.5} (O'Connell and Hurley 2009).

In summary, quantitative HIA builds upon existing epidemiologic evidence to determine population-level health effects of potential policy scenarios that are projected mitigate (or increase) environmental exposures. This method is increasingly viewed as the bridge between epidemiologic research and public policy assessment for environmental exposures.

HIA Study Considerations

In addition to the lack of universal definition and protocol for the conduct of health impact assessments, there are several other methodological considerations for the conduct of quantitative HIA:

Choice of concentration-response function. The concentration-response functions implemented in HIA are obtained from peer-reviewed, epidemiologic studies and applied to population-level data on a relevant health endpoint for a given study area. Important considerations for selection of concentration-response functions are (1) the quality of the initial epidemiologic study; and (2) the relevance and availability of the concentrationresponse function to the health impact measured (Hubbell et al. 2009). To conduct HIA for cardiovascular and pulmonary endpoints, there is a broad set of literature that may provide information on concentration-response functions for emergency department visits, hospitalizations, and mortality (Bell et al. 2006, 2009; Dominici et al. 2003; Glad et al. 2012; Jerrett et al. 2009; Pope et al. 2009). However, despite the increased number of health outcomes associated with PM_{2.5} and ozone exposure (US Environmental Protection Agency 2009, 2013), including premature birth and low birth weight (Wilhelm et al. 2012), Type II diabetes (Eze et al. 2015; Park and Wang 2014), cancer (Pope et al. 2002; Turner et al. 2014), and cognitive effects (Zanobetti et al. 2014), there is not yet a critical mass of evidence that can provide a comprehensive suite of concentration-response functions for all relevant health endpoints for other ambient air pollutants of concern. As a result, HIA may

- underestimate the total impact of planned mitigation strategies.
- Geographical Scale. Though there is an extensive history of performing national-scale HIA in the United States, particularly by the US EPA (Hubbell et al. 2009; Rhodus et al. 2013), there is a growing interest in implementing HIA at the local-scale. However, given that many well-established concentration response functions are derived from national-scale studies (Pope et al. 2002), the use of these functions for local-scale analysis could result in large errors for any given location given a difference in pollution sources, underlying disease rates, and demographics for any specific area (Hubbell et al. 2009).
- Exposure Assignment. One major factor that complicates the implementation of local-scale analyses is the pollution exposure assignment to a population in a geographic unit of analysis, such as a census tract. In the majority of national level models, pollution levels are determined by AQS monitors located within a metropolitan area (Samet et al. 2000). These studies tend to focus on between-city differences in health outcomes, rather than between-city differences. However, fixed-site monitors do not often reflect the spatial variability of pollutants over a small scale (Levy and Hanna 2011; Matte et al. 2013). For local-scale models, the level of resolution of a spatial model (often in the meters range) allows for a more spatially refined exposures but the unit of geographical analysis (e.g. census tract) contains varying levels of exposure and unequally distributed population, due to presence of land use areas such as parks, natural features, or non-residential zoning areas (e.g. commercial or industrial areas.) Currently, there are no established criteria or best practices for assigning pollution exposures to study areas in local-scale HIA.
- Choice of outcome for analysis. For national-scale HIAs that investigated the impacts of PM_{2.5} and ozone pollution, the EPA included a wide array of health endpoints for analysis, including mortality, chronic bronchitis, non-fatal heart attacks, hospital admissions, asthma emergency department visits, minor restricted activity days, asthma attacks, work loss days, worker productivity and school absence rates (US Environmental Protection Agency 2012b, 2015c, 2015b). As ambient air pollution exposure is associated with a broad array of health endpoints, inclusion of a broad suite of outcomes, including indirect effects such as missed school days and decreased worker productivity, ostensibly provide a more comprehensive

understanding of societal costs and potential benefits of air pollution mitigation strategies. However, the choice of outcome depends largely on the availability and quality of outcome data for the study area, as well as consistently reported concentration-response functions obtained from well-conducted epidemiological studies.

• Validity. High-quality epidemiologic research is conducted to maximize internal validity, or the ability to infer a relation between an exposure and an outcome in the population studied, with a secondary emphasis on external validity or generalizability, which indicate how those results can be translated to other populations (e.g. different demographics, exposure levels, geographic areas.) The assumptions that underlie the application of concentration-response functions from epidemiologic studies applied to HIA, particularly those of a local-scale, require a set of operating assumptions that may compound the uncertainty in effect estimates (Mesa-Frias et al. 2013, 2014). Though the information derived from an HIA is critical to understand the potential effects of a given pollution-control scenario, the evidence is not considered to have the same degree of scientific merit as a carefully conducted, scale relevant epidemiologic study (Bhatia and Seto 2011).

Health Impact Assessment of Southern Front Range Coal-fired Power Plants

Of environmental and public health interest in the southern front range of Colorado are two large coal-fired power plants: Comanche Generating Station, a 1,410 MW facility located in Pueblo, CO owned by Xcel Energy, and Martin Drake Power Station, a 185 MW facility located in Colorado Springs, CO owned by Colorado Springs Utility. Comanche Generating Station, the largest generating station in the state of Colorado, operates three units, each with a capacity between 325 and 750 MW; the facility has operated since the 1970's and burns low-sulfur coal as its primary fuel source (Xcel Energy 2017a). On June 6, 2018, Xcel Energy released its updated 2016 Electric Resource Plan 120-Day Report outlining a proposal to shutter Comanche Units 1 and 2 and replace this generation source with renewable energy from wind and solar sources (Xcel Energy 2017b). Martin Drake Power Station currently operates two units, and in 2016 the facility installed flue gas desulfurization scrubbers to dramatically reduce sulfur dioxide emissions from the facility (Colorado Springs Utility 2018; Stanton Anleu 2016). In 2015, the Utilities Board in Colorado Springs voted to decommission the plant by 2035; the process of decommissioning

the facility began in 2015 with the shutdown of Unit 5, the facilities smallest and oldest boiler (Colorado Springs Utility 2018).

Given the size of these units, the relatively large population living near these facilities, and the potential for significant health benefits, we conducted a health impact assessment to quantify the likely health benefits of shutting down these two generation stations. Our health impact assessment had two goals:

- 1. Quantify the health benefits (as avoided deaths, hospitalizations, and other health impacts) due to reduced air pollution exposures that would result from decommissioning these two southern front range power plants
- 2. Assess the economic and environmental justice implications for reducing exposures and adverse health effects in the study area

Completion of this health impact assessment required the use of data from multiple sources, including emissions inventories, health outcome data, and data on the populations living near the power plants. We also had to consider how plant operations have changed since the most recent reliable data became available. Therefore, we used the following CMAQ model runs in our analysis:

- Model Run 1: The baseline year of 2011, which was selected due to the availability of necessary data, including a validated emissions inventory and appropriate health outcome data;
- 2. Model Run 2: A counterfactual baseline year, which keeps all emissions inventory data the same as the baseline year of 2011, but accounts for the scrubbers installed at Martin Drake Units 6 and 7 in 2016 and the shutdown of Martin Drake Unit 5 in 2015. This scenario answers the question: "what would ambient concentrations have been in 2011 had the Martin Drake facility installed scrubbers in 2011?";
- 3. Model Run 3: A counterfactual "shutdown year" in which emissions at Comanche Units 4 and 5 are set to zero (i.e., the units are decommissioned), emissions at Comanche Unit 3 remain at 2011 levels, and emissions at the Martin Drake Power station remain at the

- 2011 baseline level. This scenario answers the question: "what would ambient concentrations have been in 2011 if Units 4 and 5 at the Comanche plant were decommissioned?";
- 4. Model Run 4: A counterfactual "shutdown year" in which emissions at Comanche Units 4 and 5 to zero (i.e., the units are decommissioned), emissions at Comanche Unit 3 remain at 2011 levels, and emissions at the Martin Drake Power station are reduced to the counterfactual baseline year level (as in Exposure Scenario 2) in order to account for scrubbers installed in 2016. This scenario answers the question: "what would ambient concentrations have been in 2011 if Units 4 and 5 at the Comanche plant were decommissioned and the Martin Drake facility had installed scrubbers in 2011?";
- 5. Model Run 5: A counterfactual "shutdown year" in which emissions at the Martin Drake Power Station are set to zero (i.e., the facility is completely decommissioned) and those from two of the older units at the Comanche Power Station are set to zero (Unit 3 at the Comanche Power Station, which was installed in 2010, was kept operational). This scenario answers the question: "what would ambient concentrations have been in 2011 had most of the units at these facilities (except Unit 3 at Comanche) been shut down in 2011?"

The use of counterfactual scenarios is needed in this health impact assessment as the power plants are scheduled to be decommissioned in the future, when projected health rates and population data are not reliable or not available at the spatial resolution required for the analysis. Still, the use of counterfactuals based on the year 2011 should provide a reasonable estimate of the magnitude of health benefits that could be expected once the power plants identified in the analysis are decommissioned.

Methods

The following section details the methods used in this health impact assessment. First, we describe the air pollutant modeling and the quantitative HIA model used to estimate attributable health impacts. Then, we include a description of the inequality assessment used to examine the environmental justice implications of retiring two coal-fired power plants in the region. Finally,

we use cost and inequality metrics to assess the potential economic and environmental justice implications of retiring these facilities.

Study Area and Spatial Unit of Analysis

The study area (Figure 2) encompassed more than 56,000 sq. kilometers on Colorado's Southern Front Range, and included the cities of Denver, Colorado Springs, and Pueblo. In total, approximately 3.8 million people live in the study area. The analysis used ZIP code tabulation areas (ZCTA) as the smallest spatial unit of analysis. ZCTAs are spatial units designed by the US Census Bureau to closely align with US Postal Service ZIP codes, though there may be some differences in unit boundaries from year to year (Grubesic and Matisziw 2006). ZCTAs were selected as the spatial unit of analysis as 1) they were small enough to partially capture the spatial variability in exposures across the study area and 2) they contained populations large enough to calculate reliable hospitalization and mortality rates used in the health impact functions (please see below).

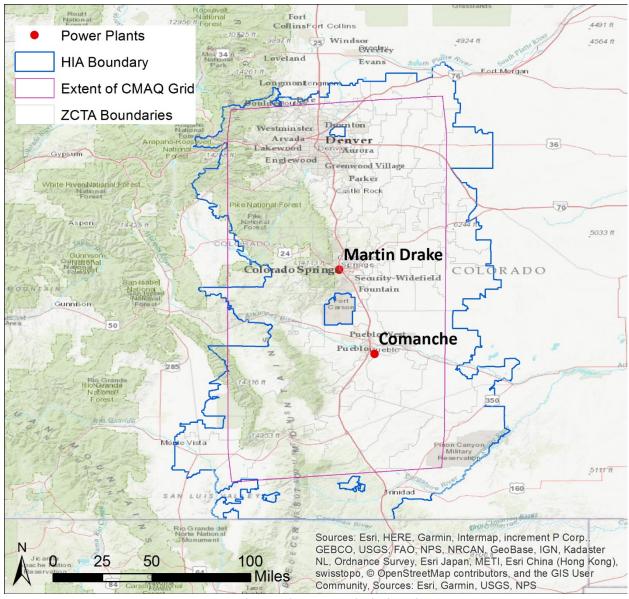


Figure 2. The study area included in the HIA

Ambient Air Pollutant Concentration Modeling

We used the Community Multiscale Air Quality (CMAQ) model (version 5.0.2) to simulate changes in the air pollutant concentrations over the study domain. CMAQ is an air-chemistry transport model that simulates the emissions, transport, chemistry, and surface deposition of air pollutants in the lower troposphere. The CMAQ model was developed and is maintained by the US Environmental Protection Agency and is the model of choice for regulatory and policy needs. Details about this version of the model can be found in Gantt et al. (2015). Briefly, the CMAQ model was used to simulate air pollutant concentrations for a representative summer (June 29).

to August 18, 2011) and a winter period (January 2 to February 21, 2011). We only modeled a representative summer and winter period because the CMAQ model is computationally intensive and we needed to perform this work in a reasonable timeframe. The year 2011 was selected since this is the most recent version for which validated pollutant emissions were available through the National Emissions Inventory (NEI; US Environmental Protection Agency 2012a). Model results for the first sixteen days were ignored to account for model spin up and the HIA analysis was performed with model results from 30 summer and winter days. Results generated for each of these months using the health impact assessment models (described next) were scaled up to represent the full year.

Model simulations were run at three different resolutions: 36 km, 12 km, and 4 km. The 36 km domain approximately covered the continental US, the 12 km domain covered parts of the Mountain West region, and the 4 km version roughly covered the four state region of Colorado, Utah, New Mexico, and Arizona. The 36 km and 12 km model results are used to inform the initial and boundary conditions for the 4 km version (299 rows x 281 columns x 25 vertical layers). Atmospheric chemistry was simulated using the Carbon Bond 2005 chemical mechanism and particle chemistry and thermodynamics was simulated using the aerosols module 5 (AE5) (Sarwar et al. 2012). As mentioned earlier, anthropogenic emissions were based on the most recent NEI product while biogenic emissions were based on the Biogenic Emission Inventory (BEIS) version 3.14 model (Carlton and Baker 2011). Meteorological inputs were generated using version 3.1 of the Weather Research and Forecasting (WRF) model (Skamarock et al. 2008).

Modeled concentrations of only O_3 and $PM_{2.5}$ were used for the HIA described below. These two pollutants were selected because they have been shown to account for the majority of adverse health outcomes and for which concentration-response functions are readily available (Fann et al. 2012b; US Environmental Protection Agency 2012b, 2015c).

Finally, the CMAQ modeling system that includes the chemical transport model and its relevant inputs for the year 2011 (e.g., meteorology, emissions, land use types) were requested from the Intermountain West Data Warehouse (Intermountain West Data Warehouse 2018).

Health Impact Assessment Model

A quantitative health impact assessment model was used to predict the number of cases of mortality and morbidity attributable to ambient air pollutant exposures in the study area. Health impact modeling requires four input variables: baseline incidence rates for health outcomes of interest, a concentration response coefficient, an estimate of exposure, and the number of persons exposed. The model implemented in this report was based upon the Environmental Benefits and Mapping and Analysis Program (BenMAP) (US Environmental Protection Agency 2016b). BenMAP is a software tool developed by US EPA to facilitate quantitative health impact assessments of ambient air pollution.

The health impact function (Eq. 1) is adapted from the expression for attributable risk, and is defined as:

$$\Delta Y = y_0 \times (1 - e^{-\beta \Delta x}) \times P \times D$$
 (Eq. 1)

where ΔY is the change in the number of attributable impacts during the study period (cases), y_0 is the baseline health outcome incidence rate (cases per person per day), β is the concentration-response coefficient (1/ppb or 1/µg m⁻³), Δx is the estimated change in ambient concentration (ppb or µg m⁻³), P is the number of people exposed, and D is the number of days in the study period (US Environmental Protection Agency 2015a).

We modeled two health benefit scenarios in our health impact assessment (Table 1). Health Benefit Scenarios 1 and 2 assessed the health benefits of reducing exposures at the Comanche plant (keeping the newest unit operational) relative to the 2011 baseline case (Scenario 1) and the 2016 counterfactual baseline year which accounted for controls at Martin Drake (Scenario 2). Health Benefits Scenario 3 estimated the benefits of eliminating emissions at Martin Drake relative to the 2011 baseline case. Health Benefit Scenarios 4 and 5 assessed the health benefits of shutting down the Martin Drake facility and the two units at Comanche at the same time. In Scenario 4, health benefits were estimated for the change in exposure relative to the 2011 baseline and in Health Benefits Scenario 5, health benefits were estimated for the change in exposure relative to the 2016 counterfactual baseline. It is important to note that

emissions at all other facilities included in the emissions inventory remain the same in both scenarios.

Table 1. Summary of the Health Benefits Scenarios used in this HIA

Scenario Name	Baseline Exposure Scenario	Post-Shutdown Exposures
Health Benefits Scenario 1	Model Run 1: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area.	Model Run 3: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area except Comanche Units 4 and 5, which were "zeroed out."
Health Benefits Scenario 2	Model Run 2: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area except Martin Drake. For this facility, we adjusted the 2011 NEI emissions to account for scrubbers installed in 2016.	Model Run 3: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area except Comanche Units 4 and 5, which were "zeroed out."
Health Benefits Scenario 3	Model Run 1: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area.	Model Run 4: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area except Martin Drake, which was "zeroed out."
Health Benefits Scenario 4	Model Run 1: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area.	Model Run 5: CMAQ was run for representative summer and winter periods using the 2011 NEI, with emissions at Martin Drake and the two oldest units at Comanche "zeroed out."
Health Benefits Scenario 5	Model Run 2: CMAQ was run for representative summer and winter periods using the 2011 NEI for emissions at all sources in the area except Martin Drake. For this facility, w adjusted the 2011 NEI emissions to account for scrubbers installed in 2016.	Model Run 5: CMAQ was run for representative summer and winter periods using the 2011 NEI, with emissions at Martin Drake and the two oldest units at Comanche "zeroed out."

We accounted for uncertainty in each of the health impact model inputs using a Monte Carlo approach. For each input, including the change in exposure, health rates, concentration-response coefficient, and population, we generate a distribution of possible values (using the mean estimate and the standard error of the mean), and then randomly draw from this distribution 1000 times. The result is a distribution of 1,000 possible health impact estimates. We report the median of this distribution as the "most likely" estimate of the health benefits and use the 2.5th and 97.5th percentiles of the distribution to generate a confidence interval around that "most likely" estimate.

Additional details on the inputs for Eq. 1 are detailed in the following sections.

Health Outcome Incidence Rates

Mortality data were obtained from the Colorado Department of Public Health and Environment. Records from the years 2010-2014 were available, and each record contained information on year of death, primary cause of death identified on the death certificate as defined by the International Classification of Disease 10th Revision (ICD-10), the ZIP code of residence at death and age at death. For our analysis, we assumed ZIP codes and ZCTA were equivalent. Mortality data were aggregated to the ZCTA level and used to calculate crude five-year average morality rates for the population 30 years of age or older for two sets of causes based on ICD-10 code: all-cause mortality (includes all ICD-10 codes) and non-accidental mortality (includes ICD-10 codes A00 – R99). The designation of "non-accidental" mortality was based on an environmental epidemiology study of the effects of ozone exposure on mortality (Bell et al. 2004). ZCTA populations were based on the American Community Survey (ACS) 5-Year Estimates at the ZCTA level (US Census Bureau 2014).

Hospitalization data were obtained from the Colorado Hospital Association. Records from the years 2010-2014 were available, and each record contained information on year of the hospitalization, primary diagnosis defined by ICD-9 codes, and the ZIP code of residence and age at the time of the hospitalization. Again, we assumed ZIP codes and ZCTA were equivalent. These hospitalization data were aggregated to the ZCTA level and used to calculate

crude 5-year average hospitalization rates for the population 65 years of age or older for two sets of causes based on ICD-9 codes: cardiovascular diseases (ICD-9 codes 390–459) and respiratory diseases (ICD-9 codes 460–519). Again, we used the ACS 5-Year estimates at the ZCTA level to calculate crude incidence rates for hospitalizations (US Census Bureau 2014).

There are several other health outcomes that are causally related to PM_{2.5} and ozone exposures for which area-specific incidence data are not available. These outcomes include: emergency department visits for asthma among children (less than 18 years of age), asthma symptom days among children (i.e., days with cough, wheeze, or shortness of breath), missed days of school among school-aged children (6 to 18 years), minor restricted activity days (adults ages 18 and older), and work loss days (for adults ages 18-64). We took baseline incidence rates for these outcomes from the BenMAP User Manual and assigned them to each of the ZCTA in the study area (US Environmental Protection Agency 2015a).

Eq. 1 requires the use of daily incidence rates to estimate the number of cases attributable to change in daily exposure concentrations. To estimate daily incidence rates, we assigned each ZCTA an annual rate (either the 5-year average annual rates calculated at the ZCTA level from the mortality and hospitalization data or the national-level rates from BenMAP) and then divide by 365 to obtain a daily rate.

Concentration-Response Functions

Concentration-response functions were based on existing environmental epidemiology studies of the health effects of exposure to PM_{2.5} and O₃. For this study, we used the same concentration-response functions for PM_{2.5} and O₃ that are available in BenMAP (US Environmental Protection Agency 2015a). BenMAP used US EPA criteria for study design, validity, and generalizability when choosing studies from the large body of air pollution epidemiology (US Environmental Protection Agency 2009, 2012b, 2013, 2015c). For premature mortality due to PM_{2.5} we used the estimate from the American Cancer Society study (Krewski et al. 2009); this concentration-response function is the most widely cited study in the HIA literature. For all other impacts, we used pooled estimates from multiple studies based on methods reported in the most recent Regulatory Impact Analysis for the PM_{2.5} NAAQS (US

Environmental Protection Agency 2012b). This pooling approach, which weighted each of the study-specific concentration-response coefficients by the precision (i.e., the standard error) of the coefficient, generated an "average" concentration-response coefficient for each pollutant-outcome pair. Pooling across several studies is useful when existing epidemiology studies may be small, conducted in areas outside of the HIA study area, or have limited generalizability (Hubbell et al. 2009). A list of the studies used in this HIA along with the pooled concentration-response coefficients is available in the Appendix A.

Exposure Assessment

Exposures were assigned to each ZCTA using a geostatistical technique called kriging. The exposure assessment methodology is illustrated in Figure 3. First, concentrations were predicted at known locations (4 km x 4 km) using the CMAQ model (see section above for details on this modeling tool) and then summarized to daily and monthly concentrations at each point. A subset of grid cells from the full grid was selected and used in the exposure assessment (the boundary for the points selected is shown in in Figure 2). Next, concentrations are estimated at a much more dense grid of points (1 km x 1 km) using a statistical model. Finally, a population-weighted average of all the estimated concentrations that fall within a ZCTA is generated for each day and the entire month. This population weighted average is assigned to every person living in the ZCTA.

For ZCTAs that fall along the edge of the CMAQ grid and are not completely covered by the grid, we assumed the population-weighted concentration estimated for the portion of the ZCTA that overlaps with the grid is applicable to the entire ZCTA. This is a reasonable approach in this application because most of the ZCTAs along the edge of the study grid have low population densities and pollutant concentrations (see Figure 3).

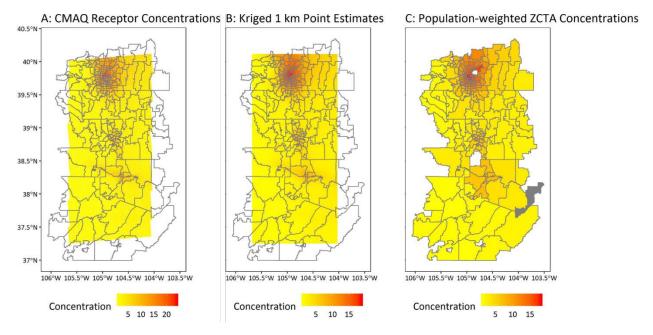


Figure 3: Example illustration of the exposure assessment methodology used in the health impact assessment

Exposed Populations

The number of exposed people in each ZCTA was taken from the 2010-2014 ACS 5-year estimates (US Census Bureau 2014). We assumed the entire population was exposed in each scenario. Exposure were based on the residential ZCTA of each person in the study area, which is consistent with most of the epidemiology studies used to generate the concentration-response functions used in this study. Although this approach may potentially bias the health impact estimates (Tchepel and Dias 2011), individual level data on commuting patterns and other mobility behaviors were not available for this study population.

For each pollutant-outcome pair, we limited the exposed population to those in the relevant age groups; for example, we assessed premature mortality only for the population in each ZCTA that is 30 years of age or older, and asthma symptom days are only assessed for children ages 7 to 14. This was done to match the population age groups used in the original studies; concentration-response coefficients for one population age group may not be appropriate for other age groups. Stratification of the population was done using the age- and sex-specific data available from the ACS (US Census Bureau 2014).

Economic Analysis

A full cost-benefit analysis is beyond the scope of this assessment. However, it was feasible to monetize the health benefits of each shutdown scenario using values assigned to each outcome by US EPA (US Environmental Protection Agency 2010). For this assessment, we used the same monetized values used in the most recent Regulatory Impact Analysis for the ozone NAAQS (US Environmental Protection Agency 015c). The monetary value of a health outcome can be determined in a number of ways. For mortality, which is assigned a value of \$10 million per premature death is based on the concept of willingness-to-pay, which asks people how much they are willing to pay to reduce their risk of dying prematurely and adjusts that value based on their expected remaining life (US Environmental Protection Agency 2010). For hospitalizations and other health-care utilization outcomes (e.g., emergency department visits), the value assigned to each hospitalization avoided is based on the average cost of care. For outcomes such as work loss days, school loss days, or asthma exacerbation days, values are often based on the potential lost wages of the employee or adult who misses work to care for a sick child (US Environmental Protection Agency 2015c).

The monetized value of the health benefits (i.e., avoided adverse health impacts) of retiring the coal-fired power plants should be considered complementary to the number of health benefits estimated for the population. The data used to inform the economic analysis are used in national-scale assessments, and in many cases, the value of a health outcome depends on several factors, including income and healthcare costs.

Environmental Justice Analysis

Given the demographic and socioeconomic diversity of the study area, one of the study goals was to assess the environmental justice implications of retiring these coal fired power plants. As stated earlier, US EPA has defined environmental justice as the fair treatment of all groups under environmental regulations, and has extended this idea of fair treatment not only to the distribution of exposures but also to the distribution of health benefits (US Environmental Protection Agency 2016a). We examined the distribution of health benefits using two complementary approaches:

- 1. Changes in exposure (Δ ppb or Δ μ g m⁻³) and health benefit rates (benefits per 1,000 people) were mapped for each pollutant and health outcome, which helps to identify areas within the study area that benefit most under the shutdown scenarios
- 2. Differences in health benefits across demographic and socioeconomic groups were assessed using the Concentration Index curve. The concentration index first ranks ZCTAs by their degree of social advantage, and then compares the change in exposures by this social advantage (Harper et al. 2013; O'Donnell et al. 2008). Because we measured a positive outcome (decreases in adverse health impacts), we interpreted a concentration index curve above the 1:1 line of inequality to mean that benefits were higher among ZCTAs with lower social advantage (Maguire and Sheriff 2011); this would be a positive outcome from an environmental justice perspective, as lower social advantage groups typically bear disproportionately higher health burdens as a result of environmental pollutants. Here, we defined low social advantage as having a higher proportion of nonwhite residents (e.g., higher proportions of residents that identify as Hispanic/Latino or Black/African American) or a lower median income (Fann et al. 2011). It is important to note that these are "neighborhood" level characteristics of social disadvantage and may not reflect individual level characteristics. For example, it is possible for a "low disadvantage" person to live in a "high disadvantage" neighborhood.

These inequality metrics are used to examine whether shutting down these plants has a "equal" benefit to all residents, and if not, whether environmental justice communities (e.g., minority and low-income communities) may be left out of the health benefits. There are no established standards against which to compare inequality metrics. In general, a scenario which reduces inequality (i.e., reduces the Atkinson index or results in a Concentration Index curve that closely matches the 1:1 line of equality) is considered favorable.

Results

The following section summarizes the changes in exposure and subsequent health benefits for each of the health benefits scenarios outlined in this report. Here, we focused primarily on Health Benefits Scenarios 4 and 5, which focus on shutdowns at the two plants simultaneously. We also present results for Health Benefits Scenarios 1-3, which focus on one plant at a time. Additional information for these single plant scenarios is included in the Appendices.

Changes in Exposure due to Reductions in Power Plant Emissions

Exposures generally decreased when emissions at the power plants were reduced, though in cases some exposures to ozone were higher after plant emissions were removed from the model. (Table 2). Table 2 shows the change in exposure concentrations for Health Benefits Scenario 4, which reduced emissions at both power plants relative to the 2011 baseline; this scenario represented the "greatest reduction" case. (Comparable tables for the single plant shutdown health benefits scenarios are available in Appendix B. In general, exposure reductions for the single plant shutdown scenarios were much smaller than the scenarios where both plants were "zeroed out" in the CMAQ model run.) For PM_{2.5}, decreases in the seasonal and daily means were greatest for the summer months; on average, the seasonal mean PM_{2.5} concentration during the summer across all ZCTAs decreased 0.04 μ g/m³ (SD = 0.05 μ g/m³) when emissions at Martin Drake are eliminated and Comanche were reduced. The highest change across all ZCTAs was 0.36 μ g/m³. Reductions in winter PM_{2.5} may be lower than in the summer due to several other seasonal sources of PM_{2.5}, including woodstoves used for residential heating (Vedal et al. 2009).

Similarly for ozone, reductions in exposure at the ZCTA level are strongest in the summer. The average decrease in ozone across ZCTAs during the summer was 0.02 ppb, and the highest decrease in ozone was 0.13 ppb. Ozone showed substantial variability in 1-hour and 8-hour exposure reductions. On average, the daily 1-hr and 8-hour maximum concentrations were reduced 0.43 and 0.20 ppb, respectively, with maximum reductions of 5.17 and 2.51 ppb,

respectively. Increases in winter seasonal mean and daily mean ozone concentrations are likely the result of alterations in the overall NOx-O₃ relationship that occurs when combustion at the plants is reduced. NO emissions generated by the plant help to "scavenge" ground-level ozone (i.e., react with O₃ to produce O₂ and NO₂; Song et al. 2011); reduced combustion at the plant results in less NO in the atmosphere to scavenge ozone generated by other sources, e.g., vehicle emissions.

Table 2. Summary statistics for the change in population-weighted exposures to PM2.5 (μ g/m³) and ozone (ppb) across all ZCTAs in the study area for Health Benefits Scenario 4

Pollutant	Season	Metric	Mean (SD)	Min	Median	Max
$PM_{2.5}$	Winter	Seasonal mean	0.01 (0.02)	0.00	0.01	0.13
	Summer	Seasonal mean	0.04 (0.05)	0.00	0.03	0.36
	Winter	Daily mean	0.01 (0.03)	-0.04	0.00	0.40
	Summer	Daily mean	0.04 (0.07)	0.00	0.02	0.90
О3	Winter	Seasonal mean	-0.05 (0.08)	-0.41	-0.01	0.00
	Summer	Seasonal mean	0.02 (0.13)	-0.97	0.05	0.13
	Winter	Daily mean	-0.05 (0.13)	-2.13	0.00	0.07
	Summer	Daily mean	0.02 (0.21)	-3.03	0.02	0.82
	Winter	Daily 1 hour max	0.02 (0.07)	-0.32	0.00	0.72
	Summer	Daily 1 hour max	0.44 (0.57)	-0.89	0.23	5.17
	Winter	Daily 8 hour max	0.00 (0.03)	-0.56	0.00	0.35
	Summer	Daily 8 hour max	0.20 (0.26)	-0.49	0.10	2.51

Note: Change in exposures are calculated as the difference between Model Run 1 (2011 baseline case) and Model Run 4 (full shutdown of Martin Drake and shutdown of all units except Unit 3 at Comanche). Negative changes in exposure mean that exposures are higher under Model Run 4 (the shutdown scenario). Abbreviations: $PM_{2.5}$: particulate matter with an aerodynamic diameter less than 2.5 μ m; O_3 : ozone; SD: standard deviation

Changes in exposures occurred were greatest near the power plants. Figure 4 shows the change in summer PM_{2.5} (A) and ozone (B) concentrations at the ZCTA level for Health Benefits Scenario 4. (Comparable maps of the changes in exposure during the winter are included in Appendix C). The power plants are indicated with a red dot. For PM_{2.5}, concentrations near the power plants were reduced, with lower reductions farther from the plants; for O₃, concentrations near the plant increased slightly under the emissions reduction scenario (indicated in Figure 4B by negative changes in concentration) and decreased slightly elsewhere across the study area.

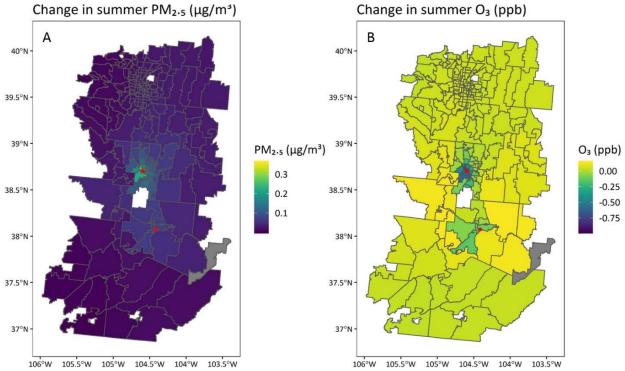


Figure 4. Changes in mean summer PM_{2.5} (A; μ g/m³) and O₃ (B; ppb) concentrations at the ZCTA level for Health Benefits Scenario 4

Reductions in exposure for Health Benefits Scenario 5, which used the counterfactual baseline scenario where 2011 emissions at Martin Drake were reduced to account for controls installed in 2016, tended to be smaller than for Health Benefits Scenario 4 (Table 3). As was the case for Health Benefits Scenario 1, changes in PM_{2.5} exposures resulting from reduced emissions were greater for the summer period than the winter period. The average reduction in summer and winter ZCTA-level PM_{2.5} (0.01 μ g/m³ and 0.008 μ g/m³, respectively) across all ZCTAs were 70% and 35% lower, respectively, than the average reductions for Health Benefits Scenario 4 (Table 2). For ozone, exposure concentrations increased for the winter period (mean difference = -0.03 ppb) and decreased for the summer period (mean difference = 0.03 ppb). (Maps showing the reductions in exposure concentrations for Health Benefits Scenario 5 are in Appendix C).

Table 3. Summary statistics for the change in population-weighted exposures to $PM_{2.5}$ (µg/m³) and ozone (ppb) across all ZCTAs in the study area for Health Benefits Scenario 5

Pollutant	Season	Metric	Mean (SD)	Min	Median	Max
$PM_{2.5}$	Winter	Seasonal mean	0.01 (0.01)	0.00	0.00	0.09
	Summer	Seasonal mean	0.01 (0.02)	0.00	0.01	0.14
	Winter	Daily mean	0.01 (0.02)	-0.02	0.00	0.30
	Summer	Daily mean	0.01 (0.02)	0.00	0.00	0.38
O ₃	Winter	Seasonal mean	-0.03 (0.05)	-0.17	-0.01	0.00
	Summer	Seasonal mean	0.03 (0.07)	-0.55	0.04	0.11
	Winter	Daily mean	-0.03 (0.09)	-1.13	0.00	0.06
	Summer	Daily mean	0.03 (0.12)	-1.47	0.02	0.66
	Winter	Daily 1 hour max	0.02 (0.05)	-0.17	0.00	0.60
	Summer	Daily 1 hour max	0.32 (0.46)	-0.28	0.16	5.33
	Winter	Daily 8 hour max	0.00 (0.02)	-0.30	0.00	0.23
	Summer	Daily 8 hour max	0.15 (0.20)	-0.10	0.08	1.76

Note: Change in exposures are calculated as the difference between Exposure Scenario 2 (counterfactual 2011 baseline case that accounts for control technologies installed at Martin Drake in 2016) and Exposure Scenario 3 (full shutdown of Martin Drake and shutdown of all units except Unit 3 at Comanche). Negative changes in exposure mean that exposures are higher under Exposure Scenario 3.

Abbreviations: $PM_{2.5}$: particulate matter with an aerodynamic diameter less than 2.5 μ m; O_3 : ozone; SD: standard deviation

Health Benefits due to Reductions in Power Plant Emissions

Health Benefits Scenarios 1-3, which estimated benefits for shutting down the power plants individually, resulted in modest health benefits for the populations (Table 4). For Health Benefits Scenario 1 (Comanche Units 3 and 4 were "zeroed out" and compared to a baseline where all other emissions were at 2011 levels), we estimated that reducing population exposures to $PM_{2.5}$ would result in 1 (95% CI: 0 - 1) fewer premature death each year (Table 4). Reductions in $PM_{2.5}$ and O_3 exposures would also result in fewer restricted activity days among adults [5 (95% CI: -3 – 95)] and fewer missed school days for children [27 (95% CI: -19- 582)]. Benefits of retiring the Comanche units were similar when emissions controls at Martin Drake are taken into account (Table 4; Health Benefits Scenario 2). For Health Benefits Scenario 3 (emissions at Martin Drake were "zeroed out"), we estimated that reducing population exposures to $PM_{2.5}$ and O_3 would result in 4 (95% CI: 2 - 5) and <1 (95% CI: 0 - 1) fewer premature deaths each year, respectively (Table 4). Reductions in $PM_{2.5}$ and O_3 exposures would also result in fewer restricted activity days among adults [10 (95% CI: 0 – 74)] and fewer missed school days for children [4 (95% CI: 2-5)]. It

is important to note that the benefits from Health Benefits Scenarios 1 and 3 are not additive due to the non-linear relationships between ozone and secondary PM_{2.5} precursors used in the CMAQ chemical transport models.

Table 4. Summary of annual health benefits (as number of avoided premature deaths and cases of morbidity) for each of the health benefits scenarios in which only one power plant was retired

	7/3		/	
		Benefits	Benefits	Benefits
		Scenario 1	Scenario 2	Scenario 3
Pollutant	Outcome	n (95% CI)	n (95% CI)	n (95% CI)
PM _{2.5}	All-cause mortality (adults)	1 (0 - 1)	1 (0 - 1)	4 (2 - 5)
	CVD hospitalization (adults 65+)	0 (0 - 1)	0 (0 - 1)	0 (0 - 2)
	Respiratory hospitalization (adults 65+)	0 (0 - 1)	0 (0 - 1)	0 (0 - 2)
	ED visit for asthma (children)	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
	Asthma symptom day (children)	0 (-7 - 19)	0 (-7 - 20)	1 (0 - 13)
	Lower respiratory infection (children)	0 (0 - 3)	0 (0 - 3)	58 (-1 - 450)
	Minor restricted activity day (adults)	5 (-3 - 95)	5 (-3 - 97)	10 (0 - 74)
	Work loss day (adults)	1 (-1 - 16)	1 (0 - 16)	
				0 (0 - 1)
O ₃	Non-accidental mortality (adults)	0 (0 - 0)	0 (0 - 0)	0 (-13 - 20)
	Respiratory hospitalization (adults 65+)	1 (-7 - 15)	1 (-7 - 15)	0 (0 - 0)
	ED visit for asthma (children)	0 (0 - 0)	0 (0 - 0)	6 (-212 - 615)
	Asthma symptom day (children)	4 (-172 - 495)	5 (-171 - 493)	40 (-23 - 567)
	Minor restricted activity day (adults)	24 (-7 - 376)	25 (-7 - 383)	41 (-52 - 839)
	School absence day (children)	27 (-19 - 582)	28 (-20 - 591)	4 (2 - 5)
	School absence day (children)	27 (-19 - 582)	28 (-20 - 591)	

Note: 95% Confidence Interval is based on the 2.5th and 97.5th percentile of the distribution of benefits generated by the Monet Carlo analysis. Outcomes listed in bold are those for which area-specific baseline incidence rates are used. All other outcomes use baseline incidence rates taken from BenMAP.

Abbreviations: ED: emergency department; $PM_{2.5}$: particulate matter with an aerodynamic diameter less than 2.5 μ m; O_3 : ozone

Benefits for retiring both plants simultaneously were greater than for retiring only one plant (Table 5). For Health Benefits Scenario 4 (Units 3 and 4 at Comanche and all units at Martin Drake were zeroed out and compared to the 2011 baseline), we estimated that reducing population exposures to $PM_{2.5}$ and O_3 would result in 4 (95% CI: 2 - 6) and < 1 (95% CI: 0 - 1) fewer premature deaths each year, respectively (Table 5). Among the largest annual health benefits were avoided asthma symptom days among children [16 (95% CI: -1 – 141) due to $PM_{2.5}$ and 13 (95% CI: -348 - 972) due to O_3] and minor restricted activity days among adults [69 (95% CI: 0 - 488) due to $PM_{2.5}$ and 71 (95% CI: -31 - 750) due to O_3]. We also estimated that, for Health

¹ Health Benefits Scenario 1 estimated the health benefits of retiring the two oldest units at Comanche relative to a 2011 baseline emissions case

² Health Benefits Scenario 2 estimated the health benefits of retiring the two oldest units at Comanche relative to a counterfactual baseline emissions case which accounts for control technologies installed at Martin Drake in 2016

³ Health Benefits Scenario 3 estimated the health benefits of retiring all units at Martin Drake relative to a 2011 baseline emissions case

Benefits Scenario 4, children in the study area would miss 77 (95% CI: -77 - 1180) fewer days of school each year due to lower O₃ exposures.

Health benefits were lower for Health Benefits Scenario 2 due to the smaller change in exposure concentration after accounting for the control technologies installed at Martin Drake in 2016 (Table 5). For Health Benefits Scenario 2, we estimated that reducing population exposures to $PM_{2.5}$ and O_3 would result in 2 (95% CI: 1 - 3) and < 1 (95% CI: 0 - 1) fewer premature deaths each year, respectively. Other annual benefits under Health Benefits Scenario 2 included 2 (95% CI: -17 - 44) and 9 (-242 - 678) avoided asthma symptom days for children due to $PM_{2.5}$ and O_3 exposures, respectively; 28 (95%CI: -2 - 188) and 48 (95%CI: -16 - 513) minor restricted activity days for adults due to $PM_{2.5}$ and O_3 exposures; and 53 (95% CI: -48 - 833) avoided school absences among children due to O_3 exposures.

Table 5. Summary of annual health benefits (as number of avoided premature deaths and cases of morbidity) for each of the health benefits scenarios in which units at both power plants were retired

		Benefits Scenario 4 ¹	Benefits Scenario 5 ²
Pollutant	Outcome	n (95% CI)	n (95% CI)
PM _{2.5}	All-cause mortality (adults)	4 (2 - 6)	2 (1 - 3)
	CVD hospitalization (adults 65+)	0 (0 - 3)	0 (0 - 1)
	Respiratory hospitalization (adults 65+)	0 (0 - 3)	0 (0 - 1)
	ED visit for asthma (children)	0 (0 - 0)	0 (0 - 0)
	Asthma symptom day (children)	16 (-1 - 141)	2 (-17 - 44)
	Lower respiratory infection (children)	2 (0 - 14)	1 (0 - 5)
	Minor restricted activity day (adults)	69 (0 - 488)	28 (-2 - 188)
	Work loss day (adults)	11 (0 - 81)	5 (0 - 31)
O ₃	Non-accidental mortality (adults)	0 (0 - 1)	0 (0 - 1)
	Respiratory hospitalization (adults 65+)	0 (-17 - 29)	1 (-11 - 21)
	ED visit for asthma (children)	0 (0 - 0)	0 (0 - 0)
	Asthma symptom day (children)	13 (-348 - 972)	9 (-242 - 678)
	Minor restricted activity day (adults)	71 (-31 - 750)	48 (-16 - 513)
	School absence day (children)	77 (-77 - 1180)	53 (-48 - 833)

Note: 95% Confidence Interval is based on the 2.5th and 97.5th percentile of the distribution of benefits generated by the Monet Carlo analysis. Outcomes listed in bold are those for which area-specific baseline incidence rates are used. All other outcomes use baseline incidence rates taken from BenMAP.

Abbreviations: ED: emergency department; $PM_{2.5}$: particulate matter with an aerodynamic diameter less than 2.5 μ m; O_3 : ozone

Most of the health benefits due to reducing emissions at the power plants occurred near the power plants for health outcomes attributable to PM_{2.5} exposures (Figure 5). Figure 5 shows the rates of avoided premature deaths due to PM_{2.5} (A) and O₃ (B). (Comparable figures for the hospitalization outcomes and for Health Benefits Scenario 5 are included in Appendix C.). The spatial patterns in health benefits rates were similar to the changes in exposure observed in Figure 4 for PM_{2.5}. Overall, changes in ozone exposures were small and tended to offset each other (i.e., decreases in the summer months were offset by increases in the winter), and changes in ozone concentrations did not lead to reductions in the number of ozone-related premature deaths. Maps for Health Benefits Scenario 5 (Appendix C) showed similar patterns.

¹ Health Benefits Scenario 4 estimated the health benefits of retiring all units at Martin Drake and the two oldest units at Comanche relative to a 2011 baseline emissions case

² Health Benefits Scenario 5 estimated the health benefits of retiring all units at Martin Drake and the two oldest units at Comanche relative to a counterfactual baseline emissions case which accounts for control technologies installed at Martin Drake in 2016

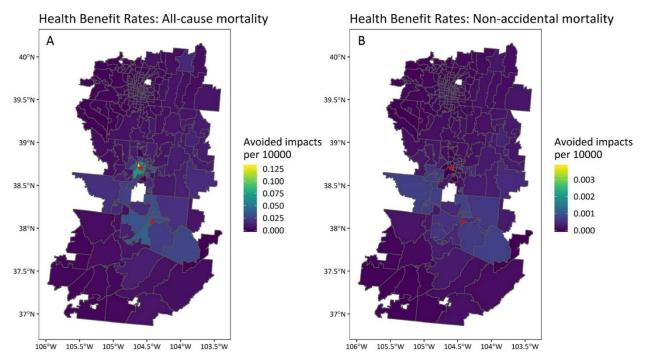


Figure 5. Maps of avoided mortality rates (per 10,000 persons) at the ZCTA level due to reductions in PM_{2.5} (A) and O₃ (B) exposures for Health Benefits Scenario 1

Economic Benefits of Decommissioning Coal-Fired Power Plants in the Southern Front Range

The total monetized benefit of retiring the oldest units at the Comanche plant ranged from \$6.3 million to \$6.4 million per year, depending on whether or not the baseline scenario accounted for the controls installed at Martin Drake in 2016 (Table 6). This result was driven by the estimated avoided premature deaths, which are valued at \$10 million per premature death (US Environmental Protection Agency 2015c). The monetized benefits of retiring the Martin Drake plant (\$35 million per year) were approximately 5 times greater than for retiring the Comanche plant. This increase in benefits was again drive by the greater number of avoided premature deaths due to reductions in exposures as a result of reducing emissions at the Martin Drake facility.

Table 6. Summary of total monetized value of annual health benefits for each of the health benefits scenarios.

		Benefits	Benefits	Benefits
		Scenario 1	Scenario 2	Scenario 3
Pollutant	Outcome	\$10,000's (95% CI)	\$10,000's (95% CI)	\$10,000's (95% CI)
PM _{2.5}	All-cause mortality (adults)	601.8 (369.2 -		3511.6 (2072.7 -
		894.7)	609.8 (374.5 - 906)	5414.6)
	CVD hospitalization (adults 65+)	0.1 (-0.1 - 2.3)	0.1 (-0.1 - 2.4)	1.3 (0 - 10.4)
	Respiratory hospitalization (adults 65+)	0.1 (-0.1 - 1.9)	0.1 (-0.1 - 2)	0.8 (-0.1 - 8.8)
	ED visit for asthma (children)	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
	Asthma symptom day (children)	0 (0 - 0.1)	0 (0 - 0.1)	0 (0 - 0)
	Lower respiratory infection (children)	0 (0 - 0)	0 (0 - 0)	0.4 (0 - 3.1)
	Minor restricted activity day (adults)	0 (0 - 0.6)	0 (0 - 0.7)	0.1 (0 - 1.1)
	Work loss day (adults)	0 (0 - 0.2)	0 (0 - 0.2)	
O ₃	Non-accidental mortality (adults)	25.6 (-94.6 -	25.8 (-94.3 -	
		419.2)	420.8)	12.7 (-203 - 552.7)
	Respiratory hospitalization (adults 65+)	2 (-24.1 - 55.6)	2 (-24.5 - 55.6)	-1.7 (-49.7 - 73.8)
	ED visit for asthma (children)	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)
	Asthma symptom day (children)	0 (-1 - 3)	0 (-1 - 3)	0 (-1.3 - 3.7)
	Minor restricted activity day (adults)	0.2 (-0.1 - 2.6)	0.2 (-0.1 - 2.6)	0.3 (-0.2 - 3.9)
	School absence day (children)	0.3 (-0.2 - 5.7)	0.3 (-0.2 - 5.8)	0.4 (-0.5 - 8.2)
-	Total	630.1 (249.1 -		
		1385.9)	638.4 (254.3 -	3526 (1817.8 -
			1399.1)	6080.2)

Note: Monetized values are reported as 2011\$ projected to a 2024 income level following methods reported in (US Environmental Protection Agency 2015c). 95% Confidence Interval is based on the 2.5th and 97.5th percentile of the distribution of benefits generated by the Monet Carlo analysis. Outcomes listed in bold are those for which area-specific baseline incidence rates are used. All other outcomes use baseline incidence rates taken from BenMAP. All monetized impacts are reported in today's dollar; no discounting was used.

Abbreviations: ED: emergency department; PM_{2.5}: particulate matter with an aerodynamic diameter less than 2.5 μ m; O₃: ozone

The total monetized value of the health benefits of shutting down most of the coal-fired boilers at the Martin Drake and Comanche power plants exceeded those of shutting down the plants individually and was approximately \$42 million (95% CI: \$2.1 million – \$7.2 million) per year (in today's dollars) for Health Benefits Scenario 4 (Table 7). Again, this result was driven by the four estimated avoided premature deaths. Other outcomes contributing to the high monetized value are the hospitalizations for cardiovascular disease (\$16,138; 95%CI: \$142 - \$141,757 per year) and respiratory disease. The monetized value of annual health benefits for Health Benefits Scenario 5 were lower due to the lower number of avoided health benefits (Table 5), but the contributions of avoided premature deaths to the total monetized value remained similar.

Table 7. Summary of total monetized value of annual health benefits for each of the health benefits scenarios.

-		Benefits Scenario 4	Benefits Scenario 5
Pollutant	Outcome	\$10,000's (95% CI)	\$10,000's (95% CI)
PM _{2.5}	All-cause mortality (adults)	4131.8 (2456.5 - 6332.6)	1655.2 (958.3 - 2587.8)
	CVD hospitalization (adults 65+)	1.6 (0.0 - 11.5)	0.7 (0.0 - 4.5)
	Respiratory hospitalization (adults 65+)	1.1 (-0.2 - 10.2)	0.4 (-0.1 - 3.8)
	ED visit for asthma (children)	0.0 (0.0 - 0.0)	0.0(0.0-0.0)
	Asthma symptom day (children)	0.1 (0.0 - 0.8)	0.0 (-0.1 - 0.3)
	Lower respiratory infection (children)	0.0 (0.0 - 0.0)	0.0 (0.0 - 0.0)
	Minor restricted activity day (adults)	0.5 (0 - 3.3)	0.2 (0.0 - 1.3)
	Work loss day (adults)	0.2 (0 - 1.2)	0.1 (0.0 - 0.5)
O ₃	Non-accidental mortality (adults)	43.7 (-250.9 - 719.5)	39.5 (-157.2 - 514.4)
	Respiratory hospitalization (adults 65+)	0.6 (-62.7 - 107.3)	2.1 (-40.4 – 77.0)
	ED visit for asthma (children)	0.0 (0.0 - 0.0)	0.0 (0.0 - 0.0)
	Asthma symptom day (children)	0.1 (-2.1 - 5.8)	0.1 (-1.5 - 4.1)
	Minor restricted activity day (adults)	0.5 (-0.2 - 5.1)	0.3 (-0.1 - 3.5)
	School absence day (children)	0.8 (-0.8 - 11.6)	0.5 (-0.5 - 8.2)
-	Total	4180.8 (2139.7 - 7208.9)	1699.1 (758.4 - 3205.2)

Note: Monetized values are reported as 2011\$ projected to a 2024 income level following methods reported in (US Environmental Protection Agency 2015c). 95% Confidence Interval is based on the 2.5th and 97.5th percentile of the distribution of benefits generated by the Monet Carlo analysis. Outcomes listed in bold are those for which area-specific baseline incidence rates are used. All other outcomes use baseline incidence rates taken from BenMAP. All monetized impacts are reported in today's dollar; no discounting was used. Abbreviations: ED: emergency department; PM_{2.5}: particulate matter with an aerodynamic diameter less than 2.5 µm; O₃: ozone

Environmental Justice Implications of Decommissioning Coal-Fired Power Plants in the Southern Front Range

Many of the communities closest to the Comanche and Drake power plants are considered "environmental justice" communities due to having higher proportions of persons of color and lower median incomes that the surrounding areas. Figure 6 shows the median income and percentage of the population that is not non-Hispanic White alone (i.e., persons of color) in ZCTAs within and near the Pueblo municipal boundary (shown in red). Pueblo, CO has higher proportions of non-White residents and lower median incomes than neighboring communities. Similar patterns were observed for Colorado Springs, where ZCTAs near the Martin Drake power plant had the highest proportion of residents of color and the lowest median incomes (Figure 7).

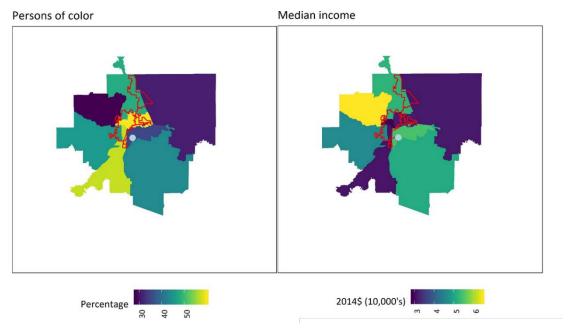


Figure 6. The percentage of ZCTA populations that are persons of color and median income at the ZCTA level for ZCTAs near the Comanche power plant in Pueblo, CO

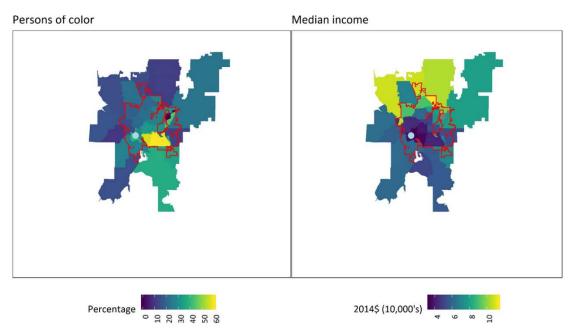


Figure 7. The percentage of ZCTA populations that are persons of color and median income at the ZCTA level for ZCTAs near the Martin Drake power plant in Colorado Springs, CO

Maps displaying the benefits of reduced emissions under Health Benefits Scenario 4 (in which Martin Drake was decommissioned and only one unit at Comanche remained operational)

show that high highest benefit rates (e.g., avoided deaths per 10,000 residents) were incurred in ZCTAs with lower median incomes and higher proportions of residents of color (Figures 8 and 9).

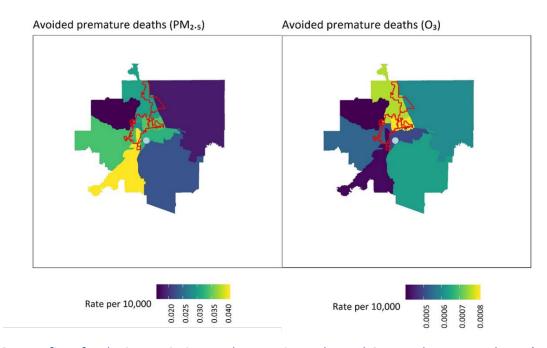


Figure 8. Benefits of reducing emission at the Martin Drake and Comanche power plants (Health Benefits Scenario 4) as avoided deaths attributable to $PM_{2.5}$ and O_3 for ZCTAs near Pueblo, CO

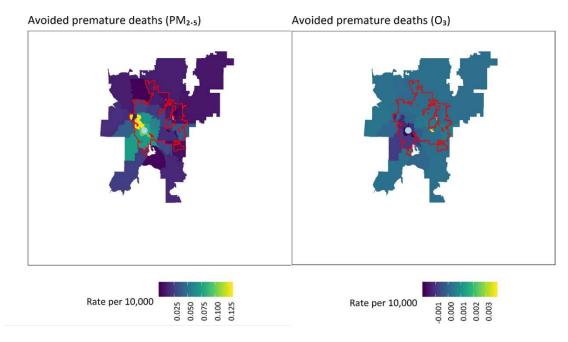


Figure 9. Benefits of reducing emission at the Martin Drake and Comanche power plants (Health Benefits Scenario 4) as avoided deaths attributable to $PM_{2.5}$ and O_3 for ZCTAs near Colorado Springs, CO

As discussed above, most of the health benefits from shutting down both of the power plants (Health Benefits Scenarios 4 and 5) occur in the areas closest to the facilities (Figures 4 and 5), and most of these benefits are due to reductions in PM_{2.5} exposures. Figure 10 shows the median ZCTA-level income and the percentage of the ZCTA population that identifies as a race or ethnicity other than non-Hispanic white alone across the entire study area. The ZCTAs closest to the power plants (indicated with red dots) tended to have lower median incomes and somewhat higher proportions of persons of color (particularly near the Comanche plant in Pueblo, CO). Visual comparisons of these maps in Figure 10 to Figures 4 and 5 suggested that these communities, which would traditionally be identified as environmental justice communities, received greater benefits as a result of the reduction in emissions at the power plants.

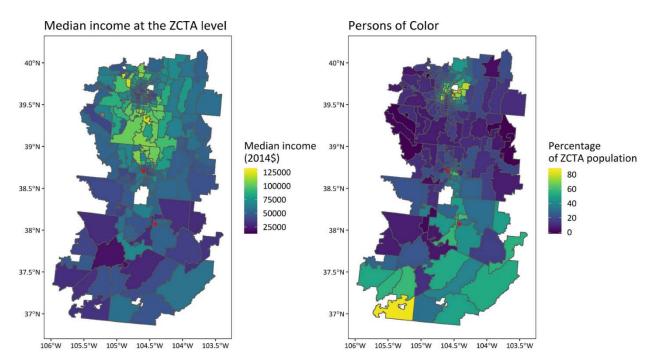


Figure 10. Maps showing the median ZCTA level income and the percentage of each ZCTA population that is persons of color (excludes non-Hispanic white alone)

The Concentration Index curves, which measure the degree of inequality in health benefits by social advantage, indicated that ZCTAs with lower median incomes tended to receive a higher proportion of the health benefits (measured as avoided deaths and hospitalizations per 10,000 people), and that populations with a lower proportion of persons of color tended to receive similar health benefits relative to ZCTAs with higher proportions of persons of color.

Figure 11 shows the concentration curves for avoided PM_{2.5} and ozone mortality rates when ranking populations by income and race/ethnicity for Health Benefits Scenario 4. (Similar curves for Health Benefits Scenario 1-3 and 5 are in Appendix C.) Because avoided premature deaths are a positive outcome, concentration index curves above the 1:1 equality line are considered favorable to ZCTAs with lower social advantage; in other words, when the curve is above the 1:1 line, ZCTAs that are typically disadvantaged in other ways receive a greater proportion of the health benefits of decommissioning the power plants. Figure 11 also highlights that the degree of health benefits from reductions in PM_{2.5} may be stronger than that for health benefits from reductions in O₃. This is likely driven by the more localized change in PM_{2.5} concentrations relative to those of O₃ (Figure 3). Plots for Health Benefits Scenarios 1-3 and 5 (Appendix B) show similar patterns.

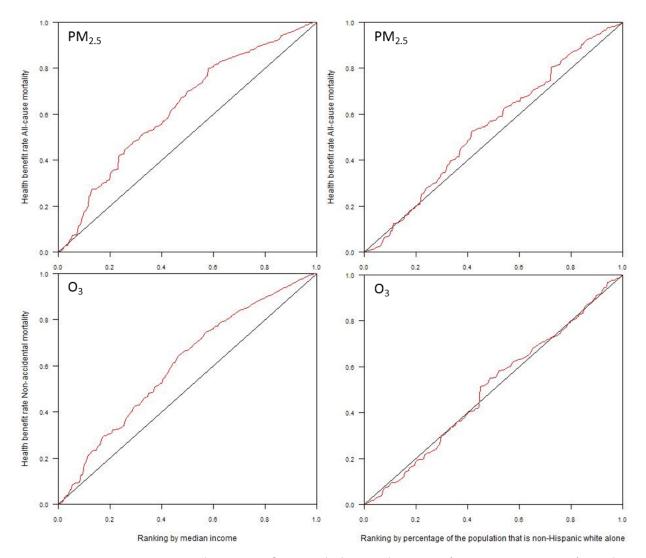


Figure 11. Concentration index curves for avoided mortality rates (per 10,000 persons) resulting from reduced $PM_{2.5}$ or O_3 exposures under Health Benefits Scenario 1 when ranking ZCTAs by median income and the percentage of the population that is non-Hispanic white alone

A comparison of two ZCTA in the study area helps to illustrate the environmental justice implications of the HIA results. We compared two ZCTAs near the Martin Drake power plant in Colorado Springs: 80904 and 80924 (Figure 12).

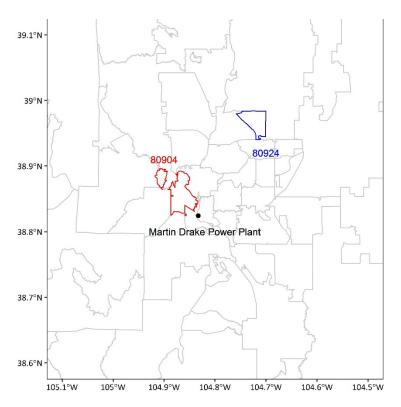


Figure 12. Map showing the location of two ZIP codes in Colorado Springs near the Martin Drake power plant

These two ZIP codes are located less than 20 km apart, but have very different degrees of social advantage (Table 8). For example, ZIP code 80904 had a median income of \$44,928 compared to 80924, which had a median income of \$113,503. ZIP Code 80904 is located in close proximity to the Martin Drake plant and experienced a larger reduction in average PM_{2.5} concentrations (0.17 µg/m³) compared with ZIP code 80924 (0.06 µg/m³) under Health Benefits Scenario 4. This larger decrease in exposures resulted in about 0.13 avoided deaths each year in 80904, or about one death every six years. The avoided death rate for this ZIP code (0.13 per 10,000) is roughly 24 times higher than the avoided death rate for 80524 (0.005 per 10,000). Both communities were shown to have decreased exposures and resulting health benefits as a result of reduced emissions, and the ZCTA-level analysis suggested that many of those benefits are incurred by communities which have historically dealt with higher environmental pollutant burdens.

Table 8. Summary of population, changes in exposure, health benefits, and, socioeconomic characteristics for two ZIP codes near the Martin Drake power plant under Health Benefits Scenario 4

	80924	80904
Mean change in PM _{2.5} (μg/m ³)	0.055	0.17
Avoided deaths per year (n, 95% CI)	0.003 (0.001 - 0.006)	0.258 (0.15 - 0.403)
Avoided death rate (n per 10,000)	0.005	0.130
Total population (n)	5,975	19,884
Population that is persons of color (%)	27.7	14.1
Median income (2014\$)	113,503	44,928

Discussion

We conducted a quantitative HIA to assess the potential health benefits of decommissioning the Martin Drake power plant in Colorado Springs and substantially reducing emissions at the Comanche power plant in Pueblo, CO for residents of Colorado's Southern Front Range. We found that reducing emissions at these facilities would have modest health benefits for the region, including 2 – 4 avoided premature deaths each year, and that these benefits would largely be concentrated in the ZCTAs closest to the power plants. In our assessment, health benefits were greater for reductions in PM_{2.5} than for reductions in O₃.

Although the total number of health benefits is modest relative to the number of deaths and hospitalizations that occur in the area each year, we found that ZIP codes with lower median incomes would benefit most from the shutdown of these facilities. Throughout the United States, lower income populations are more likely to live near industrial facilities such as the coal-fired power plants identified in this HIA (Mohai et al. 2009). Our finding that low-income ZIP codes receive a substantial proportion of the health benefits has potential environmental justice implications for the study area.

Limitations

There are several limitations to this analysis that should be noted when interpreting the results. First, as in most HIAs, the concentration response functions implemented in this analysis were based on previous epidemiologic studies and the from these original studies may not be

fully generalizable to our study area (Hubbell et al. 2009). When available, we have selected concentrations-response functions from nationally representative studies; for example. the American Cancer Society cohort study from which our PM_{2.5}-related mortality function was taken is considered to be robust and has been used in other HIAs (Fann et al. 2012b; Levy et al. 2002; US Environmental Protection Agency 2012b). However, generalizability to the entire study population may be limited, as the ACS cohort has few non-white participants, with higher than average income education levels compared to the general population (Krewski et al. 2009). For other outcomes for which national studies were not available, e.g., hospitalizations, we pooled concentration response coefficients from multiple studies. This pooling approach help to address differences across study populations and is also widely used in the HIA literature (Hubbell et al. 2009; US Environmental Protection Agency 2012b). Further, concentration response functions used for this analysis were applied only to specific age groups. Optimally, health impact estimates should implement age, sex, and race-specific concentration response functions that can address demographic risk differences in exposure and risk differences in the outcome. However, availability of sex- and race-specific health outcome rates and concentration response functions is limited for the study area.

Second, the concentration-response functions implemented in this study are based upon studies that have assessed the relation between ambient (outdoor) exposures and health. Given that most people, particularly adults, tend to spend more of their time indoors, there is some uncertainty related to actual exposure (and inhaled dose) to pollutants. However, the original epidemiological studies from which the concentration-response functions are taken typically rely on outdoor concentrations at residential locations.

Third, there are some uncertainties inherent in using chemical transport models to assess ambient concentrations of $PM_{2.5}$ and O_3 . Model predictions are provided at a 4 km resolution and hence the model cannot resolve gradients or hotspots within the 4 x 4 km grid cell that might result in certain populations within a grid cell (e.g., those living close to a major highway) experiencing much higher concentrations than the rest (e.g., those living in a residential neighborhood). Instead, the model predictions provide an average concentration exposure over

the 4 x 4 km grid cell. The CMAQ results do not agree perfectly with observations of PM_{2.5} and O₃ at all sites but agree reasonably with the ensemble of measurements over the four state modeled domain. A comprehensive model-measurement comparison is beyond the scope of this work, but more details on the performance of the CMAQ model, run with a slightly different configuration, of 2011 found over the entire vear can be here: http://views.cira.colostate.edu/tsdw/Modeling/ModelObsComparison_IWDW.aspx. Regardless of the model performance of the absolute concentrations at a specific site, the model offers fairly robust predictions to changes in pollutant concentrations. In this study, we have only examined the change in pollutant concentrations from reducing/eliminating emissions from these power plants, without modifying emissions from other sources. Hence, the benefits simulated are only attributed to the closure at these power plants and the net costs/benefits in the future could be different than those simulated here as emissions from other sources will vary with time.

Fourth, in our analysis, we assumed that the average exposure estimates for the modeled periods (roughly 60 days per year) implemented in the study represents a consistent long-term exposure for the study population. This assumes that sources and concentrations of exposure have remained fairly stable over time, and that the population is static. These underlying assumptions are common to HIA, as well as epidemiologic studies, but nonetheless may cause some uncertainty in the estimates of avoided deaths, particularly in neighborhoods that have had meaningful changes in ambient air pollution levels or a high level of residential mobility.

Finally, the health benefits outline in this report should be considered a subset of the total benefits of decommissioning the two coal-fired power plants. In this HIA, we were only able to include health impacts for which there is sufficient evidence of causality (US Environmental Protection Agency 2009, 2013). There are likely other health outcomes that would be affected be reductions in ambient concentrations of PM_{2.5} and O₃, including neurocognitive effects and adverse birth outcomes. Similarly, we have not accounted for environmental or climate change impacts resulting from reduced emissions at these facilities. Due to this lack of complete benefits assessment, any cost-benefit analysis based on the monetized impacts presented in this report

would likely under-represent the true economic value of the benefits of decommissioning the plants.

References

- Adler NE, Newman K. 2002. Socioeconomic disparities in health: pathways and policies. Health Aff Proj Hope 21:60–76; doi:10.1377/hlthaff.21.2.60.
- Bell M, McDermott A, Zeger, Samet JM, Dominici F. 2004. Ozone and short-term mortality in 95 us urban communities, 1987-2000. JAMA 292:2372–2378; doi:10.1001/jama.292.19.2372.
- Bell ML, Dominici F, Samet JM. 2005. A Meta-Analysis of Time-Series Studies of Ozone and Mortality With Comparison to the National Morbidity, Mortality, and Air Pollution Study. Epidemiol Camb Mass 16: 436–445.
- Bell ML, Ebisu K, Peng RD, Samet JM, Dominici F. 2009. Hospital admissions and chemical composition of fine particle air pollution. Am J Respir Crit Care Med 179:1115–1120; doi:10.1164/rccm.200808-1240OC.
- Bell ML, Ebisu K, Peng RD, Walker J, Samet JM, Zeger SL, et al. 2008. Seasonal and Regional Short-term Effects of Fine Particles on Hospital Admissions in 202 US Counties, 1999–2005. Am J Epidemiol 168:1301–1310; doi:10.1093/aje/kwn252.
- Bell ML, Peng RD, Dominici F. 2006. The Exposure-Response Curve for Ozone and Risk of Mortality and the Adequacy of Current Ozone Regulations. Environ Health Perspect 114: 532–536.
- Bhatia R, Farhang L, Heller J, Orenstein M, Richardson M, Wernham A. 2014. Minimum elements and practice standards for health impact assessments, Version 3.
- Bhatia R, Seto E. 2011. Quantitative estimation in Health Impact Assessment: Opportunities and challenges. Environ Impact Assess Rev 31:301–309; doi:10.1016/j.eiar.2010.08.003.
- Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV, et al. 2010. Particulate Matter Air Pollution and Cardiovascular Disease An Update to the Scientific Statement From the American Heart Association. Circulation 121:2331–2378; doi:10.1161/CIR.0b013e3181dbece1.
- Brown P. 1995. Race, class, and environmental health: a review and systematization of the literature. Environ Res 69:15–30; doi:10.1006/enrs.1995.1021.
- Brulle RJ, Pellow DN. 2006. ENVIRONMENTAL JUSTICE: Human Health and Environmental Inequalities. Annu Rev Public Health 27:103–124; doi:10.1146/annurev.publhealth.27.021405.102124.
- Caiazzo F, Ashok A, Waitz IA, Yim SHL, Barrett SRH. 2013. Air pollution and early deaths in the United States. Part I: Quantifying the impact of major sectors in 2005. Atmos Environ 79:198–208; doi:10.1016/j.atmosenv.2013.05.081.
- Carlton AG, Baker KR. 2011. Photochemical Modeling of the Ozark Isoprene Volcano: MEGAN, BEIS, and Their Impacts on Air Quality Predictions. Environ Sci Technol 45:4438–4445; doi:10.1021/es200050x.

- Cesaroni G, Badaloni C, Gariazzo C, Stafoggia M, Sozzi R, Davoli M, et al. 2013. Long-term exposure to urban air pollution and mortality in a cohort of more than a million adults in Rome. Environ Health Perspect 121:324–331; doi:10.1289/ehp.1205862.
- Chan G, Stavins R, Stowe R, Sweeney R. 2012. The SO2 Allowance Trading System and the Clean Air Act Amendments of 1990: Reflections on Twenty Years of Policy Innovation.; doi:10.3386/w17845.
- Cole BL, Shimkhada R, Morgenstern H, Kominski G, Fielding JE, Wu S. 2005. Projected health impact of the Los Angeles City living wage ordinance. J Epidemiol Community Health 59:645–650; doi:10.1136/jech.2004.028142.
- Colorado Air Quality Control Commission. 2016. Moderate Area Ozone SIP for the Denver Metro and North Front Range Nonattainment Area. State Implementation Plan for the 2008 8-Hour Ozone National Ambient Air Quality Standard.
- Colorado Department of Public Health & Environment. 2017. Colorado Annual Monitoring Network Plan 2017.
- Colorado Springs Utility. 2018. Martin Drake Power Plant. Available: https://www.csu.org/pages/martin-drake-b.aspx [accessed 21 June 2018].
- Cooper OR, Parrish DD, Stohl A, Trainer M, Nédélec P, Thouret V, et al. 2010. Increasing springtime ozone mixing ratios in the free troposphere over western North America. Nature 463:344–348; doi:10.1038/nature08708.
- Correia AW, Pope CA, Dockery DW, Wang Y, Ezzati M, Dominici F. 2013. Effect of air pollution control on life expectancy in the United States: an analysis of 545 U.S. counties for the period from 2000 to 2007. Epidemiol Camb Mass 24:23–31; doi:10.1097/EDE.0b013e3182770237.
- Daniels MJ, Dominici F, Zeger SL, Samet JM. 2004. The National Morbidity, Mortality, and Air Pollution Study. Part III: PM10 concentration-response curves and thresholds for the 20 largest US cities. Res Rep Health Eff Inst 1–21.
- Dannenberg AL. 2016. A Brief History of Health Impact Assessment in the United States. Chron Health Impact Assess 1; doi:10.18060/21348.
- Delucchi MA. 2003. Environmental Externalities of Motor Vehicle Use. In: *Handbook of Transport and the Environment*. Vol. 4 of *Handbooks in Transport*. Emerald Group Publishing Limited. 429–449.
- Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, et al. 1996. Health effects of acid aerosols on North American children: respiratory symptoms. Environ Health Perspect 104: 500.
- Dominici F, McDermott A, Zeger SL, Samet JM. 2003. National maps of the effects of particulate matter on mortality: exploring geographical variation. Environ Health Perspect 111: 39–44.
- Eze IC, Hemkens LG, Bucher HC, Hoffmann B, Schindler C, Künzli N, et al. 2015. Association between ambient air pollution and diabetes mellitus in Europe and North America: systematic review and meta-analysis. Environ Health Perspect 123:381–389; doi:10.1289/ehp.1307823.

- Fann N, Baker KR, Fulcher CM. 2012a. Characterizing the PM_{2.5}-related health benefits of emission reductions for 17 industrial, area and mobile emission sectors across the U.S. Environ Int 49:141–151; doi:10.1016/j.envint.2012.08.017.
- Fann N, Fulcher CM, Hubbell BJ. 2009. The influence of location, source, and emission type in estimates of the human health benefits of reducing a ton of air pollution. Air Qual Atmosphere Health 2:169–176; doi:10.1007/s11869-009-0044-0.
- Fann N, Lamson AD, Anenberg SC, Wesson K, Risley D, Hubbell BJ. 2012b. Estimating the National Public Health Burden Associated with Exposure to Ambient PM2.5 and Ozone. Risk Anal 32:81–95; doi:10.1111/j.1539-6924.2011.01630.x.
- Fann N, Roman HA, Fulcher CM, Gentile MA, Hubbell BJ, Wesson K, et al. 2011. Maximizing Health Benefits and Minimizing Inequality: Incorporating Local-Scale Data in the Design and Evaluation of Air Quality Policies. Risk Anal 31:908–922; doi:10.1111/j.1539-6924.2011.01629.x.
- Forouzanfar MH, Afshin A, Alexander LT, Anderson HR, Bhutta ZA, Biryukov S, et al. 2016. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. The Lancet 388:1659–1724; doi:10.1016/S0140-6736(16)31679-8.
- Gantt B, Kelly JT, Bash JO. 2015. Updating sea spray aerosol emissions in the Community Multiscale Air Quality (CMAQ) model version 5.0.2. Geosci Model Dev 8:3733–3746; doi:10.5194/gmd-8-3733-2015.
- Gauderman WJ, Urman R, Avol E, Berhane K, McConnell R, Rappaport E, et al. 2015. Association of improved air quality with lung development in children. N Engl J Med 372:905–913; doi:10.1056/NEJMoa1414123.
- Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, et al. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. Epidemiology 12: 43–54.
- Glad JA, Brink LL, Talbott EO, Lee PC, Xu X, Saul M, et al. 2012. The relationship of ambient ozone and PM2. 5 levels and asthma emergency department visits: Possible influence of gender and ethnicity. Arch Environ Occup Health 67: 103–108.
- Grubesic TH, Matisziw TC. 2006. On the use of ZIP codes and ZIP code tabulation areas (ZCTAs) for the spatial analysis of epidemiological data. Int J Health Geogr 5:58; doi:10.1186/1476-072X-5-58.
- Harper S, Ruder E, Roman HA, Geggel A, Nweke O, Payne-Sturges D, et al. 2013. Using Inequality Measures to Incorporate Environmental Justice into Regulatory Analyses. Int J Environ Res Public Health 10:4039–4059; doi:10.3390/ijerph10094039.
- Health Effects Institute. 2010. Traffic-related air pollution: a critical review of the literature on emissions, exposure, and health effects.

- HEI Panel on the Health Effects of Traffic-Related Air Pollution. 2009. Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects. 2009.
- Holland M, Watkiss P, Pye S. 2005. Cost-Benefit Analysis of Policy Option Scenarios for the Clean Air for Europe programme.
- Hubbell B. 2012. Understanding urban exposure environments: new research directions for informing implementation of U.S. air quality standards. Air Qual Atmosphere Health 5:259–267; doi:10.1007/s11869-011-0153-4.
- Hubbell BJ, Fann N, Levy JI. 2009. Methodological considerations in developing local-scale health impact assessments: balancing national, regional, and local data. Air Qual Atmosphere Health 2:99–110; doi:10.1007/s11869-009-0037-z.
- Intermountain West Data Warehouse [IWDW]. 2018. Intermountain West Data Warehouse. Available: http://views.cira.colostate.edu/tsdw/ [accessed 11 July 2018].
- Ito K, De Leon SF, Lippmann M. 2005. Associations between ozone and daily mortality: analysis and meta-analysis. Epidemiol Camb Mass 16: 446–457.
- Ito K, Thurston GD, Silverman RA. 2007. Characterization of PM2.5, gaseous pollutants, and meteorological interactions in the context of time-series health effects models. J Expo Sci Environ Epidemiol 17 Suppl 2:S45-60; doi:10.1038/sj.jes.7500627.
- Jerrett M, Burnett RT, Kanaroglou P, Eyles J, Finkelstein N, Giovis C, et al. 2001. A GIS–Environmental Justice Analysis of Particulate Air Pollution in Hamilton, Canada. Environ Plan Econ Space 33:955–973; doi:10.1068/a33137.
- Jerrett M, Burnett RT, Pope III CA, Ito K, Thurston G, Krewski D, et al. 2009. Long-Term Ozone Exposure and Mortality. N Engl J Med 360:1085–1095; doi:10.1056/NEJMoa0803894.
- Katsouyanni K, Samet JM, Anderson HR, Atkinson R, Le Tertre A, Medina S, et al. 2009. Air pollution and health: a European and North American approach (APHENA). Res Rep Health Eff Inst 5–90.
- Kelsall JE, Samet JM, Zeger SL, Xu J. 1997. Air pollution and mortality in Philadelphia, 1974-1988. Am J Epidemiol 146: 750–762.
- Kemm JR. 2000. Can health impact assessment fulfil the expectations it raises? Public Health 114: 431–433.
- Kloog I, Coull BA, Zanobetti A, Koutrakis P, Schwartz JD. 2012. Acute and Chronic Effects of Particles on Hospital Admissions in New-England. PLoS ONE 7:e34664; doi:10.1371/journal.pone.0034664.
- Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, et al. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Res Rep Health Eff Inst 5–114.

- Künzli N, Kaiser R, Medina S, Studnicka M, Chanel O, Filliger P, et al. 2000. Public-health impact of outdoor and traffic-related air pollution: a European assessment. Lancet Lond Engl 356:795–801; doi:10.1016/S0140-6736(00)02653-2.
- Laden F, Schwartz J, Speizer FE, Dockery DW. 2006. Reduction in Fine Particulate Air Pollution and Mortality. Am J Respir Crit Care Med 173:667–672; doi:10.1164/rccm.200503-443OC.
- Lei Chen BLJ Wei Yang, Stanley T Omaye. 2000. Elementary School Absenteeism and Air Pollution. Inhal Toxicol 12:997–1016; doi:10.1080/08958370050164626.
- Leigh JP, Geraghty EM. 2008. High Gasoline Prices and Mortality From Motor Vehicle Crashes and Air Pollution. J Occup Environ Med 50:249; doi:10.1097/JOM.0b013e318162f5c4.
- Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A. 2015. The contribution of outdoor air pollution sources to premature mortality on a global scale. Nature 525:367-+; doi:10.1038/nature15371.
- Levy JI, Greco SL, Spengler JD. 2002. The importance of population susceptibility for air pollution risk assessment: a case study of power plants near Washington, DC. Environ Health Perspect 110: 1253–1260.
- Levy JI, Hanna SR. 2011. Spatial and temporal variability in urban fine particulate matter concentrations. Environ Pollut Barking Essex 1987 159:2009–2015; doi:10.1016/j.envpol.2010.11.013.
- Maguire K, Sheriff G. 2011. Comparing Distributions of Environmental Outcomes for Regulatory Environmental Justice Analysis. Int J Environ Res Public Health 8:1707–1726; doi:10.3390/ijerph8051707.
- Mar TF, Koenig JQ. 2009. Relationship between visits to emergency departments for asthma and ozone exposure in greater Seattle, Washington. Ann Allergy Asthma Immunol Off Publ Am Coll Allergy Asthma Immunol 103:474–479; doi:10.1016/S1081-1206(10)60263-3.
- Mar TF, Koenig JQ, Primomo J. 2010. Associations between asthma emergency visits and particulate matter sources, including diesel emissions from stationary generators in Tacoma, Washington. Inhal Toxicol 22:445–448; doi:10.3109/08958370903575774.
- Mar TF, Larson TV, Stier RA, Claiborn C, Koenig JQ. 2004. An analysis of the association between respiratory symptoms in subjects with asthma and daily air pollution in Spokane, Washington. Inhal Toxicol 16:809–815; doi:10.1080/08958370490506646.
- Masters RK, Hummer RA, Powers DA. 2012. Educational Differences in U.S. Adult Mortality: A Cohort Perspective. Am Sociol Rev 77:548–572; doi:10.1177/0003122412451019.
- Matte TD, Ross Z, Kheirbek I, Eisl H, Johnson S, Gorczynski JE, et al. 2013. Monitoring intraurban spatial patterns of multiple combustion air pollutants in New York City: Design and implementation. J Expo Sci Environ Epidemiol 23:223–231; doi:10.1038/jes.2012.126.
- McKenzie LM, Witter RZ, Newman LS, Adgate JL. 2012. Human health risk assessment of air emissions from development of unconventional natural gas resources. Sci Total Environ 424:79–87; doi:10.1016/j.scitotenv.2012.02.018.

- Mesa-Frias M, Chalabi Z, Foss AM. 2013. Assessing framing assumptions in quantitative health impact assessments: A housing intervention example. Environ Int 59:133–140; doi:10.1016/j.envint.2013.06.002.
- Mesa-Frias M, Chalabi Z, Foss AM. 2014. Quantifying uncertainty in health impact assessment: A case-study example on indoor housing ventilation. Environ Int 62:95–103; doi:10.1016/j.envint.2013.10.007.
- Miranda ML, Edwards SE, Keating MH, Paul CJ. 2011. Making the Environmental Justice Grade: The Relative Burden of Air Pollution Exposure in the United States. Int J Environ Res Public Health 8:1755–1771; doi:10.3390/ijerph8061755.
- Mohai P, Lantz PM, Morenoff J, House JS, Mero RP. 2009. Racial and socioeconomic disparities in residential proximity to polluting industrial facilities: evidence from the Americans' Changing Lives Study. Am J Public Health 99 Suppl 3:S649-656; doi:10.2105/AJPH.2007.131383.
- Moolgavkar SH. 2003. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook counties. Revis Anal Time-Ser Stud Air Pollut Health 183–198.
- Moolgavkar SH. 2000. Air pollution and hospital admissions for diseases of the circulatory system in three US metropolitan areas. J Air Waste Manag Assoc 50: 1199–1206.
- Moolgavkar SH, Luebeck EG, Hall TA, Anderson EL. 1995. Air pollution and daily mortality in Philadelphia. Epidemiol Camb Mass 6: 476–484.
- Morello-Frosch R, Zuk M, Jerrett M, Shamasunder B, Kyle AD. 2011. Understanding The Cumulative Impacts Of Inequalities In Environmental Health: Implications For Policy. Health Aff (Millwood) 30:879–887; doi:10.1377/hlthaff.2011.0153.
- Morrow RH, Bryant JH. 1995. Health policy approaches to measuring and valuing human life: conceptual and ethical issues. Am J Public Health 85:1356–1360; doi:10.2105/AJPH.85.10.1356.
- Mortimer KM, Neas LM, Dockery DW, Redline S, Tager IB. 2002. The effect of air pollution on inner-city children with asthma. Eur Respir J 19:699–705; doi:10.1183/09031936.02.00247102.
- Murray CJ, Lopez AD. 1997. Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. The Lancet 349:1498–1504; doi:10.1016/S0140-6736(96)07492-2.
- National Research Council. 2004. *Air quality management in the United States*. National Academies Press: Washington, DC.
- National Research Council. 2011. *Improving health in the United States: The role of health impact assessment*. National Academies Press:Washington, DC.
- New York City Department of Health and Mental Hygiene. 2013. New York City Community Air Survey (NYCCAS): New York City Trends in Air Pollution and its Health Consequences.

- O'Connell E, Hurley F. 2009. A review of the strengths and weaknesses of quantitative methods used in health impact assessment. Public Health 123:306–310; doi:10.1016/j.puhe.2009.02.008.
- O'Connor GT, Neas L, Vaughn B, Kattan M, Mitchell H, Crain EF, et al. 2008. Acute respiratory health effects of air pollution on children with asthma in US inner cities. J Allergy Clin Immunol 121:1133-1139.e1; doi:10.1016/j.jaci.2008.02.020.
- O'Donnell O, van Doorslaer E, Wagstaff A, Lindelow M. 2008. *Analyzing health equity using household survey data: A guide to techniques and their implementation*. The World Bank.
- Ostro B, Lipsett M, Mann J, Braxton-Owens H, White M, others. 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. Epidemiology 12: 200–208.
- Ostro BD. 1987. Air pollution and morbidity revisited: A specification test. J Environ Econ Manag 14:87–98; doi:10.1016/0095-0696(87)90008-8.
- Ostro BD, Rothschild S. 1989. Air pollution and acute respiratory morbidity: An observational study of multiple pollutants. Environ Res 50:238–247; doi:10.1016/S0013-9351(89)80004-0.
- Pappas G, Queen S, Hadden W, Fisher G. 1993. The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. N Engl J Med 329:103–109; doi:10.1056/NEJM199307083290207.
- Park SK, Wang W. 2014. Ambient Air Pollution and Type 2 Diabetes: A Systematic Review of Epidemiologic Research. Curr Environ Health Rep 1:275–286; doi:10.1007/s40572-014-0017-9.
- Parry IWH, Walls M, Harrington W. 2007. Automobile Externalities and Policies. J Econ Lit 45:373–399; doi:10.1257/jel.45.2.373.
- Pearce J, Kingham S, Zawar-Reza P. 2006. Every Breath You Take? Environmental Justice and Air Pollution in Christchurch, New Zealand. Environ Plan Econ Space 38:919–938; doi:10.1068/a37446.
- Peel JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Todd K, et al. 2005. Ambient Air Pollution and Respiratory Emergency Department Visits: Epidemiology 16:164–174; doi:10.1097/01.ede.0000152905.42113.db.
- Peng RD, Bell ML, Geyh AS, McDermott A, Zeger SL, Samet JM, et al. 2009. Emergency admissions for cardiovascular and respiratory diseases and the chemical composition of fine particle air pollution. Env Health Perspect 117: 957–963.
- Peng RD, Chang HH, Bell ML, et al. 2008. Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among medicare patients. JAMA 299:2172–2179; doi:10.1001/jama.299.18.2172.
- Pope CA, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, et al. 2009. Cardiovascular Mortality and Exposure to Airborne Fine Particulate Matter and Cigarette Smoke Shape of the Exposure-Response Relationship. Circulation 120:941–948; doi:10.1161/CIRCULATIONAHA.109.857888.

- Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, et al. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287: 1132–1141.
- Pope CA, Cropper M, Coggins J, Cohen A. 2015. Health benefits of air pollution abatement policy: Role of the shape of the concentration-response function. J Air Waste Manag Assoc 65:516–522; doi:10.1080/10962247.2014.993004.
- Popp D. 2001. Pollution Control Innovations and the Clean Air Act of 1990.; doi:10.3386/w8593.
- Rhodus J, Fulk F, Autrey B, O'Shea S, Roth A. 2013. A review of health impact assessments in the U.S.: Current state-of-science, best practices and area for improvement.
- Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, et al. 2000. The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. Res Rep Health Eff Inst 94: 5–70; discussion 71-79.
- Sarnat SE, Sarnat JA, Mulholland J, Isakov V, Özkaynak H, Chang HH, et al. 2013. Application of alternative spatiotemporal metrics of ambient air pollution exposure in a time-series epidemiological study in Atlanta. J Expo Sci Environ Epidemiol 23:593–605; doi:10.1038/jes.2013.41.
- Sarwar G, Simon H, Bhave P, Yarwood G. 2012. Examining the impact of heterogeneous nitryl chloride production on air quality across the United States. Atmospheric Chem Phys 12:6455–6473; doi:https://doi.org/10.5194/acp-12-6455-2012.
- Schildcrout JS, Sheppard L, Lumley T, Slaughter JC, Koenig JQ, Shapiro GG. 2006. Ambient Air Pollution and Asthma Exacerbations in Children: An Eight-City Analysis. Am J Epidemiol 164:505–517; doi:10.1093/aje/kwj225.
- Schulz A, Northridge ME. 2004. Social Determinants of Health: Implications for Environmental Health Promotion. Health Educ Behav 31:455–471; doi:10.1177/1090198104265598.
- Schwartz J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. Thorax 50: 531–538.
- Schwartz J, Laden F, Zanobetti A. 2002. The concentration-response relation between PM(2.5) and daily deaths. Environ Health Perspect 110: 1025–1029.
- Schwartz J, Neas LM. 2000. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. Epidemiology 11: 6–10.
- Skamarock WC, Klemp JB, Dudhia J, Gill DO, Barker DM, Duda MG, et al. 2008. A Description of the Advanced Research WRF Version 3.
- Slaughter JC, Kim E, Sheppard L, Sullivan JH, Larson TV, Claiborn C. 2005. Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington. J Expo Sci Environ Epidemiol 15: 153–159.

- Song F, Young Shin J, Jusino-Atresino R, Gao Y. 2011. Relationships among the springtime ground–level NOx, O3 and NO3 in the vicinity of highways in the US East Coast. Atmospheric Pollut Res 2:374–383; doi:10.5094/APR.2011.042.
- Stanton Anleu B. 2016. Colorado Springs Utilities takes charge of Drake scrubbers. The Gazette, September 26.
- Tchepel O, Dias D. 2011. Quantification of health benefits related with reduction of atmospheric PM₁₀ levels: implementation of population mobility approach. Int J Environ Health Res 21:189–200; doi:10.1080/09603123.2010.520117.
- Turner MC, Cohen A, Jerrett M, Gapstur SM, Diver WR, Pope CA, et al. 2014. Interactions between cigarette smoking and fine particulate matter in the Risk of Lung Cancer Mortality in Cancer Prevention Study II. Am J Epidemiol 180:1145–1149; doi:10.1093/aje/kwu275.
- US Census Bureau. 2014. 2010-2014 American Community Survey (ACS) 5-year Estimates. Available: https://www.census.gov/programs-surveys/acs/ [accessed 6 October 2016].
- US Energy Information Administration. 2018a. Coal and the Environment. Energy Explain. Available: https://www.eia.gov/energyexplained/index.php?page=coal_environment [accessed 8 June 2018].
- US Energy Information Administration. 2018b. EIA projects that U.S. coal demand will remain flat for several decades. Today Energy. Available: https://www.eia.gov/todayinenergy/detail.php?id=35572 [accessed 8 June 2018].
- US Energy Information Administration. 2018c. What is U.S. electricity generation by energy source? Available: https://www.eia.gov/tools/faqs/faq.php?id=427&t=3 [accessed 26 June 2018].
- US Environmental Protection Agency. 2018. 8-Hour Ozone (2008) Designated Area/State Information with Design Values. Green Book. Available: https://www3.epa.gov/airquality/greenbook/hbtcw.html [accessed 26 June 2018].
- US Environmental Protection Agency. 2014. Air Quality Trends. Available: http://www.epa.gov/airtrends/aqtrends.html [accessed 11 March 2015].
- US Environmental Protection Agency. 2015a. BenMAP User's Manual Appendices.
- US Environmental Protection Agency. 2016a. EJ 2020 Action Agenda: Environmental Justice Strategic Plan 2016-2020.
- US Environmental Protection Agency. 2016b. Environmental Benefits Mapping and Analysis Program Community Edition (BenMAP-CE). Available: https://www.epa.gov/benmap [accessed 6 May 2016].
- US Environmental Protection Agency. 2010. Guidelines for Preparing Economic Analyses.
- US Environmental Protection Agency. 2013. *Integrated Science Assessment for Ozone and Related Photochemical Oxidants*.

- US Environmental Protection Agency. 2009. Integrated Science Assessment for Particulate Matter.
- US Environmental Protection Agency. 2012a. National Emissions Inventory. Available: http://www.epa.gov/ttn/chief/net/2005inventory.html#inventorydata [accessed 10 March 2015].
- US Environmental Protection Agency. 2016c. National Multi-pollutant Emissions Comparison. Air Emiss Invent. Available: https://www.epa.gov/air-emissions-inventories/multi-pollutant-comparison [accessed 26 June 2018].
- US Environmental Protection Agency. 2015b. Regulatory Impact Analysis for Residential Wood Heaters NSPS Revision.
- US Environmental Protection Agency. 2012b. Regulatory Impact Analysis for the Final Revisions to the National Ambient Air Quality Standards for Particulate Matter.
- US Environmental Protection Agency. 2015c. Regulatory Impact Analysis of the Final Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone.
- Vedal S, Hannigan MP, Dutton SJ, Miller SL, Milford JB, Rabinovitch N, et al. 2009. The Denver Aerosol Sources and Health (DASH) Study: Overview and Early Findings. Atmospheric Environ Oxf Engl 1994 43:1666–1673; doi:10.1016/j.atmosenv.2008.12.017.
- Vrijheid M, Martinez D, Aguilera I, Ballester F, Basterrechea M, Esplugues A, et al. 2012. Socioeconomic status and exposure to multiple environmental pollutants during pregnancy: evidence for environmental inequity? J Epidemiol Community Health 66:106–113; doi:10.1136/jech.2010.117408.
- Wilhelm M, Ghosh JK, Su J, Cockburn M, Jerrett M, Ritz B. 2012. Traffic-related air toxics and term low birth weight in Los Angeles County, California. Environ Health Perspect 120:132–138; doi:10.1289/ehp.1103408.
- Wilson AM, Wake CP, Kelly T, Salloway JC. 2005. Air pollution, weather, and respiratory emergency room visits in two northern New England cities: an ecological time-series study. Environ Res 97:312–321; doi:10.1016/j.envres.2004.07.010.
- Witter RZ, McKenzie L, Stinson KE, Scott K, Newman LS, Adgate J. 2013. The Use of Health Impact Assessment for a Community Undergoing Natural Gas Development. Am J Public Health 103:1002–1010; doi:10.2105/AJPH.2012.301017.
- Xcel Energy. 2017a. Comanche Generating Station. Available: https://www.xcelenergy.com/energy_portfolio/electricity/power_plants/comanche [accessed 21 June 2018].
- Xcel Energy. 2017b. Resource Plan. Available: https://www.xcelenergy.com/company/rates_and_regulations/resource_plans [accessed 21 June 2018].

- Zanobetti A, Dominici F, Wang Y, Schwartz JD. 2014. A national case-crossover analysis of the short-term effect of PM2.5 on hospitalizations and mortality in subjects with diabetes and neurological disorders. Environ Health Glob Access Sci Source 13:38; doi:10.1186/1476-069X-13-38.
- Zanobetti A, Franklin M, Schwartz J. 2008. Fine particulate air pollution and its components in association with cause-specific emergency admissions in 26 US cities. Epidemiology 19: S315–S316.

Appendix A. Concentration-Response Functions

Table A1. Summary of pollutants and health outcomes included in the HIA, studies from which concentration-response coefficients were taken, and pooled concentration-response coefficients used in the health impact functions.

Pollutant	Outcome Name	CR	SE of CR	Source	Pooled CR	SE of Pooled CR
O ₃	Asthma ED visit	0.00306	0.00117	(Glad et al. 2012)	0.00288	0.00098
		0.00521	0.00091	(Ito et al. 2007)		
		0.00770	0.00284	(Mar and Koenig 2009)		
		0.01044	0.00436	(Mar and Koenig 2009)		
		0.00087	0.00053	(Peel et al. 2005)		
		0.00111	0.00028	(Sarnat et al. 2013)		
		0.00300	0.00100	(Wilson et al. 2005)		
		-0.00100	0.00200	(Wilson et al. 2005)		
	Respiratory hospitalization	0.00064	0.00040	(Katsouyanni et al. 2009)	0.00194	0.00110
Asthma sym		0.00178	0.00094	(Schwartz 1995)		
		0.00493	0.00177	(Schwartz 1995)		
	Asthma symptom day	0.00929	0.00387	(Mortimer et al. 2002)	0.00357	0.00229
		0.00097	0.00299	(O'Connor et al. 2008)		
		0.00222	0.00282	(Schildcrout et al. 2006)		
	Minor restricted activity day	0.00260	0.00078	(Ostro and Rothschild 1989)	0.00260	0.00078
	School loss day	0.00158	0.00499	(Chen 2000)	0.00511	0.00339
		0.00815	0.00463	(Gilliland et al. 2001 p. 200)		
	Non-accidental mortality	0.00052	0.00013	(Bell et al. 2004)	0.00095	0.00018
		0.00039	0.00013	(Bell et al. 2004)		
		0.00079	0.00013	(Ito et al. 2005)		
		0.00172	0.00035	(Ito et al. 2005)		
		0.00140	0.00027	(Moolgavkar et al. 1995)		
		0.00140	0.00038	(Moolgavkar et al. 1995)		
		0.00094	0.00030	(Kelsall et al. 1997)		
PM2.5	Acute bronchitis	0.02721	0.01710	(Dockery et al. 1996)	0.02721	0.01710

 Asthma ED visit	0.00516	0.00324	(Glad et al. 2012)	0.00473	0.00148
	0.00560	0.00210	(Mar et al. 2010)		
 	0.00296	0.00271	(Slaughter et al. 2005)		
CVD hospitalization	0.00080	0.00011	(Bell et al. 2008)	0.00111	0.00021
	0.00140	0.00034	(Moolgavkar 2000)		
	0.00158	0.00034	(Moolgavkar 2003)		
	0.00071	0.00013	(Peng RD et al. 2008)		
	0.00064	0.00026	(Peng et al. 2009)		
	0.00187	0.00028	(Zanobetti et al. 2008)		
 Respiratory hospitalization	0.00070	0.00004	(Kloog et al. 2012)	0.00130	0.00067
	0.00205	0.00044	(Zanobetti et al. 2008)		
 Asthma symptom day	0.01906	0.00983	(Mar et al. 2004)	0.08363	0.77244
	0.00099	0.00075	(Ostro et al. 2001)		
	0.01222	0.01385	(Mar et al. 2004)		
	0.00257	0.00134	(Ostro et al. 2001)		
	0.00194	0.00080	(Ostro et al. 2001)		
 Lower respiratory symptoms	0.01901	0.00600	(Schwartz and Neas 2000)	0.01901	0.00600
 Minor restricted activity day	0.00741	0.00070	(Ostro and Rothschild 1989)	0.00741	0.00070
 Work Loss Day	0.00460	0.00036	(Ostro 1987)	0.00460	0.00036
 All-cause mortality	0.00583	0.00096	(Krewski et al. 2009)	0.00583	0.00096
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Appendix B. Supplemental Tables

Table B1. Summary statistics for the change in population-weighted exposures to $PM_{2.5}$ (µg/m³) and ozone (ppb) across all ZCTAs in the study area for Health Benefits Scenario 1

Pollutant	Season	Metric	Mean (SD)	Min	Median	Max
PM _{2.5}	Winter	Seasonal mean	0.00 (0.01)	0.00	0.00	0.03
	Summer	Seasonal mean	0.00 (0.00)	0.00	0.00	0.03
	Winter	Daily mean	0.00 (0.01)	-0.02	0.00	0.22
	Summer	Daily mean	0.00 (0.01)	0.00	0.00	0.07
O ₃	Winter	Seasonal mean	-0.02 (0.03)	-0.16	0.00	0.00
	Summer	Seasonal mean	0.03 (0.03)	-0.16	0.03	0.09
	Winter	Daily mean	-0.02 (0.07)	-1.14	0.00	0.06
	Summer	Daily mean	0.03 (0.07)	-0.99	0.01	0.57
	Winter	Daily 1 hour max	0.01 (0.04)	-0.08	0.00	0.60
	Summer	Daily 1 hour max	0.24 (0.43)	-0.01	0.07	5.25
	Winter	Daily 8 hour max	0.00 (0.02)	-0.29	0.00	0.23
	Summer	Daily 8 hour max	0.10 (0.17)	-0.11	0.04	1.70

Abbreviations: $PM_{2.5}$: particulate matter with an aerodynamic diameter less than 2.5 μ m; O_3 : ozone; SD: standard deviation

Table B2. Summary statistics for the change in population-weighted exposures to $PM_{2.5}$ (µg/m³) and ozone (ppb) across all ZCTAs in the study area for Health Benefits Scenario 2

Pollutant	Season	Metric	Mean (SD)	Min	Median	Max
PM _{2.5}	Winter	Seasonal mean	0.00 (0.01)	0.00	0.00	0.03
	Summer	Seasonal mean	0.00 (0.00)	0.00	0.00	0.03
	Winter	Daily mean	0.00 (0.01)	-0.01	0.00	0.22
	Summer	Daily mean	0.00 (0.01)	0.00	0.00	0.07
О3	Winter	Seasonal mean	-0.02 (0.03)	-0.16	0.00	0.00
	Summer	Seasonal mean	0.03 (0.03)	-0.16	0.03	0.10
	Winter	Daily mean	-0.02 (0.07)	-1.13	0.00	0.06
	Summer	Daily mean	0.03 (0.07)	-0.99	0.01	0.57
	Winter	Daily 1 hour max	0.01 (0.04)	-0.08	0.00	0.60
	Summer	Daily 1 hour max	0.24 (0.43)	-0.01	0.08	5.25
	Winter	Daily 8 hour max	0.00 (0.02)	-0.29	0.00	0.23
	Summer	Daily 8 hour max	0.10 (0.17)	-0.11	0.04	1.70

Abbreviations: $PM_{2.5}$: particulate matter with an aerodynamic diameter less than 2.5 μ m; O_3 : ozone; SD: standard deviation

Table B3. Summary statistics for the change in population-weighted exposures to $PM_{2.5}$ (µg/m³) and ozone (ppb) across all ZCTAs in the study area for Health Benefits Scenario 3

Pollutant	Season	Metric	Mean (SD)	Min	Median	Max
PM _{2.5}	Winter	Seasonal mean	0.01 (0.02)	0.00	0.00	0.10
	Summer	Seasonal mean	0.04 (0.04)	0.00	0.02	0.35
	Winter	Daily mean	0.01 (0.02)	-0.04	0.00	0.40
	Summer	Daily mean	0.04 (0.07)	0.00	0.01	0.96
О3	Winter	Seasonal mean	-0.03 (0.06)	-0.42	-0.01	0.00
	Summer	Seasonal mean	-0.01 (0.13)	-1.02	0.03	0.05
	Winter	Daily mean	-0.03 (0.10)	-1.86	0.00	0.07
	Summer	Daily mean	-0.01 (0.19)	-3.04	0.01	0.64
	Winter	Daily 1 hour max	0.02 (0.05)	-0.31	0.00	0.72
	Summer	Daily 1 hour max	0.28 (0.43)	-0.89	0.12	4.40
	Winter	Daily 8 hour max	0.00 (0.03)	-0.57	0.00	0.34
	Summer	Daily 8 hour max	0.11 (0.17)	-0.50	0.05	1.95

Abbreviations: $PM_{2.5}$: particulate matter with an aerodynamic diameter less than 2.5 μ m; O_3 : ozone; SD: standard deviation

Appendix C. Supplemental Figures

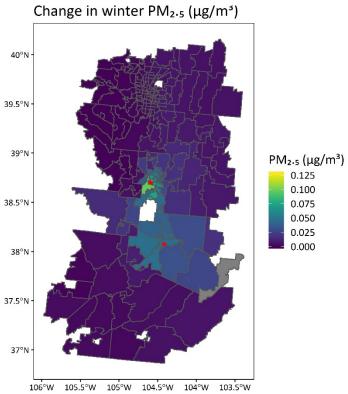


Figure C1. Changes in mean winter PM_{2.5} (μ g/m³) concentrations at the ZCTA level for Health Benefits Scenario 4

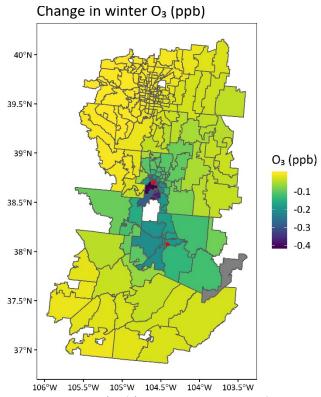


Figure C2. Changes in mean winter O_3 (ppb) concentrations at the ZCTA level for Health Benefits Scenario 4

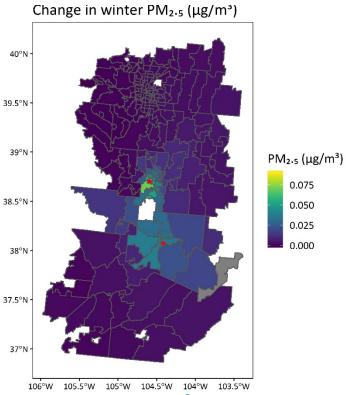


Figure C3. Changes in mean winter PM_{2.5} (μ g/m³) concentrations at the ZCTA level for Health Benefits Scenario 5

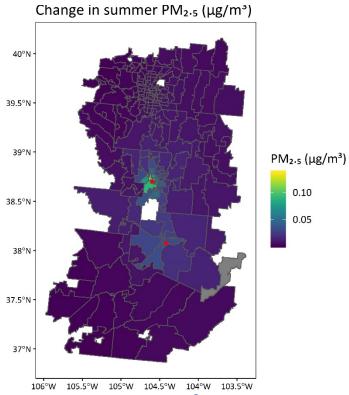


Figure C4. Changes in mean summer PM_{2.5} (μ g/m³) concentrations at the ZCTA level for Health Benefits Scenario 5

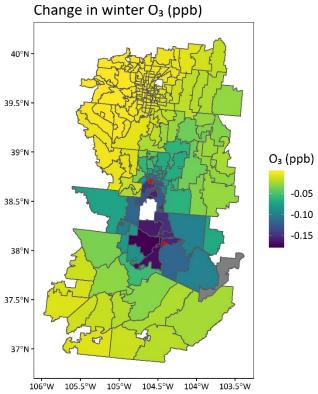


Figure C5. Changes in mean winter O_3 (ppb) concentrations at the ZCTA level for Health Benefits Scenario 5

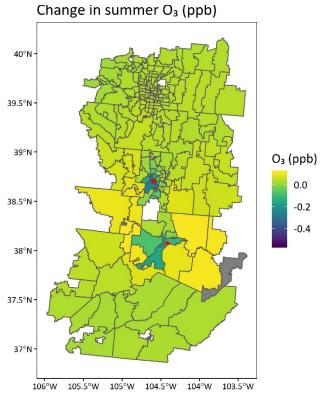


Figure C6. Changes in mean summer O_3 (ppb) concentrations at the ZCTA level for Health Benefits Scenario 5

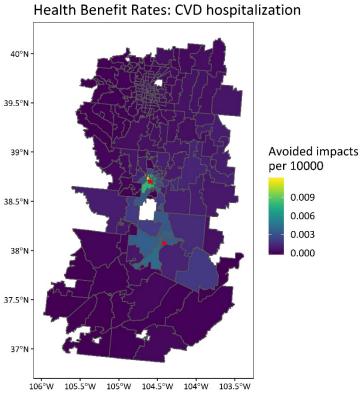


Figure C7. Maps of annual avoided cardiovascular disease rates (per 10,000 persons) at the ZCTA level due to reductions in $PM_{2.5}$ (Health Benefits Scenario 4)

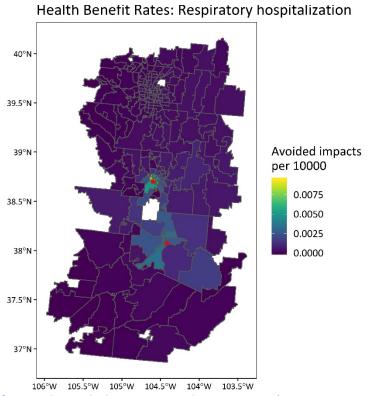


Figure C8. Map of annual avoided respiratory disease rates (per 10,000 persons) at the ZCTA level due to reductions in $PM_{2.5}$ (Health Benefits Scenario 4)

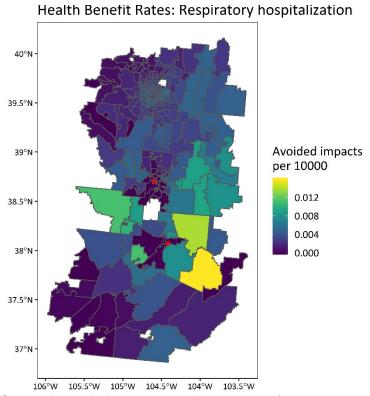


Figure C9. Map of annual avoided respiratory disease rates (per 10,000 persons) at the ZCTA level due to reductions in O₃ (Health Benefits Scenario 4)

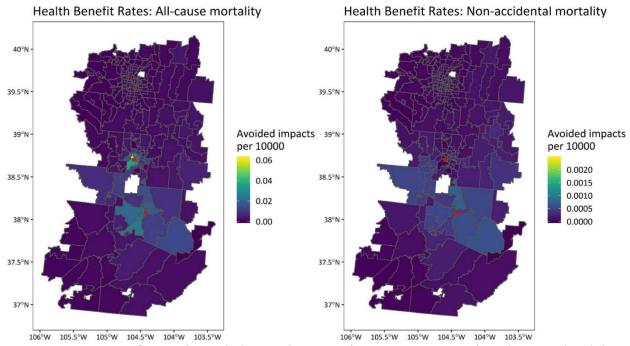


Figure C10. Maps of annual avoided mortality rates (per 10,000 persons) at the ZCTA level due to reductions in $PM_{2.5}$ (A) and O_3 (B) exposures for Health Benefits Scenario 5

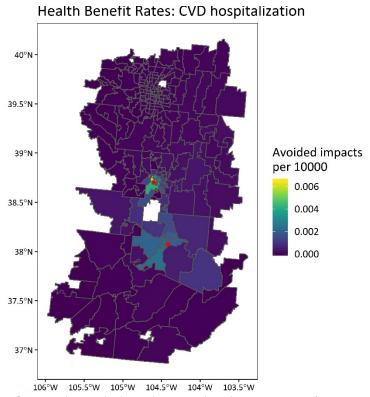


Figure C11. Maps of annual avoided cardiovascular disease rates (per 10,000 persons) at the ZCTA level due to reductions in $PM_{2.5}$ (Health Benefits Scenario 5)

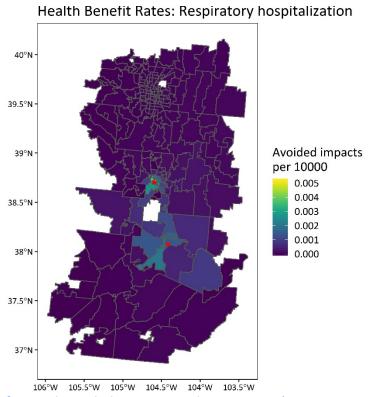


Figure C12. Map of annual avoided respiratory disease rates (per 10,000 persons) at the ZCTA level due to reductions in $PM_{2.5}$ (Health Benefits Scenario 5)

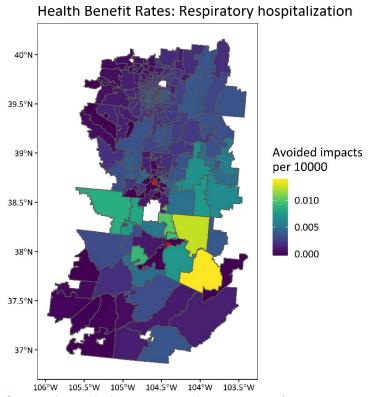


Figure C13. Map of annual avoided respiratory disease rates (per 10,000 persons) at the ZCTA level due to reductions in O_3 (Health Benefits Scenario 5)

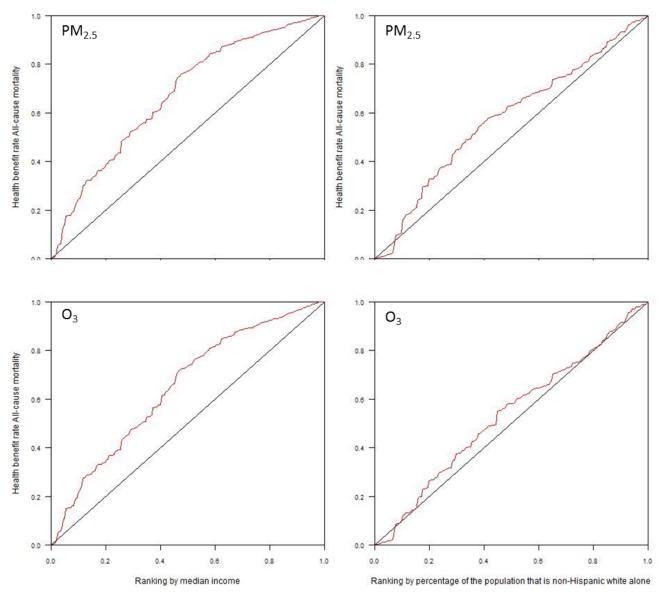


Figure C14. Concentration index curves for avoided mortality rates (per 10,000 persons) resulting from reduced $PM_{2.5}$ or O_3 exposures under Health Benefits Scenario 1 when ranking ZCTAs by median income and the percentage of the population that is non-Hispanic White alone

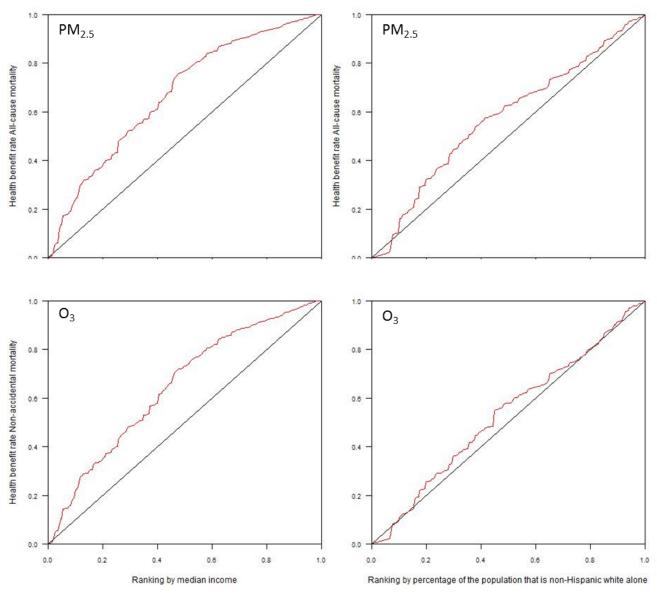


Figure C15. Concentration index curves for avoided mortality rates (per 10,000 persons) resulting from reduced $PM_{2.5}$ or O_3 exposures under Health Benefits Scenario 2 when ranking ZCTAs by median income and the percentage of the population that is non-Hispanic white alone

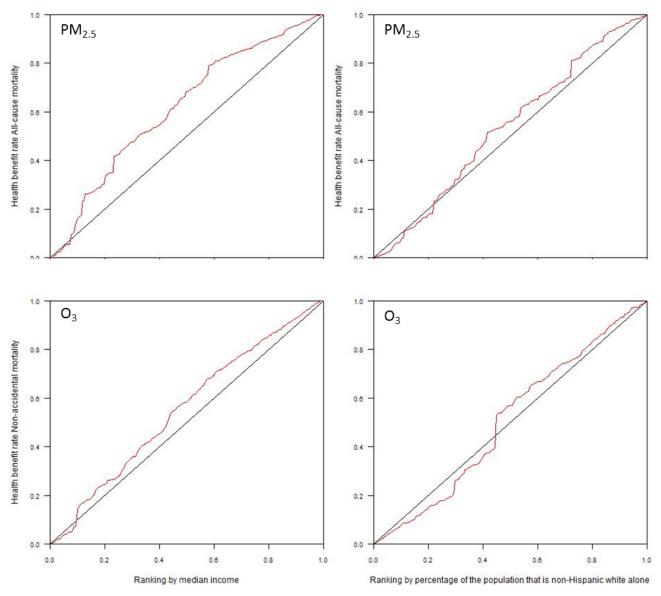


Figure C16. Concentration index curves for avoided mortality rates (per 10,000 persons) resulting from reduced $PM_{2.5}$ or O_3 exposures under Health Benefits Scenario 3 when ranking ZCTAs by median income and the percentage of the population that is non-Hispanic White alone.

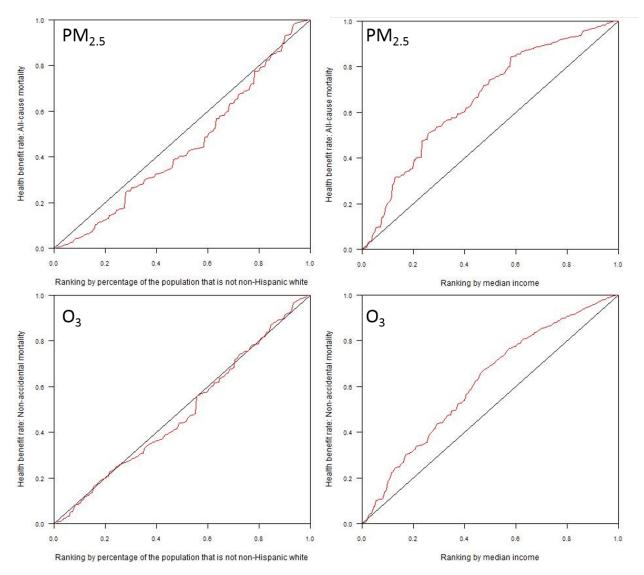


Figure C17. Concentration index curves for avoided mortality rates (per 10,000 persons) resulting from reduced $PM_{2.5}$ or O_3 exposures under Health Benefits Scenario 5 when ranking ZCTAs by median income and the percentage of the population that is non-Hispanic White alone.