

US power plant carbon standards and clean air and health co-benefits

Emissions Modeling

For each scenario, we used emissions output from ICF International's Integrated Planning Model (IPM) conducted by ICF International. IPM is a dynamic power sector production cost linear optimization model for North America. It incorporates many drivers of generation and power sector demands, including wholesale power, system reliability needs, environmental limitations, fuel selection, power transmission, capacity, and operational elements of generators on the power grid, to estimate generation and resulting emissions. By running IPM the least-cost means of meeting electric generation energy and capacity requirements are determined, while complying with the requirements specified in each of the policy scenarios.

A sensitivity analysis was conducted to test assumptions of IPM calculations within a range of uncertainty for: a) natural gas price, b) cost of end-use efficiency, and c) nuclear plant relicensing/retirement. The results suggest that generation mix, coal retirements, cost of electricity, and building of new generation capacity are all sensitive to varying levels to natural gas price and cost of demand-side energy efficiency.

Air Quality Modeling

We used CMAQ version 4.7.1 based on EPA's 2007/2020 modeling platform with CB05 gas chemistry, AE5 aerosol chemistry and multi-pollutant options for mercury chemistry. We held

meteorology constant using Year 2007 meteorology from the Weather Research and Forecasting (WRF) model version 3.1.

Health Co-benefits Modeling

We used the spatially explicit air quality results from CMAQ as input to BenMAP CE v1.0.8{BenMAP:um}. BenMAP CE is a Geographic Information System (GIS)-based software tool designed for calculating the health co-benefits of air quality management scenarios. BenMAP CE accepts two air quality grids as inputs, representing air pollutant concentrations under a policy scenario, and a reference case. It then estimates the benefits of a policy as the difference between the two. BenMAP CE performs the health impact calculations for each scenario as below:

$$\text{Change in health impact} = \text{Exposed population} \times \text{baseline incidence or prevalence of health endpoint} \times \text{concentration-response function} \times \text{change in concentration of air pollutant}$$

Concentration-response Functions Used in BenMAP CE

Concentration-response functions for changes in PM_{2.5} concentrations used in BenMAP CE

Premature deaths avoided (adult mortality): We used a concentration-response function relating long-term exposure to PM_{2.5} to all-cause mortality rate in adults 25 years of age or older¹. This concentration-response function has a central estimate of a 1% increase in mortality rate per $\mu\text{g}/\text{m}^3$ increase in annual average PM_{2.5} concentrations. We determined uncertainty bounds for this function that approximately encompass the range of estimates of estimates from two major U.S. cohorts, using a standard error of 0.4%.

Heart attacks avoided (acute non-fatal myocardial infarction): We used a concentration-response function derived from Mustafic et al.², a meta-analysis of 34 studies examining the relationship between short-term exposure to PM_{2.5} in adults over 18 years of age and risk of non-fatal myocardial infarction (heart attack).

Other cardiovascular hospital admissions avoided (excluding myocardial infarctions): We selected two large multi-city studies as the foundation for these estimates^{3,4}. Both studies related short-term PM_{2.5} exposure and hospital admissions for cardiovascular causes other than myocardial infarctions (heart attacks) in adults 65 years of age and over. We pooled the estimates from these two studies using inverse variance weighting, which places a greater weight on more statistically precise estimates. However, by using these two studies we may underestimate the risk of these outcomes by not including increased risk from long-term exposure⁵. We focus on the impact of short-term exposures to be consistent with current U.S. EPA regulatory impact analysis methods.

Respiratory hospital admissions avoided: We selected two large multi-city studies as the foundation for these estimates^{3,4}. Both studies related short-term PM_{2.5} exposure and hospital admissions for respiratory causes in adults 65 years of age and over. As above, we pooled the estimates from these two studies using inverse variance weighting. As above, we possibly underestimate the overall effect by not including results for long-term exposure⁵.

Concentration-response functions for change in ozone concentrations used in BenMAP CE

Premature deaths avoided (adult mortality): We used a concentration-response function derived from the Jerrett et al.⁶ study of the American Cancer Society cohort for the ozone season average 1-hour maximum and respiratory mortality risk in adults 30 years of age and over. We note that

several recent Medicare cohort studies have reported associations between summer ozone exposure and cardiovascular deaths, so this may be an underestimate of the true benefits of ozone reduction.

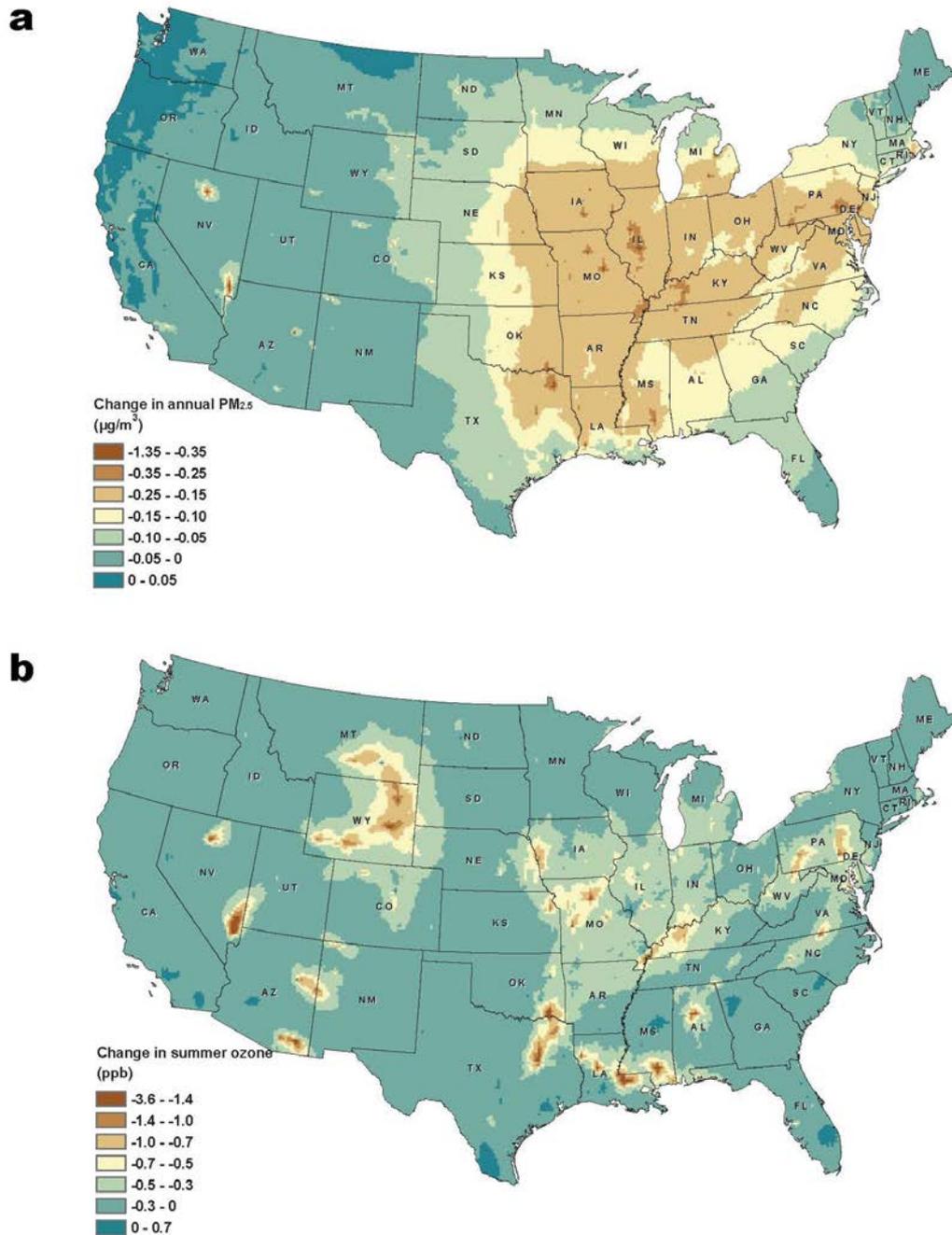
Respiratory hospital admissions: We used a concentration-response function derived from Ji et al.⁷, a meta-analysis of 96 studies relating short-term ozone exposure and increased risk of hospital admissions for respiratory causes in adults 65 years of age and over.

References

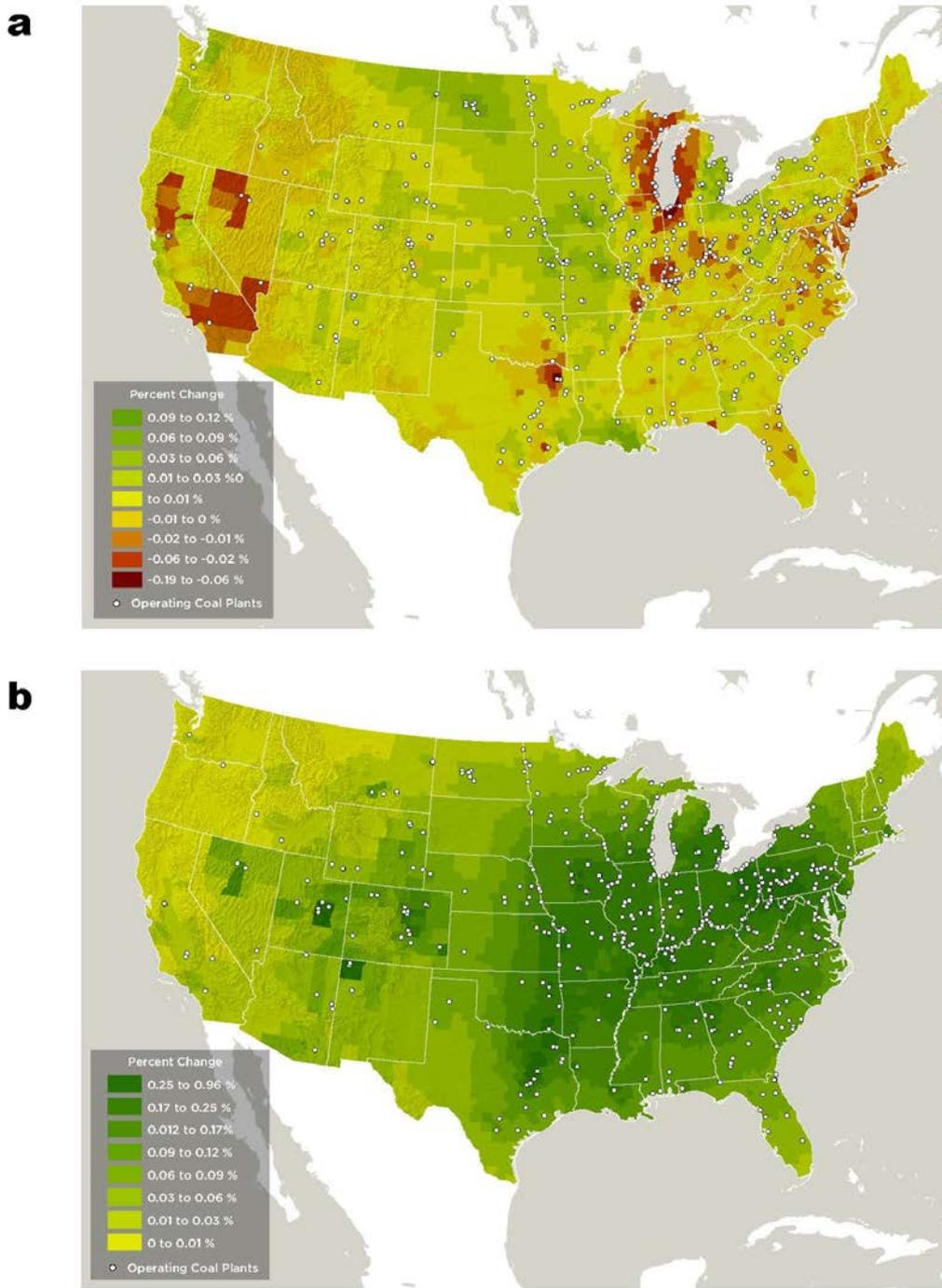
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Supplementary Table 1. Concentration-response functions from the literature used to estimate health co-benefits from changes in air pollutant concentrations for the three policy scenarios.

Study	Health Outcome	Pollutant	Metric	Response (% increase or decrease in rate)	Standard Error (% increase or decrease in rate)
Roman et al. ¹	Premature death (all causes)	PM _{2.5}	Annual average concentration (µg/m ³)	1.0	0.4
Levy et al. ³ , Zanobetti et al. ⁴ , pooled	Respiratory hospitalizations	PM _{2.5}	Daily average concentration (µg/m ³)	0.11	0.027
Levy et al. ³ , Zanobetti et al. ⁴ , pooled	Cardiovascular hospitalizations	PM _{2.5}	Daily average concentration (µg/m ³)	0.094	0.015
Mustafic et al. ²	Heart attack (acute non-fatal myocardial infarction)	PM _{2.5}	Daily average concentration (µg/m ³)	0.25	0.0536
Jerrett et al. ⁶	Premature death (respiratory causes)	Ozone	April – Sept. average of the 1-hour maximum (ppb)	0.39	0.13
Ji et al. ⁷	Respiratory Hospitalizations	Ozone	Annual average of the 8-hour maximum (ppb)	0.16	0.052



Supplementary Figure 1. Maps for the continental U.S. of difference in annual average concentrations of fine particulate matter ($PM_{2.5}$) in micrograms per cubic meter (a) and the difference in annual average concentrations of peak summertime ozone in parts per billion (b) for Scenario 3 less the reference scenario in 2020.



Supplementary Figure 2. Percent change in premature deaths avoided by county from the 2020 reference case for scenarios 1 (a) and 2 (b).