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Fine Particulate Air Pollution from Electricity Generation in the US: Health Impacts by Race, Income, and Geography

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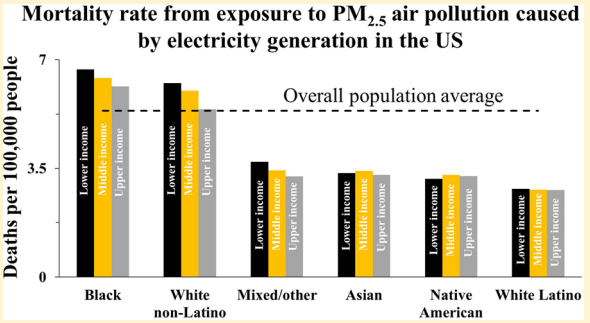
Supporting Information

ABSTRACT: Electricity generation is a large contributor to fine particulate matter ($PM_{2.5}$) air pollution. However, the demographic distribution of the resulting exposure is largely unknown. We estimate exposures to and health impacts of $PM_{2.5}$ from electricity generation in the US, for each of the seven Regional Transmission Organizations (RTOs), for each US state, by income and by race. We find that average exposures are the highest for blacks, followed by non-Latino whites. Exposures for remaining groups (e.g., Asians, Native Americans, Latinos) are somewhat lower. Disparities by race/ethnicity are observed for each income category, indicating that the racial/ethnic differences hold even after accounting for differences in income. Levels of disparity differ by state and RTO. Exposures are higher for lower-income than for higher-income, but disparities are larger by race than by income. Geographically, we observe large differences between where electricity is generated and where people experience the resulting $PM_{2.5}$ health consequences; some states are net exporters of health impacts, other are net importers. For 36 US states, most of the health impacts are attributable to emissions in other states. Most of the total impacts are attributable to coal rather than other fuels.



1. INTRODUCTION

Fine particulate matter ($PM_{2.5}$) is the largest environmental health risk in the United States (US) and globally.[1,2] $PM_{2.5}$ is associated with increased mortality rates from, e.g., cardiovascular disease (ischemic heart disease and stroke), chronic obstructive pulmonary disease, and lung cancer.[3−5]

Fuel combustion emits $PM_{2.5}$ directly ("primary $PM_{2.5}$") as well as sulfur dioxide (SO_2) and oxides of nitrogen (NO_x), which can react with ammonia (NH_3) in the atmosphere to form $PM_{2.5}$ ("secondary $PM_{2.5}$").[6] The US Environmental Protection Agency (US EPA) estimates that in 2014, electricity generating units (EGUs) contributed 67% of SO_2, 13% of NO_x, and 3% of primary $PM_{2.5}$ emissions nationwide.[7] In 2014, coal-fired EGUs generated ∼39% of the electricity in the US and contributed to 97%, 86%, and 81%, respectively, of SO_2, NO_x, and $PM_{2.5}$ total electricity emissions.[7] Although the health damages associated with these emissions continue to be important, EGU emissions have declined in recent decades[7,8] owing to environmental regulations[9] and a transition from coal to natural gas driven largely by market prices.

Existing estimates of annual $PM_{2.5}$-related mortality from EGUs in the US include the following: (i) for year 2005: 52 000 (Caiazzo et al. 2013),[10] 41 500 (Dedoussi et al. 2014),[11] 19 000 (Penn et al. 2017),[12] 38 000 (Fann et al. 2013);[13] (ii) for year 2010: 17 050 (Lelieveld et al. 2015);[14] (iii) for year 2014: 10 400 (Tessum et al. 2019);[15] and (iv) for year 2016 projected emissions: 17 000 (Fann et al. 2013).[13] Levy et al. (2009)[16] modeled the monetized damages associated with 407 coal-fired power plants in the United States. Buonocore et al. (2014)[17] estimated monetized health impacts of $PM_{2.5}$ from individual power plants and normalized to "per-ton emitted" using the Community Multiscale Air Quality (CMAQ) Model. Penn et al. (2017)[12] also quantified impacts from EGUs by state, finding 21 000 premature mortalities per year from EGU emissions ($PM_{2.5}$ and ozone [O_3]). Mortality estimates vary among studies owing to differences in methods, models, concentration−response functions, and years considered (total EGU emissions are decreasing over time).

The consideration of how exposure to air pollution differs by the demographic group is relevant to environmental justice (EJ).[18] Several studies have estimated health-impact disparities for air pollution from various source sectors,[19−27] but few studies have investigated EJ aspects of electricity generation in the US. Studying EGUs in the US, Levy et al. (2007)[28] quantified health benefits and the change in the spatial inequality of health risk for potential EGU pollution control strategies. They report 17 000−21 000 fewer premature deaths per year for hypothetical power-plant control scenarios in the

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

US that aim to determine optimal control strategies. Martenies et al. (2017),[24] studying $PM_{2.5}$ health disparities in Detroit, Michigan, reported disproportionate burdens to Hispanics/Latinos owing to industrial emissions and to low-income populations owing to traffic emissions. Tessum et al. (2019) reported that although minorities are exposed to air pollution, they consume less and so, therefore, are less "responsible" for the generation of those emissions.[15] We are unaware of any prior national-scale investigation of EJ aspects of total $PM_{2.5}$ from electricity generation.

2. MATERIALS AND METHODS

We estimate $PM_{2.5}$-related health impacts from fossil-fuel-fired EGUs at the national scale, for each Regional Transmission Organizations (RTO), and, in terms of emissions and impacts (i.e., as a source vs receptor for pollution), for each US state. We use the year 2014 as our reference year. We characterize impacts disaggregated by race and income.

US Regional Transmission Organizations (RTOs) are electricity power markets responsible for dispatching more than 60% of the net electricity generation in the US (eGRID 2014 and 2016).[29] The seven US RTOs are Midcontinent Independent System Operator (MISO), California Independent System Operator (CAISO), Electric Reliability Council of Texas (ERCOT), Southwest Power Pool (SPP), Pennsylvania−New Jersey−Maryland (PJM) Interconnection, New York Independent System Operator (NYISO), and New England Independent System Operator (NEISO). Coal intensity is the greatest for MISO and SPP (>50% of generation comes from coal), followed by ERCOT and PJM (32 and 43%, respectively); the remaining RTOs (CAISO, NYISO, NEISO) are low coal (<5%) (Figure S1; data from eGRID 2014).[29] RTO demographics (Table S1) also differ by region; CAISO and ERCOT have >50% non-white population. We use RTOs as one of our units of analysis because they represent important geographic regions for electricity generation; they generally carry out electricity dispatch, which strongly impacts fuel use and emissions; and, they typically operate and make decisions independently.[30]

Multiple air-quality models are used in regulatory and research communities to link emissions with ambient concentrations, each with strengths and weaknesses.[17,31−53] Chemical Transport Models (CTMs) represent a state-of-the-science understanding of atmospheric chemistry and physics; they provide the most robust estimates available (i.e., the most robust representation of chemistry and physics) when time and computational constraints are not limiting.[49−53] CTMs are time- and resource-intensive. Reduced-complexity models (RCMs) are a less-intensive alternative.[17,31−48] RCMs are potentially less accurate than CTMs, but their reduced complexity allows for a far greater number of runs, thereby opening the door to sensitivity analyses, Monte Carlo approaches, longer simulation duration, and new understandings of source−receptor relationships. The RCM employed here, the Intervention Model for Air Pollution (InMAP),[37] employs smaller-sized grid cells than in conventional CTMs, thereby opening the door to the fine-scaled analysis generally thought to be important for EJ questions. Analyses carried out here would not be feasible using a conventional CTM, with current computational capacity. Other studies have demonstrated that InMAP and other RCMs can answer questions that could not be modeled using conventional CTMs.[28,54−58]

Details of InMAP, including model design, operation, and validation, are available elsewhere.[37,59] Briefly, InMAP is an Eulerian grid model that predicts the change in annual-average $PM_{2.5}$ concentration attributable to a change in annual emissions, based on steady-state solutions to equations representing pollution emission, transport, transformation, and removal. InMAP's representation of chemistry and meteorology is simplified relative to a conventional CTM, but it incorporates spatially varying parameters (e.g., rate constants) that are obtained from a CTM simulation. InMAP reflects the spatially varying rates of formation of $PM_{2.5}$, based on the (spatially varying) chemistry of the atmosphere (i.e., based on a CTM and a baseline [all-sources] emission inventory, here, the WRF-Chem model coupled with the National Emission Inventory [NEI]). Model runs carried out here focus only on emissions, concentrations, and health impacts from EGUs. The grid-cell size in InMAP varies from 1 km × 1 km (typically in urban areas) to 48 km × 48 km (typically in rural areas), depending on the gradient in the population density and pollutant concentrations. As configured here, InMAP also incorporates information about population, demographics, and concentration−response functions; we, therefore, use it to estimate concentrations and health impacts from EGU-attributable $PM_{2.5}$. Specifically, InMAP output for each grid cell includes annual-average $PM_{2.5}$ concentration ($\mu g/m^3$), the number of premature deaths by race and household income category, population size, and baseline mortality.

For research questions considered here, InMAP requires three main inputs:

(1) Annual emissions of VOC, NO_x, NH_3, SO_2, and primary $PM_{2.5}$ for each EGU; we employ US EPA's 2014 National Emission Inventory (NEI).[7] For point sources, such as the EGUs, the NEI provides stack attributes including height, diameter, temperature, and exit velocity.

(2) Census data on population (by block group) and household income (tract) for 2014 from the 2014 American Community Survey (ACS).[60] Demographic groups given in the ACS are self-reported white non-Latino, white Latino, black non-Latino, black Latino, Asian Latino, Asian non-Latino, Native American non-Latino, Native American Latino, mixed/other non-Latino, and mixed/other Latino. In our summaries, "non-white" refers to all categories except white non-Latino; "black", "Asian", and "Native American" include both Latino and non-Latino ethnicities. "Latino" refers to white Latinos, black Latinos, Asian Latinos, Native American Latinos, and mixed/other Latinos (according to the 2014 ACS, Latinos are 65% white, 31% mixed/other, 2% black, 1% Native American, and 0.3% Asian). Census income data are divided into 16 income groups for each of six racial categories: white non-Latino, white Latino, black, Asian, Native American, and mixed/other race groups. Therefore, following the Census data, our summaries present income-based results for "white Latino" category and not all "Latinos". We calculate the percentage of households in each income category in a given census tract and apply those percentages to population counts at the block group level (matching census tracts to the block groups that they contain) to calculate an estimate of people in each income group.

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

(3) Baseline all-cause mortality data are for the year 2014 from the National Center for Health Statistics (NCHS) Office of Analysis and Epidemiology (OAE) at the Centers for Disease Control and Prevention (CDC).[61] Race-specific health impacts are calculated using all-cause mortality rates for the entire population of all age groups. Our analysis does not differentiate impacts by age group. Literature-derived estimates of mortality rates and the concentration−response (C−R) function are not sufficiently robust to allow race-ethnicity-specific values for those two parameters. Consistent with prior research[11−17,27] and in keeping with EPA norms,[62−64] we assume that all $PM_{2.5}$ particles are equally toxic.

We employ an expression derived from Krewski et al. (2009)[65] for our $PM_{2.5}$ concentration−response function, which is used to estimate $PM_{2.5}$-related health impacts (Cox proportional hazards model):

$$\text{number of premature deaths} = (e^{(\text{PM}_{2.5}\text{ linear coefficient}\times[\text{PM}_{2.5}])} - 1) \times P \times \frac{\text{all-cause mortality rate}}{100\,000}$$

Here, $PM_{2.5}$ linear coefficient is assumed to be $\ln(1.078)/10 = 0.00751$, i.e., a 7.8% increase in the number of premature deaths for every 10 $\mu g/m^3$ increase in the concentration of $PM_{2.5}$. $[PM_{2.5}]$ is the concentration of $PM_{2.5}$, P is the total population. Mortality is then estimated as

$$\text{deaths per 100 000 people} = \frac{\text{number of premature deaths}}{\text{population}} \times 100\,000$$

We estimate premature mortality at the national scale, for each RTO, for each US state, and for each race/ethnicity and race/ethnicity−income group. We run InMAP for all EGUs to estimate impacts at the national scale. We also run InMAP separately for coal-fired EGUs, natural gas-fired EGUs, and all other fuel-type EGUs to estimate impacts by fuel-type at the national scale. Finally, we apportion health impacts by constituents of $PM_{2.5}$ at the national scale: primary $PM_{2.5}$, particulate SO_4 (pSO_4), particulate NO_3 (pNO_3), particulate NH_4 (pNH_4), and secondary organic aerosol (SOA; caused by VOC emissions). We run InMAP separately for each RTO. For each of 49 geographies in the contiguous US (i.e., the 48 states plus the District of Columbia; for brevity, we refer here to these 49 geographies as "states"), we use the InMAP Source−Receptor Matrix (ISRM) to estimate impacts from EGUs within and across state boundaries.[66] Total number of premature deaths, by race and income, are aggregated by RTO and by state. In some cases, analyses are constrained by available Census data. For example, Census data on ethnicity are available for whites, blacks, Asian, Native Americans, and mixed/other groups, i.e., each racial group is differentiated by ethnicity (Latino/non-Latino). In contrast, for combined race and income data, demographic data is differentiated by ethnicity for whites only, not for other races; results by income categories are available for six racial/ethnic categories (white non-Latino, white Latino, black, Asian, Native American, and mixed/other race groups), reflecting the race−income data available from the US Census. If robust, more-detailed demographic informations were publicly available, it would be straightforward to update our estimates.

Population-weighted concentrations are calculated as follows:

$$\text{population weighted average PM}_{2.5}\text{ concentration} = \frac{\sum_{i=1}^{n}(P_i \times [\text{PM}_{2.5}]_i)}{\sum_{i=1}^{n} P_i}$$

Here, P_i is the number of people of a specific demographic group in grid cell i, $[PM_{2.5}]_i$ is the concentration in grid cell i, and n is the total number of grid cells.

Damages are presented in terms of three types of metrics:

(a) We compute total damages in terms of deaths, by aggregating deaths (from total $PM_{2.5}$) in each grid cell for each state, RTO, and nationally and each race/ethnicity and race/ethnicity−income group in each spatial scale. For mortality rate in terms of deaths per 100 000 people, health impacts are normalized to the respective population in each geographic scale, race/ethnicity and race/ethnicity−income groups.

(b) We estimate "risk gap" for state-level mortality estimates to quantify the difference in environmental health risk (deaths per 100 000 people per year) between the most- and least-exposed group in a state. The advantage of this metric is that it does not force a preselection of specific groups to compare (e.g., blacks relative to whites, Asians relative to population average, lowest income relative to highest income) but instead is flexible across geographies to which groups are most and least exposed in that state.

(c) We compute the deaths per unit of electricity service provided (deaths per TWh) for the state, RTO, and nationally. This metric facilitates nationwide comparisons of the impacts of EGUs.

3. RESULTS

3.1. Total Premature Deaths and Deaths Per Unit of Electricity: Nationally, Regionally, and by State.

3.1.1. Total National Damages. For 2014, we estimate ~16 400 $PM_{2.5}$-related premature deaths attributable to EGUs, for an average of ~4 deaths/TWh electricity generated, corresponding to a total national population-weighted estimate of ~0.82 $\mu g/m^3$ of EGU-$PM_{2.5}$. Most of the deaths (~14 200 deaths or ~86%; see Table 1) are attributable to EGUs that are in an RTO (the rest are caused by EGUs that are not in an RTO).

Deaths per unit energy generated (Table 1) vary substantially among regional grids, by up to a factor of ~30 (MISO vs CAISO), with higher values in coal-heavy grids (MISO, SPP). Total attributable premature deaths vary even more by state (Table S2) from ~1850 deaths/year (Pennsylvania) to ~1 death/year (Montana). Among states, deaths per unit electricity generation (units: deaths/TWh) for each state (Table S3) vary from 0.02 (Vermont) to 14 (Indiana).

Of the ~16 400 premature deaths from EGUs nationwide, we estimate that the vast majority (~15 200 or ~93%) is from coal EGUs, ~800 (5%) from natural gas EGUs, and ~460 from other fuel EGUs (Figure S2). Similar patterns hold in most states: coal-fired EGUs are the largest contributor to total EGU-$PM_{2.5}$ deaths (Table S4). Nationally, most EGU-$PM_{2.5}$

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

Table 1. Estimated Deaths Per Unit Electricity Generation by RTO

RTO	annual net generation (TWh)[a]	total deaths attributable to RTO's emissions: total deaths	total deaths attributable to RTO's emissions: deaths per TWh	percent of generation by fuel[a]: coal (%)	natural gas (%)	oil, biomass, and other fossil fuels (%)
CAISO	170	45	0.3	0.5	59	4
ERCOT	365	1788	4.9	32	46	0.7
MISO	691	5649	8.2	56	19	4
NEISO	110	48	0.4	5	43	10
NYISO	140	162	1.2	3	42	4
PJM	809	4868	6.0	43	17	2
SPP	238	1599	6.7	59	19	0.8

[a]From year 2014 in eGRID.[29]

impacts (~73%) are from pSO_4, which is dominated by sulfur emissions from coal EGUs; contributions by other species (Figure S3) are 15% (pNO_3), 11% (primary $PM_{2.5}$), 1% (pNH_4), and ~0% (SOA).

3.2. Differences in Damages by Demographic Group. *3.2.1. Race.* We find that year 2014 mortality rates from EGU-$PM_{2.5}$ are largest for black people, second largest for white non-Latino people, and lower-than-average for Asian, Native American, and Latino people (Figure 1). Differences by race vary by RTO (Figure S4) and species (Figure S5).

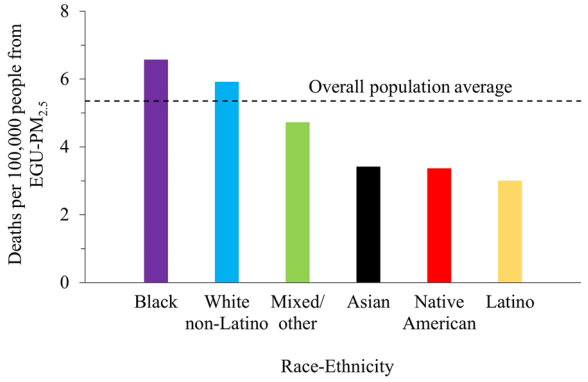

Figure 1. Deaths per 100 000 people attributable to $PM_{2.5}$ from electricity generation in the US in 2014.

The overall average mortality rates from EGU-$PM_{2.5}$ are 5.3 for all people, 6.6 for blacks, 5.9 for white non-Latinos, and 3.6 averaged across the remaining groups (Figure 1). We assessed the spatial distribution by race-ethnicity of mortality rate density (i.e., per km^2) from EGU-$PM_{2.5}$ (Figure S6A–C) and to identify where each group is most impacted. For example, for Native Americans, the density of deaths is greater in western Oklahoma than in most locations. Subdividing by fuel-type (Figure S7) reveals that blacks are the most-exposed group for all three fuel categories. For natural gas, exposures are higher than average for blacks, Asians, and Latinos and lower than average for white non-Latinos. For coal emissions, relative exposures are similar to Figure 1 except that Native Americans are slightly more exposed to coal emissions than Asians are.

3.2.2. Income and Race. Differences by race are observed across all income categories (Figure 2). Thus, differences by

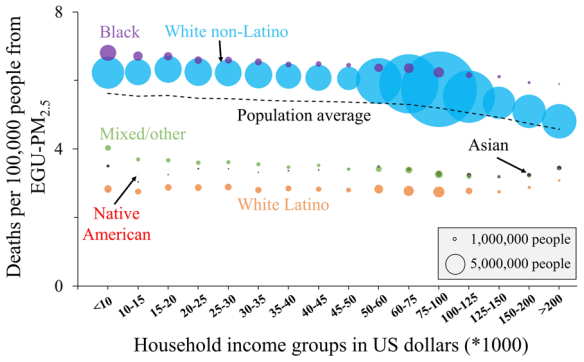

Figure 2. Deaths per 100 000 people by income group and race/ethnicity. Icon area is proportional to population size.

race/ethnicity (Figure 1) are not "merely" income differences; race/ethnicity differences are present even after accounting for differences in income (Figure 2).

On average, exposures are higher for lower-income households than for higher-income households. Considering Figure 2 (deaths per 100 000 people attributable to EGU-$PM_{2.5}$), the difference between most- and least-exposed income group is ~1.0 for the overall population; the same difference is 1.5 for white non-Latino, 0.9 [black], 0.3 [white Latino], 0.5 [Asian], 0.9 [Native American], and 0.8 [mixed/other]. The difference between most- and least-exposed race is 3.6 for the overall population, 4.0 for the lowest-income population, and 2.8 for the highest-income population. Thus, differences by race are larger than differences by income.

Based on results in Figure 2, if we calculate the risks by race, but making the adjustment that all race/ethnicity groups have an income distribution equal to the national average distribution, then mortality rates from EGU-$PM_{2.5}$ (deaths per 100 000 people) would be 5.3 for all people, 6.4 for blacks, 5.9 for white non-Latinos, and 3.2 averaged across the remaining groups. Here too, analyses reveal that exposure differences by race are observed even after accounting for income differences.

Race–income results differ substantially by RTO (Figure S8), for example, exposures are higher for white non-Latino income groups than for black income groups for CAISO, MISO, NEISO, and SPP but not ERCOT, NYISO, and PJM.

We estimate exposures by household income group, for each state, from total EGU-$PM_{2.5}$; Figure 3 shows the most-exposed income category in each state and the risk gap (premature deaths per 100 000 people) between most- and least-exposed household income group. The "$10 000–$15 000 per year" household income category is the most-exposed category for 19 out of 49 states (risk gap varies between 0.06 and 3.6 for these 19 states). Overall, low-income categories are most exposed in a majority of the states [38 states], followed by middle income [5 states] and upper income [6 states]. The gap between most- and least-exposed household income category is sizeable (>2 premature deaths per 100 000 people) in only three states (Maryland [3.5], Virginia [3.6], Indiana [2.9]; total population = 21 million).

3.3. Health Damages by State. Our results estimate EGU-$PM_{2.5}$ health impacts, with each state as a source and a receptor of pollution (Figure 4). The maps reveal geographic differences between where EGU emissions are produced and where exposures and health impacts are experienced. For example, Texas experienced an estimated total of ~1360 EGU-

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Environ. Sci. Technol. XXXX, XXX, XXX–XXX

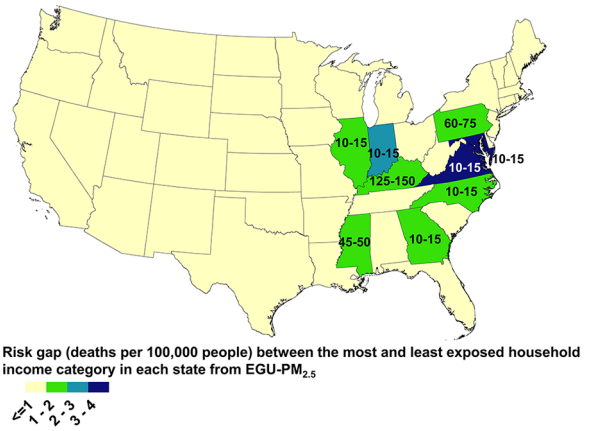

Figure 3. Most-exposed household income group in thousand US dollars (for overall population) and risk gap (units: deaths per 100 000 people attributable to EGU-$PM_{2.5}$ from all EGUs in the US) between the most- and least-exposed household income group in each US state. The income group that is the most exposed is shown for states where the gap in the mortality rate is greater than 1 death per 100 000 people. The remaining states are unlabeled because the gap between most- and least-exposed income group is small (less than 1 per 100 000 people). A version of the map displaying labels for all states is shown in Figure S9. Risk gap is shown by color gradation.

$PM_{2.5}$ premature deaths in 2014 (Figure 4A), most of which (~1160, or >80%) are attributable to EGU emissions occurring inside Texas (Figure 4B); the remainder (~200 deaths/year in Texas) were attributable to $PM_{2.5}$ in Texas caused by EGU emissions outside of Texas. EGU emissions in Texas caused an additional ~524 deaths/year in states other than Texas (Figure 4C). Thus, Texas is a net exporter of EGU-$PM_{2.5}$ deaths: the number of premature deaths per year caused by Texas EGU emissions (~1684; the sum of ~1160 in-state plus ~524 out-of-state) exceeds the number of EGU-$PM_{2.5}$ premature deaths in Texas (~1360) by ~325 (Figure 4D). For some states (e.g., Arizona), damages from their EGU emissions are large in many downwind states; for other states (e.g., Washington), damages are more local (Figure S10A). As expected, many of the net importing states are on the East Coast (Figure 4D). A bar chart of Figure 4 (Figure S11) reveals the comparison between health impacts among different states.

States with the largest EGU-$PM_{2.5}$ mortality are Pennsylvania, Texas, Ohio, New York, Indiana, Virginia, Maryland, Kentucky, North Carolina, New Jersey, Illinois, and Florida; states with the smallest values are New Hampshire, South Dakota, Nevada, New Mexico, Maine, Arizona, Utah, North Dakota, Vermont, Wyoming, Oregon, Idaho, and Montana.

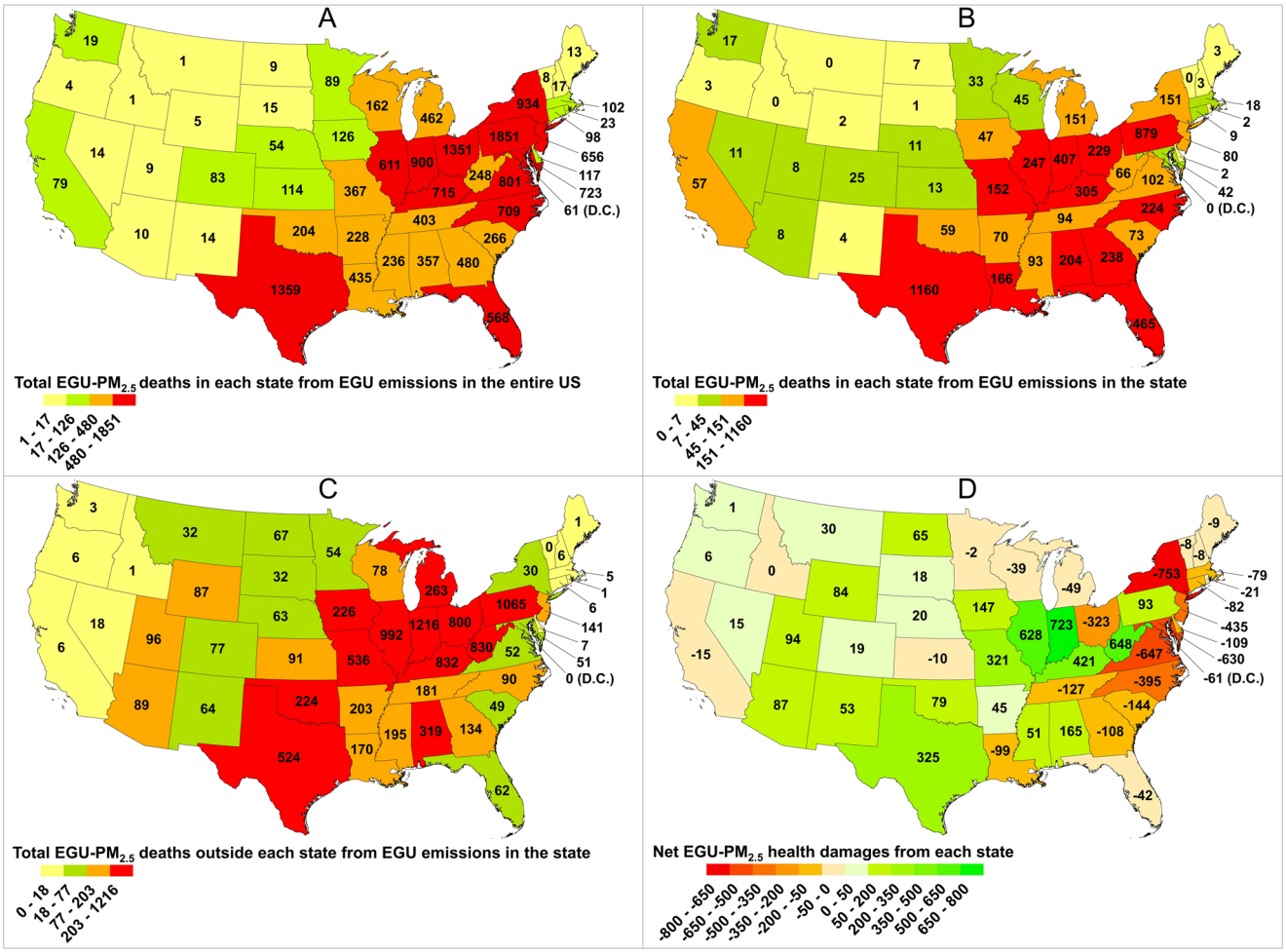

Figure 4. Deaths from EGU-$PM_{2.5}$ by state. (A) Total deaths in each state from EGUs throughout the US, (B) total deaths in-state from EGUs in that state, (C) total deaths out-of-state from EGUs in that state, (D) net imports (negative values) or exports (positive values) of deaths. Values in D are calculated as B + C − A. Range limits for color bars in A, B, and C are by quantiles.

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

The states with the largest import−export mortality imbalance are New York, Virginia, Maryland, New Jersey, North Carolina, and Ohio, which are net importers, i.e., the impacts from other states exceed impacts onto other states; Missouri, Texas, Kentucky, Illinois, West Virginia, and Indiana are the largest net exporters (i.e., the impacts to other states exceed impacts from other states). Among the largest net importing states, New York, Maryland, and New Jersey import the largest share of out-of-state impacts from Pennsylvania [29%, 36%, and 49%, respectively], North Carolina's largest share of out-of-state impacts comes from Kentucky [16%], Ohio from Indiana [37%], and Virginia from West Virginia [20%]. A similar number of states are net importers versus net exporters of EGU-$PM_{2.5}$ mortality (24 states and 24 states, respectively); importers (exporters) are more likely to be found in the Eastern (Western) portion of the US.

Our results reflect that EGU emissions travel great distances. For example, when considering states as receptors, in 39 states, a majority (>50%) of EGU-$PM_{2.5}$ mortality is attributable to EGU emissions in other states (Figure S12). When considering states as sources, in 35 states, a majority (>50%) of the state's EGU-$PM_{2.5}$ mortality impacts occurs outside of that state (Figure S10B).

3.4 Variations by Geography and Race. *3.4.1. Regional Transmission Organizations.* The most-impacted race/ethnicity varies by RTO (Figure S13): white non-Latino for NEISO; Asian [CAISO and NYISO]; white Latino [ERCOT]; black [PJM and MISO]; and Native American [SPP]. MISO and SPP (the two RTOs with >50% of generation from coal; Table 1) are the only RTOs where more than 50% of total premature deaths occurs outside RTO boundaries (Figures S14 and S15).

3.4.2. States. Environmental justice aspects of EGU-$PM_{2.5}$ vary by state and by EJ metric employed (Figures 5 and S16, Table S5). For example, the impacts in Texas from nationwide emissions are greater for blacks than any other race-ethnicity group, with a gap of ∼2.4 deaths/100 000 people between the most-exposed (black) and least-exposed (white Latino) group. In contrast, in Oklahoma, impacts are greatest for Native Americans, with a gap of ∼2.1 deaths/100 000 people between the most-exposed (Native American) and least-exposed (Asian) group. Among the seven states with the largest gaps by race (Figure 5; >2 premature deaths per 100 000 people), one can find examples where the most-exposed group is white non-Latino (Maryland, Pennsylvania, Virginia), black (Kentucky, Texas), white Latino (Mississippi), and Native American (Oklahoma); in total, these seven states comprise 64 million people (compared to 21 million people in the three analogous states (i.e., risk gap >2) from Figure 3). A map, analogous to Figures 3 and 5, but combining race/ethnicity and income, is in the Supporting Information (Figure S17). Impacts within each state by fuel-type and race-ethnicity are in Table S6.

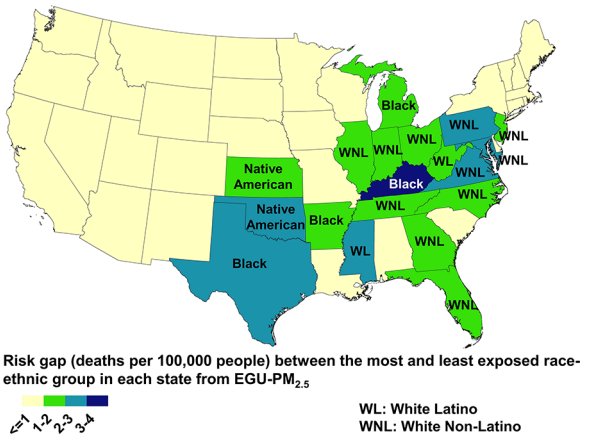

Figure 5. Most-exposed race-ethnic group and risk gap (units: deaths per 100 000 people) between the most- and least-exposed race-ethnic groups in each US state from total EGU-$PM_{2.5}$ emissions in the entire US. Race-ethnicity labels are displayed for states with a gap in the mortality rate greater than 1 death per 100 000 people. The remaining states are unlabeled because the gap between most- and least-exposed race-ethnicity groups is relatively small (less than 1 per 100 000 people). A version of the map displaying labels for all states is in Figure S16. Risk gap is shown by color gradation.

4. DISCUSSION

Previous studies have estimated the total damages associated with $PM_{2.5}$ from the US electricity sector. This work complements those findings by systematically analyzing the damages for different geographical boundaries (RTOs and states) and for different demographic groups (race and income).

We find that blacks are disproportionately affected by EGU-$PM_{2.5}$ nationally, but most-exposed race/ethnicity varies by state and by RTO. Differences by race/ethnicity hold across all income groups. Exposures are higher for lower-income than for higher-income households, but differences by race/ethnicity are larger than differences by income.

A substantial portion of the health risks in most states is attributable to out-of-state emissions, reflecting that EGU-$PM_{2.5}$ is a long-range pollutant. Some states are net imports of harm, others are net exporters. Regarding issues related to cross-state damages, EPA regulations such as the Cross-State Air Pollution Rule (CSAPR)[67] address air pollution from upwind states that crosses state lines and affect air quality in downwind states. Our findings highlight the interstate nature of EGU-$PM_{2.5}$ impacts: $PM_{2.5}$ mortality impacts in a state often are highly impacted by out-of-state EGU emissions.

Our estimate of total premature deaths (∼16 400 EGU-$PM_{2.5}$ premature deaths in 2014) is on the low end of the range given in the literature (see above); reasons include (1) reduced emissions in 2014 relative to 2005 and (2) differences in concentration response functions used in other studies (see below).

Our investigation considers exposure and health impacts from EGU-$PM_{2.5}$, instead of proximity to EGUs, as considered by previous studies.[68] Previous work[68] reported that the proportion of the population living within 30 miles of a power plant is greater for blacks than whites. Power plants are mostly located in rural areas, but the long-range transport of emissions can impact downwind population groups at varying magnitudes and distances. In considering an effective policy for reducing EGU impacts and disparities in impacts, it is important to consider the spatial distribution of emissions and populations, and the long-range transport of $PM_{2.5}$ and its emission precursors.

Future research could usefully explore the impact on results of (1) alternative models and modeling approaches, (2) grid-cell size, (3) alternative concentration−response functions (e.g., a supralinear C−R) or allowing the C−R to vary by source, geography, or chemical components,[69−72] (4) health

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Environ. Sci. Technol. XXXX, XXX, XXX−XXX

impacts of tropospheric (i.e., ground-level) ozone (O_3) formation from NO_x and VOCs emissions from EGUs, and (5) updated emissions for a more-recent year, such as 2017 NEI emissions.

Paolella et al. (2018)[73] demonstrated the importance of fine spatial resolution for identifying and quantifying exposure disparities. Our approach employs a reduced-complexity air-quality model (InMAP; ISRM) that uses a variable resolution grid (i.e., spatially fine grid in high population density areas). Thus, although our approach addresses the needs identified by Paolella et al. (2018)[73] (i.e., smaller-scale grid cells), we hypothesize that grid-cell size is less important for issues considered here because (1) EGUs typically have tall stacks, which spread pollution and reduce the relative importance of local conditions and (2) EGU-$PM_{2.5}$ is a long-range pollutant (secondary $PM_{2.5}$ formation is dominated by emissions of SO_2, which typically takes several days to transform into $PM_{2.5}$). Future research could test whether results seen here are also observed using other RCMs[74] or a conventional CTM.

Our study employs the mortality hazard ratio of 1.078 for all-cause mortality from the American Cancer Society (ACS) reanalysis study (Krewski et al. 2009)[65] to estimate premature deaths using a linear C−R function with no threshold. This C−R function is considered a US EPA standard and is commonly used in regulatory and academic air-quality research.[54,75,76] To understand the impact of alternative mortality hazard ratios on the premature deaths calculated in this work, as a sensitivity analysis we use values from Lepeule et al. (2012) (i.e., reanalysis of the Harvard Six Cities (H6C) study) [1.14, 95% CI = 1.07−1.22],[77] Vodonos et al. (2018) [1.129, 95% CI=1.109−1.150],[78] and Pope et al. (2019) [1.12, 95% CI = 1.08−1.15].[79] These alternative hazard ratios increase the national premature deaths estimated in this work substantially: by 75% [Lepeule et al. (2012)], 62% [Vodonos et al. (2018)], and 51% [Pope et al. (2019)]. Future research in the $PM_{2.5}$ C−R functions may benefit from relationships that are specific to the source sector, geographical region, or chemical constituents. Straightforward interpretation of the values individually reported by those three studies suggests an uncertainty of up to a factor of 2 (i.e., for Lepeule, the lower CI relative to the central estimate) if considering the 95% CIs, an uncertainty of 17% if considering just the three central-tendency estimates used in this sensitivity analysis, and an uncertainty of up to 75% if comparing those three studies against our base-case predictions. Detailed quantification of uncertainty in the C−R, via meta-analysis or other techniques, is outside the scope of research for this article; however, these comparisons suggest that our results are conservative, i.e., likely underestimate the true health impacts from air pollution.

Health impacts of ozone from EGU emissions have been previously modeled for the year 2005 and are estimated to be much smaller (∼1−10% of the total impacts from EGUs in the US) than $PM_{2.5}$-related impacts.[10,12,13] We studied the year 2014 because it reflects the most-current, well-vetted NEI dataset available. Emissions and population patterns may differ for the year 2014 than for more-recent (and future) years, which would impact results here. In general, electricity generation has lower emissions and uses less coal today than in 2014.[80] For that reason, total deaths per year from EGU-$PM_{2.5}$ are likely lower for the present year than for 2014.

Our analysis could be extended to other specific sectors of the economy. We hope that results here are useful for scientists and policymakers to understand and address disparities in air pollution exposure by race, income, and geography. Reductions in EGU emissions and EGU-$PM_{2.5}$ would not only save lives but also can reduce environmental and health inequalities.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.9b02527.

Map of US RTOs and their demographics, health impacts by race-ethnicity groups at national, RTO, and state level, health impacts by household income groups and race-ethnicity for each US RTO and state, population-weighted average $PM_{2.5}$ concentration for RTOs and states (PDF)

EGU-$PM_{2.5}$ health impacts from each state to the rest of the states and other summary tables (XLSX)

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