Inequity in consumption of goods and services adds to racial–ethnic disparities in air pollution exposure

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Fine particulate matter ($PM_{2.5}$) air pollution exposure is the largest environmental health risk factor in the United States. Here, we link $PM_{2.5}$ exposure to the human activities responsible for $PM_{2.5}$ pollution. We use these results to explore "pollution inequity": the difference between the environmental health damage caused by a racial–ethnic group and the damage that group experiences. We show that, in the United States, $PM_{2.5}$ exposure is disproportionately caused by consumption of goods and services mainly by the non-Hispanic white majority, but disproportionately inhaled by black and Hispanic minorities. On average, non-Hispanic whites experience a "pollution advantage": They experience ∼17% less air pollution exposure than is caused by their consumption. Blacks and Hispanics on average bear a "pollution burden" of 56% and 63% excess exposure, respectively, relative to the exposure caused by their consumption. The total disparity is caused as much by how much people consume as by how much pollution they breathe. Differences in the types of goods and services consumed by each group are less important. $PM_{2.5}$ exposures declined ∼50% during 2002–2015 for all three racial–ethnic groups, but pollution inequity has remained high.

air quality | environmental justice | fine particulate matter | input–output | life cycle assessment

Fine particulate matter ($PM_{2.5}$) exposure is a major health risk factor in the United States, responsible for 63% of deaths from environmental causes and 3% of deaths from all causes (1). It is a risk factor that is inequitably distributed among demographic groups, including racial–ethnic groups, owing in part to differences in pollution concentrations at locations of residence (2, 3). The extent to which differences in consumption of goods and services by racial–ethnic groups contribute to observed disparities in exposure is unknown, as is whether racial–ethnic groups have benefited equitably from recent improvements in $PM_{2.5}$ air quality.

Here, we explore racial–ethnic disparities in the causation and effect of exposure to $PM_{2.5}$ in the United States. We do this by investigating links among pollution, the parties responsible for its emission, and the health impacts that result. First, we estimate mortality from $PM_{2.5}$ for all emission sources in the United States. Next, we attribute these emissions to the end-use activities and to the end-user parties ultimately responsible for their generation. Finally, we compare results among racial–ethnic groups to explore what we term "pollution inequity": the extent to which groups disproportionately contribute to or bear the burden of pollution.

We estimate mortality impacts in the United States from $PM_{2.5}$ exposure using spatially explicit emissions data from all pollutant emission sources (4), the Intervention Model for Air Pollution (InMAP) air quality model (5), and spatially explicit population and health data (ref. 6; see *Materials and Methods*). We consider emissions of primary $PM_{2.5}$ and of secondary $PM_{2.5}$ precursors, both of which contribute to increased atmospheric $PM_{2.5}$ concentrations. Our approach yields estimates of premature deaths caused by $PM_{2.5}$ exposure in the United States for each year during 2003–2015, disaggregated by 5,435 emissions source types, at a spatial resolution varying between 1 and 48 km depending on population density. We aggregate impacts into 15 emitter groups. (See *Materials and Methods*; *SI Appendix*, Tables S1–S14 show the largest emitter types in the 14 anthropogenic and domestic emitter groups.)

We estimate a population-weighted average ambient $PM_{2.5}$ exposure concentration of 7.7 $\mu g \cdot m^{-3}$ for the United States in 2015, causing 131,000 premature deaths (Fig. 1 and *SI Appendix*, Fig. S1; see *SI Appendix*). Of these, 102,000 are caused by US anthropogenic emissions and 29,000 by other sources, largely wildfires and natural biogenic emissions (26,000), with minor contributions from Canadian and Mexican emissions (3,000). The total number of deaths reported here is higher than a commonly cited estimate of 93,000 (1), but at the low end of the range of a recently published estimate of 121,000–213,000 deaths (7), which uses a concentration–response relationship similar to the one employed here (6). (*SI Appendix*, Table S15 reports estimates of $PM_{2.5}$ mortalities using several concentration–response functions.)

Responsibility for air pollution is typically assigned to its emitters (8) (e.g., a factory), but it can also be ascribed to end uses (e.g., the purchase and use of manufactured goods) by end users (e.g., individual consumers) that ultimately result in its release (Fig. 1). Here, we connect $PM_{2.5}$ air pollution and its health impacts to end uses

Significance

Racial–ethnic disparities in pollution exposure and in consumption of goods and services in the United States are well documented. Some may find it intuitive that, on average, black and Hispanic minorities bear a disproportionate burden from the air pollution caused mainly by non-Hispanic whites, but this effect has not previously been directly established, let alone quantified. Our "pollution inequity" metric is generalizable to other pollution types and provides a simple and intuitive way of expressing a disparity between the pollution that people cause and the pollution to which they are exposed. Our results are timely, given public debate on issues relating to race, equity, and the regulation of pollution.

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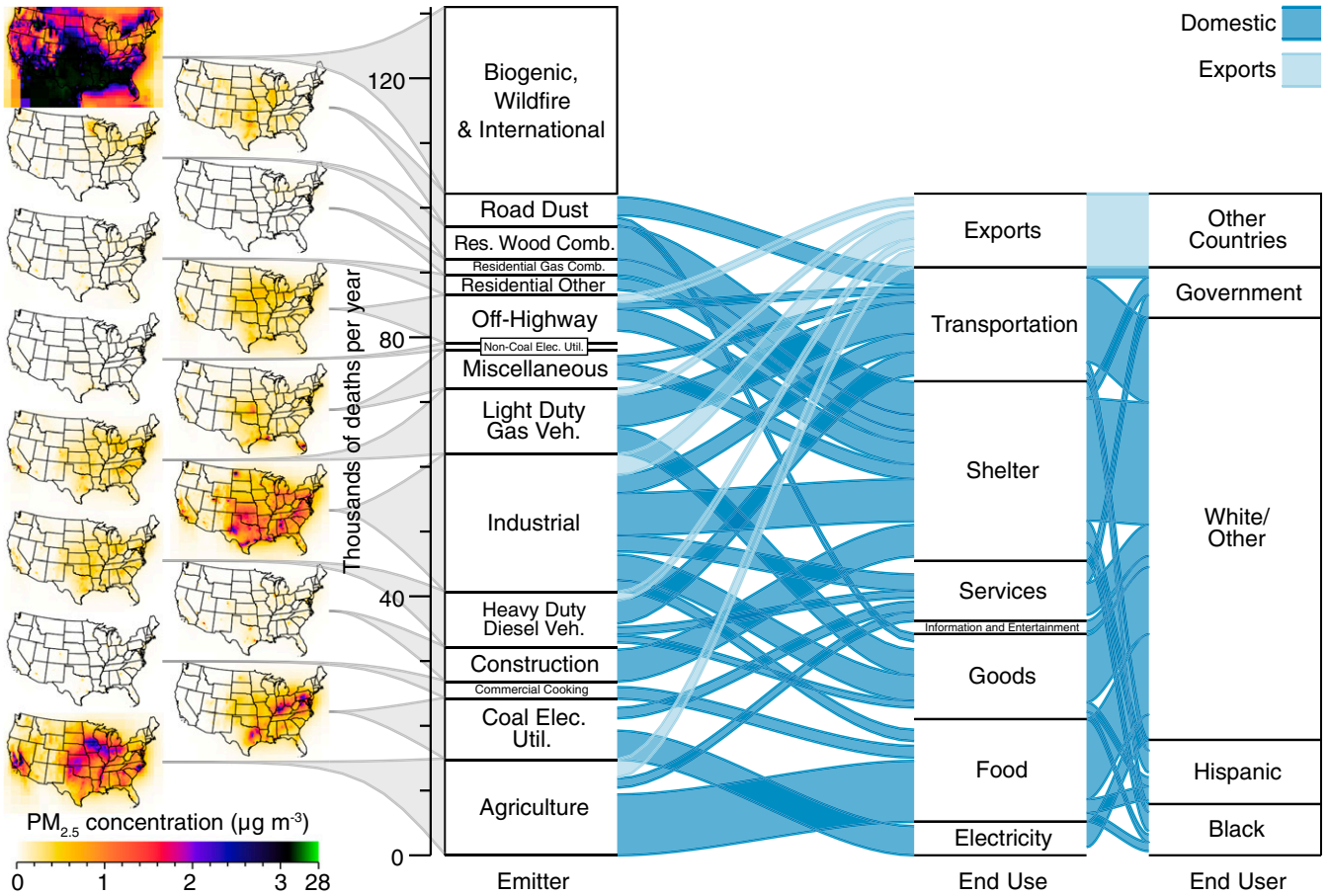

Fig. 1. Sources of US mortality from $PM_{2.5}$. $PM_{2.5}$ concentrations resulting from emissions from each emitter group [maps on *Left*; color scale contains a discontinuity at the 99th percentile of concentrations (i.e., 3.1 $\mu g \cdot m^{-3}$)] and relationships among $PM_{2.5}$ health impacts as attributed to emitters (*Left* bar), end uses (*Middle* bar), and end users (*Right* bar). The height of the bar on the *Left* shows the number of $PM_{2.5}$-attributable premature deaths caused by the physical production of emissions from each group of emitters, the height of the *Middle* bar shows the number of deaths caused by demand for each group of end uses, and the height of the bar on the *Right* shows the number of deaths caused by different types of end users. The blue connecting lines show relationships among emitters, end uses, and end users; connecting lines representing <1,000 deaths are not shown. (Detailed relationships between end uses and emitters for each racial–ethnic end-user group are shown in Fig. 2; time trends are shown in *SI Appendix*, Fig. S4.)

and end users by coupling economic input–output relationships to pollution emission sources (https://www.bea.gov/industry/io_annual.htm). Our approach allows us to attribute responsibility to (*i*) emitter entities that physically emit air pollutants; (*ii*) end uses that lead to air pollution emissions, often through intermediate economic transactions; and (*iii*) end users. We track 19 end-user types, which we aggregate here into four groups (personal consumption by each of three racial–ethnic groups, as well as government consumption), and 389 end-use categories, which we aggregate here into seven groups (electricity, food, goods, information and entertainment, services, shelter, and transportation).

Of 102,000 premature deaths from domestic anthropogenic emissions, we estimate 11,000 (11%) are caused by demand for goods that are exported (Fig. 1). Of the remaining 91,000 premature deaths caused by end uses within the United States, 83,000 (91%) are attributed to personal consumption (i.e., individual consumers); the remaining 8,000 (9%) are caused by pollution related to governmental expenditures.

To determine racial–ethnic inequity, we disaggregate personal consumption and exposure to $PM_{2.5}$ by race–ethnicity. Here, "exposure" is the population-weighted average ambient concentration at places of residence. We focus on the subset of impacts (83,000 premature deaths) that we can attribute to consumption by individuals in the United States, excluding the 48,000 premature deaths caused by governmental end uses, exports, and nonanthropogenic sources. (Racial–ethnic disparities in overall exposure to $PM_{2.5}$ from all sources are shown in *SI Appendix*, Fig. S2.) We consider persons self-identifying as black or African-American (hereafter, "black"; 12% of the population), Hispanic or Latino ("Hispanic"; 17% of the population), and the remainder [non-Hispanic white (62% of the population) plus all other race–ethnicity groups (8% of the population); hereafter, "white/other" (70% of the population)].

We define and quantify pollution inequity for a group g (I_g) as the fractional difference between a racial–ethnic group's exposure to $PM_{2.5}$ caused by all groups (E_g) and that group's population-adjusted contribution to the overall $PM_{2.5}$ exposure of all groups (C_g) (Eq. **1**):

$$\left[I_g = \frac{E_g}{C_g} - 1 \right]. \qquad [1]$$

Positive values for pollution inequity indicate that a group experiences more exposure than it causes (on average and after adjusting for population sizes); negative values indicate the opposite.

We find that blacks are exposed to 6.0 $\mu g \cdot m^{-3}$ of $PM_{2.5}$ (E_g), which is 21% greater than the overall population average exposure of 5.0 $\mu g \cdot m^{-3}$, while their population-adjusted consumption causes $PM_{2.5}$ exposure of 3.8 $\mu g \cdot m^{-3}$ to the overall population (C_g), which is 23% less exposure than average (Fig. 2). We therefore estimate for blacks a pollution inequity of 56% (Fig. 3*A*; $6.0\,\mu g \cdot m^{-3} / 3.8\,\mu g \cdot m^{-3} - 1 = (1 + 0.21)/(1 - 0.23) - 1 = 56\%$). Hispanics are exposed to 12% more $PM_{2.5}$ than average (5.5 $\mu g \cdot m^{-3}$),

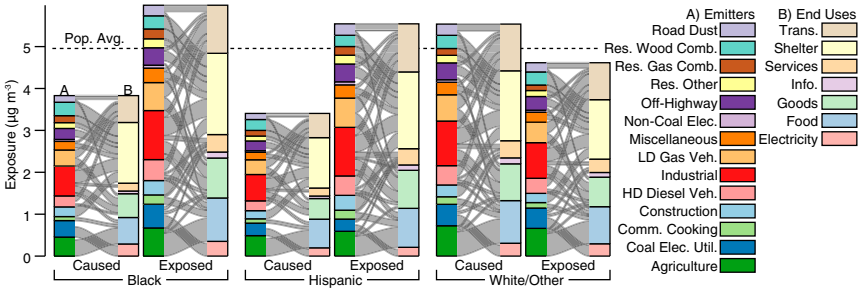

Fig. 2. Average $PM_{2.5}$ exposure experienced and caused by racial–ethnic groups. Total exposure to $PM_{2.5}$ caused by population-adjusted group consumption ("caused," or C_g) and group exposure to $PM_{2.5}$ caused by total personal consumption ("exposed," or E_g), stratified by racial–ethnic group. Pollution inequity is the percent difference between a group's "exposed" and "caused" bars. Each group of bars shows the (*A*) emitters and (*B*) end uses responsible for the exposure, with gray connecting lines showing relationships among emitters and end uses. Connecting lines representing <0.04 $\mu g \cdot m^{-3}$ are not shown.

but cause 31% less exposure than average (3.4 $\mu g \cdot m^{-3}$), for a pollution inequity of 63%. Whites/others are exposed to 7% less $PM_{2.5}$ than average (4.6 $\mu g \cdot m^{-3}$), but cause 12% more exposure than average (5.5 $\mu g \cdot m^{-3}$), for a pollution inequity of –17%.

Blacks are more exposed than whites/others to pollution from every emitter group (Fig. 2). The same holds for Hispanics, with the exceptions of $PM_{2.5}$ originating from agriculture, from coal electric utilities, and from residential wood combustion, for which they are exposed to 11%, 40%, and 14% less, respectively, than whites/others. Those three types of emissions are concentrated in regions of the United States with relatively low Hispanic populations (Fig. 1). Whites/others consume more—and cause more exposure—than do blacks and Hispanics across all seven end-use categories; the end uses representing the greatest differences in consumption-caused exposure are food (for which whites/others cause 61% and 49% more exposure than blacks and Hispanics, respectively), transportation (74% and 93%), and services (118% and 114%).

Differences in consumption across groups are comparable or larger contributors to pollution inequity than are differences in exposure across groups. Consumption differences account for 52%, 73%, and 63% of overall pollution inequity for blacks, Hispanics, and whites/others, respectively (Fig. 3*A*). Previous analyses have found that when considering only differences in locations of residence, exposure disparities by race are much larger than disparities by income (9, 10). Our results suggest that income, to the extent that it correlates with consumption, is an important factor in determining how much pollution a person causes, even if it may be statistically less important as a determinant of exposure. We also find that differences in racial–ethnic groups' contribution to exposure are driven more by differences in their overall amount of consumption (magnitude effect) than by differences in the types of goods and services they consume (composition effect) (Fig. 4 and *SI Appendix*, Fig. S3).

Exposure to $PM_{2.5}$ caused by personal consumption by all three racial–ethnic groups decreased by an average of 51% during 2003–2015 (Fig. 3*B*, *SI Appendix*, Fig. S4, and Movie S1), even as personal consumption expenditures increased (*SI Appendix*, Fig. S4 and Tables S16–S18). Furthermore, absolute differences in exposure caused by overall consumption decreased among groups, as did absolute differences in overall exposure caused by each group's consumption (Fig. 3*B*). Pollution inequity has remained high, however, decreasing by 23% for blacks (from 73% in 2003 to 56% in 2015) but increasing by 5% for Hispanics (from 60% in 2003 to 63% in 2015; Fig. 3*C*). Increases in consumption during 2003–2015 were larger for blacks than Hispanics for most sectors of the economy; two notable examples are spending on shelter (17% and 2% increases, respectively) and goods (21% and 6%, respectively) (*SI Appendix*, Tables S16–S18). Decreases in absolute exposure differences were primarily caused by decreases in the $PM_{2.5}$ concentrations where blacks and Hispanics live, rather than by blacks and Hispanics moving to locations with lower $PM_{2.5}$ concentrations (*SI Appendix*, Fig. S2).

Here, we have described linkages between human end-use activities and air pollution—and the racial–ethnic disparities therein. We find that, in the United States, $PM_{2.5}$ air pollution is disproportionately induced by the racial–ethnic majority and disproportionately inhaled by racial–ethnic minorities. All have benefited from recent reductions in atmospheric $PM_{2.5}$ concentrations. Our analysis shows for the first time how pollution inequity is driven by differences among racial–ethnic groups in both exposure and the consumption that leads to emissions. Still, questions remain about the spatial context of pollution inequity, its underlying causes, how best to address it, and its generalizability. For example, little is known about the "spatial scale" of inequity, such as whether consumers tend to live near to or far from the people exposed to the pollution resulting from their consumption. Further information on this issue would clarify whether this inequity could best be investigated and addressed at the city, state, or national level. Another open question is whether the patterns of pollution inequity described here are observed for other pollutants, times, or locations (e.g., in other countries). The pollution inequity metric defined here could be used to explore such questions and to inform discussion of inequity in other environmental burdens, including climate change, for which inequities can occur across continents and generations, in addition to across race–ethnicities.

Materials and Methods

Environmentally extended economic input–output analysis has been used to track air pollutant and greenhouse gas emissions induced by economic demand within and among national economies (11–16). Fewer studies have reported air quality-related health impacts induced by domestic (17, 18) and international trade (19–21). Here, we explore relationships among human end-use activities in the United States, $PM_{2.5}$-related health impacts caused by those activities, the corresponding consumption–exposure inequity among race–ethnicity groups, and related temporal trends.

Unlike analyses of greenhouse gas emissions for use in climate-change impact assessment, analyses of health impacts from non-greenhouse gas air pollution strongly benefit from spatial differentiation. For example, within the United States, health impacts per unit of emissions of $PM_{2.5}$ and its precursors

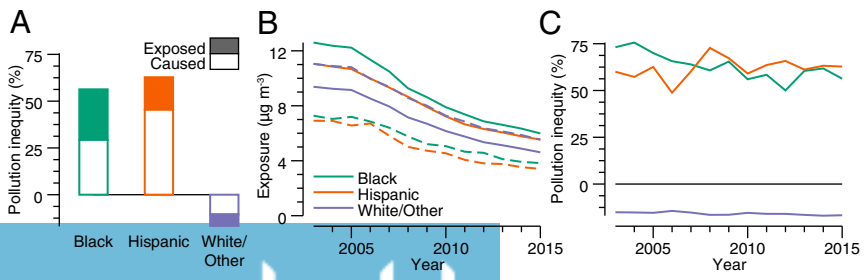

Fig. 3. Pollution inequity contributions and trends. (*A*) Contributions of differences in consumption (caused, or C_g) and location of residence (exposed, or E_g) to pollution inequity. (*B*) Exposure of each racial–ethnic group to $PM_{2.5}$ caused by the total combined personal consumption of all groups (exposed or E_g; solid lines) and total-population exposure to $PM_{2.5}$ caused by each group's population-adjusted consumption (caused, or C_g; dashed lines), 2003–2015. (*C*) Pollution inequity levels, 2003–2015.

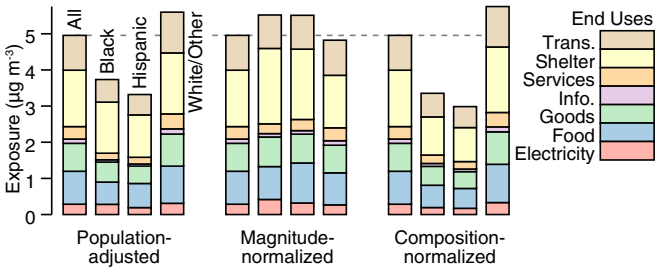

Fig. 4. Effect of magnitude and composition of consumption on $PM_{2.5}$ exposure. Population-adjusted $PM_{2.5}$ exposure (*Left*): actual population-adjusted exposure (as seen in Fig. 2). Magnitude-normalized $PM_{2.5}$ exposure (*Middle*): hypothetical exposure in which the overall magnitude of per capita consumption for each race–ethnicity is adjusted to match "All" without changing the composition of goods and services consumed. Composition-normalized $PM_{2.5}$ exposure (*Right*): hypothetical exposure where the composition of goods and services consumed by each race–ethnicity is adjusted to match All without changing the overall magnitude of consumption.

vary greatly across emission locations (22, 23). Spatial resolution is especially important when quantifying disparities in exposure among demographic groups (9). Therefore, to create a spatially explicit environmentally extended economic input–output model for the United States, we couple economic input–output (https://www.bea.gov/industry/io_annual.htm) and consumption (https://www.bls.gov/cex/) data with spatially explicit emissions data (4), the InMAP air quality model (5), and spatially explicit population and health data (ref. 6; https://www.census.gov/programs-surveys/acs/technical-documentation/table-and-geography-changes/2015/5-year.html; https://www.cdc.gov/nchs/data_access/cmf.htm). The resulting model relates air pollution emissions, concentrations, and health impacts with economic activity in the United States at a spatial scale varying between 1 and 48 km, depending on population density and emissions density. We refer to the model as the Extended InMAP Economic Input–Output (EIEIO) model, which is freely available at the Zenodo repository (24). InMAP is able to spatially resolve both the entire contiguous United States and within-city concentration gradients, which is critical for quantifying within- and among-city differences in exposure.

EIEIO uses economic data to trace human end-use activities that directly and indirectly cause air pollutant emissions and the resulting human exposure to $PM_{2.5}$. The model tracks relationships between final "end users," the activities or "end uses" they are participating in (or "final demand for commodities," in economic input–output terminology) that induce air pollution emissions, and the "emitter" entities that are physically releasing air pollutant emissions. EIEIO also tracks "intermediate uses." Intermediate uses are purchases by businesses to produce something that they are selling, whereas end uses are purchases or activities for reasons other than producing something to be directly sold. For example, the purchase of electricity to heat a home is an end use, whereas the purchase of electricity to manufacture fertilizer is an intermediate use. Our analysis includes both the emissions caused by an end use itself (e.g., tailpipe emissions from driving a car) and the emissions from economic activities in support of the end use (e.g., emissions from the production of gasoline to fuel the car).

EIEIO tracks 19 end-user types, 389 end-use categories, and 5,434 categories of emitters. For ease of display and communication, we present results here in groups of four users, seven uses, and 14 emitters; further details are in *SI Appendix*. Mappings from the use and emitter categories to corresponding groups are in Tessum et al. (24). Unless otherwise noted, all results are for year 2015.

Methods are described below and in Tessum et al. (24). The model source code includes a graphical interface that can be used for exploratory analysis and visualization. Results here were generated using a 2018-vintage Google Compute Engine instance with 32 CPU cores, 208 GB of RAM, and a 500-GB hard drive.

Economic Production. To relate final economic demand for commodities to economic activity or production in individual industries, we use the following US Bureau of Economic Analysis (BEA) Input–Output Accounts Data (https://www.bea.gov/industry/io_annual.htm):

i) Final demand (d_f): Economic activity that leads to the final consumption of a good or service and that is not induced by economic activity in another sector of the economy. This can include demand for exports.
ii) Import final demand (d_{fi}): Economic activity that leads to the final consumption of an imported good or service and that is not induced by economic activity in another sector of the economy.
iii) Total requirements (R_t): Direct plus indirect purchases from an industry required to produce a dollar of output of a commodity (25).
iv) Total domestic requirements ($R_{t,d}$): Domestic (i.e., within the United States) direct plus indirect purchases from an industry required to produce a dollar of output of a commodity.
v) Total import requirements ($R_{t,i}$): Calculated as $R_{t,i} = R_t - R_{t,d}$.

where d_f and $d_{f,i}$ are vectors with one entry for each of 389 commodity sectors, and R_t, $R_{t,d}$, and $R_{t,i}$ are matrices with one row for each of 389 industry sectors and one column for each of 389 commodity sectors.

We calculate economic production, ρ, caused by final demand as in Eq. **2**:

$$\rho = R d_f, \quad [2]$$

where R is one of R_t, $R_{t,d}$, or $R_{t,i}$ depending on whether total, domestic, or international economic production is desired. For imports, d_f is replaced with d_{fi}. ρ is a vector with one entry for each industry sector.

BEA input–output data are disaggregated to the detailed level of 389 industries and 389 commodities for year 2007, and to the summary level of 71 industries and 73 commodities for years 1997–2015. To perform calculations for years other than 2007, we scale the detailed 2007 data as in Eq. **3**:

$$v_{d,i,c,y} = \frac{v_{d,i,c,2007}\, v_{s,i,c,y}}{v_{s,i,c,2007}}, \quad [3]$$

where $v_{d,i,c,y}$ is a value at the detailed level of aggregation for industry i and commodity c for the year of interest, $v_{d,i,c,2007}$ is the corresponding value at the detailed level of aggregation for year 2007, and $v_{s,i,c,y}$ and $v_{s,i,c,2007}$ are values for the corresponding summary level of aggregation for the year of interest and 2007, respectively.

Some negative values for final demand exist in the BEA input–output data tables. These typically relate to divestments or reductions in amounts of stocks. Because our objective is to use economic relationships to model air pollution emissions and impacts, and divestments or stock reductions do not cause negative emissions in the same way that investments and increases in stocks can be said to cause positive emissions, we set all negative final demand values to zero.

Demographic-Specific Personal Consumption Demand. BEA input–output data report final demand from personal expenditures, but the data do not disaggregate consumption by racial or ethnic groups. To calculate demographic-specific consumption, we match categories in the US Bureau of Labor Statistics Consumer Expenditure Survey (CES) (https://www.bls.gov/cex/) to the BEA input–output sectors, then use the demographic information in the CES data to allocate BEA personal expenditures among demographic groups. The CES data report expenditures separately for the following: Hispanics or Latinos; Not Hispanic or Latino: whites and all other races; and Not Hispanic or Latino: blacks or African-Americans.

As of this writing, CES data are available for the years 2003–2015. EIEIO does not account for geographic variation in consumption amounts or in the proportions of goods and services consumed.

Augmented Personal Consumption. In addition to personal consumption (causing 46,000 premature deaths from $PM_{2.5}$), we also attribute BEA private expenditure final demand categories to individual end users and allocate the expenditures among demographic groups. We do this by directly adding final demand for "Residential private fixed investment" (16,000 premature deaths from $PM_{2.5}$) to personal consumption, as individuals are the ultimate end users of residential buildings. The remaining private expenditure categories include expenditures on nonresidential structures (9,400 deaths), nonresidential equipment (9,400 deaths), and intellectual property (500 deaths), as well as changes in inventory (1,700 deaths). Because consumption activities provide the revenue streams that organizations use to make capital investments and to generate inventory, albeit with time lags that we do not account for here, we consider these expenditures—and the resulting air pollution—to be caused by personal consumption. Therefore, we attribute these additional categories of demand to demographic groups proportionate to each group's overall fraction of combined personal consumption and residential investments. Although government expenditures are also ultimately funded by individuals, the taxes that fund the government are compulsory, and relationships between individual tax contributions and government spending decisions are uncertain. Therefore, we do not attribute government expenditures to individuals, but instead track and display them as their own category.

Emission Factors. We create spatially explicit emissions factors—in units of mass per time of emissions of primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors [oxides of nitrogen (NO_x), oxides of sulfur (SO_x), ammonia (NH_3), and volatile

organic compounds (VOCs)] per dollar—for each of the 5,434 EPA source classification codes (SCCs) in the year 2014 US National Emissions Inventory (NEI), version 1 (4). Each emissions record in the NEI contains an SCC that specifies the type of source creating the emissions. First, we match each SCC to one or more of the 389 BEA industries. Some sources of emissions cannot be directly matched to BEA industries because they do not result from economic transactions. We match these sources to the BEA industry to which it is most closely related. The largest source of these nontransactional emissions is the personal use of light-duty vehicles, which we match to the "automobile manufacturing" industry based on the assumption that the individuals and entities that drive light-duty vehicles and create the resulting emissions are the same as the individuals and entities that purchase automobiles. Other nontransactional sources of emissions include leisure activities such as barbecuing and operating recreational vehicles, which we attribute to relevant residential or recreational industries. The cross-walk between SCCs and BEA industries can be found in Tessum et al. (24). We use this cross-walk to map the economic production vector, ρ, which has one element for each BEA industry, to vector $\hat{\rho}$, which has one element for each SCC equal to the sum of economic production in the BEA industry or industries that the SCC is matched to. $\hat{\rho}$ double counts economic production in some cases, but is used in a way that ensures emissions are not double counted.

Next, we process the NEI emissions (excluding emissions occurring in Canada and Mexico, which are tracked separately) using the InMAP Air Emissions Preprocessor program, also included in Tessum et al. (24). We assign each emissions record to the BEA industry or industries it belongs to and allocate the emissions to a spatial grid with cell edge lengths varying between 1 and 48 km, depending on population density and emission density. [The grid employed by InMAP is described further by Tessum et al. (5).] We allocate county-specific emissions to grid cells within counties using spatial surrogates, as described by the US EPA (4).

Finally, we calculate spatially explicit emissions factors by dividing the emissions from each SCC by the total domestic economic production in the matched industry or industries (i.e., $\hat{\rho}$) resulting from domestic and export final demand. The result is a series of emissions factor matrices, E_p, where p is one of the pollutants in (primary $PM_{2.5}$, NO_x, SO_x, NH_3, VOC). Each emissions factor matrix has one row for each spatial grid cell, one column for each SCC, and dimensions of [mass·time^{-1}·\$$^{-1}$].

For analysis years other than 2014, we adjust the 2014 NEI emissions according to state- and source-group-specific annual trends in emissions published by the US EPA (https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data). To quantify health impacts from non–human-related emissions sources, we also include combined biogenic and wildfire emissions from year 2005, as processed by Tessum et al. (26). Further information is in *SI Appendix*. We calculate spatially explicit emissions of a pollutant p (e_p) induced by human activity (using economic final demand as a surrogate for human activity) as shown in Eq. **4**:

$$e_p = E_p \hat{\rho}, \quad [4]$$

where e_p is a vector with length equaling the number of spatial grid cells and dimensions of [mass·time^{-1}].

$PM_{2.5}$ Concentrations. Primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors are emitted into the atmosphere where they are transported by wind, transformed by chemistry, and ultimately inhaled by humans or otherwise removed. We account for these phenomena using InMAP, version 1.2.1 (5); InMAP creates spatially explicit estimates of ambient $PM_{2.5}$ concentrations caused by the emissions estimated by EIEIO. For computational expedience, we use InMAP to create a set of source–receptor matrices, which describe linear relationships between (*i*) emissions in each of many source locations and (*ii*) concentrations in each of many receptor locations. We create the InMAP source–receptor matrix (ISRM) by running separate InMAP simulations that estimate the ground-level changes in $PM_{2.5}$ concentrations of emissions of SO_x, NO_x, VOCs, NH_3, and primary $PM_{2.5}$ in each of ∼50,000 InMAP grid cells. This is repeated three times to consider emissions plume height ranges of 0–57, 240–380, and 760–1,000 m, for a total of ∼150,000 simulations. The result can be represented as a rank-four tensor describing independent linear relationships between emissions and $PM_{2.5}$ concentrations for discrete combinations of pollutant emitted, emissions source location, emissions plume height, and concentration receptor location. By using linear interpolation to calculate impacts for sources with plume heights that do not fall within the modeled height ranges, ISRM can quickly calculate $PM_{2.5}$ concentrations resulting from arbitrary combinations of emissions sources and locations. ISRM model performance evaluation is in *SI Appendix*.

Ground-level concentrations of $PM_{2.5}$ depend on the height and location of emissions; therefore, instead of directly using the E_p matrices to calculate concentration impacts, we create a separate series of matrices for the concentration factor, C_p, for each emitted pollutant, p, by using the ISRM to calculate total concentrations from the NEI emissions records associated with each SCC—while accounting for individual plume heights from each emissions record—and dividing the result by the total transformed domestic economic production, $\hat{\rho}$. The resulting matrices, C_p, have one row for each spatial grid cell, one column for each SCC, and units of micrograms per cubic meter per dollar. Total $PM_{2.5}$ concentration impacts (c) of economic final demand are calculated by summing impacts from each emitted pollutant as in Eq. **5**:

$$c = \sum_p \{C_p \hat{\rho}\}, \quad [5]$$

where c is a vector with length equaling the number of spatial grid cells and units of micrograms per cubic meter.

Health Impacts. Air pollution-related health impacts from economic final demand are a function of population counts, underlying incidence rates, and concentration–response relationships, in addition to the $PM_{2.5}$ concentrations themselves.

Population counts. Population counts are based on data from the US Census Bureau American Community Survey (ACS) 5-Year Estimates (https://www.census.gov/programs-surveys/acs/technical-documentation/table-and-geography-changes/2015/5-year.html) for midpoint years 2007–2014, plus the year 2000 decennial census, downloaded from the National Historical Geographic Information System (27) at census block-group spatial resolution. We calculate health impacts for several race–ethnicity categories:

i) Total population (314 million people in our study domain, as of 2014).
ii) People of all races who are Hispanic or Latino; we refer to this group as Hispanic (54 million people).
iii) People who are not Hispanic or Latino and are black or African American alone; we refer to this group as black (39 million people).
iv) All people who are not in the Hispanic or black groups; we refer to this group as white/other; this group includes 196 million whites, 15 million Asians or Pacific Islanders, 2 million American Indians, and 8 million Others/Multiple Races.

Population counts for years 2001–2006 are estimated using spatially explicit interpolation with 2000 and 2007 as the endpoints, years 1997–1999 use year 2000 population counts without modification, and year 2015 uses year 2014 population counts without modification. Data for years 2007–2014 are directly available from ACS. We use the total population count to calculate total health impacts, and we use the separate counts for each demographic group to calculate inequity in $PM_{2.5}$ exposure. The racial–ethnic groups used here were chosen to align with the demographic groups in the Consumer Economics Survey (https://www.bls.gov/cex/). We use population counts for people of all ages, rather than restricting the analysis to a specific age range. One reason for this is that publicly available US Census data do not include both race–ethnicity and age information at the block-group spatial resolution. We allocate population counts to spatial grid cells, using area weighting for census block groups that overlap more than one grid cell. The resulting vectors, p_g, where g is the set of demographic groups above, have one row for each grid cell and units of [persons].

Underlying incidence rates. We use county-specific data for baseline all-cause mortality rates from the US Centers for Disease Control and Prevention (https://www.cdc.gov/nchs/data_access/cmf.htm) for years matching the population years above. We use mortality rates for the full population, rather than for a specific age range. Following Apte et al. (28), we calculate the county-average underlying mortality incidence rate, I_o, as in Eqs. **6** and **7**:

$$I_{o,c} = \frac{I_c}{\overline{\mathrm{HR}}_c}, \quad [6]$$

$$\overline{\mathrm{HR}}_c = \frac{\sum_{i=1}^{N_c} P_i \times \mathrm{HR}(C_i) f_{i,c}}{\sum_{i=1}^{N} P_i}, \quad [7]$$

where I_c is the reported mortality rate in a given county; $\overline{\mathrm{HR}}_c$ is the average mortality hazard ratio caused by $PM_{2.5}$ in county c; i is one of N_c grid cells in county c; P_i is population count in grid cell i; $\mathrm{HR}(C_i)$ is the result of the concentration–response relationship described below for total $PM_{2.5}$ concentration C_i, calculated as described in *$PM_{2.5}$ Concentrations*; and $f_{i,c}$ is the area fraction of grid cell i that overlaps with county c. The term $I_{o,c}$ represents a hypothetical mortality incidence rate in the absence of ambient $PM_{2.5}$. For health impact calculations, we assume that the underlying incidence rate for all racial–ethnic groups is the same as the population average. We calculate a US population-average $I_{o,c}$ of 763 deaths per 100,000 people per year in 2014.

Concentration–response relationship. We represent the effect of changes in $PM_{2.5}$ concentration on mortality rates using the relationship described by Nasari et al. (6) and Burnett et al. (7), as in Eq. **8**:

$$\mathrm{HR}(C) = \exp\left(\frac{\gamma * \ln(C+1)}{1+\exp[-(C-\delta)/\lambda]}\right), \quad [8]$$

where $\mathrm{HR}(C)$ is the hazard ratio of mortality incidence at $PM_{2.5}$ concentration C—in units of micrograms per cubic meter—compared with a hypothetical underlying incidence rate, I_o, in the absence of ambient $PM_{2.5}$. γ, δ, and λ are empirically determined constants. Nasari et al. use an ensemble version of Eq. **8**, where γ, δ, and λ take many combinations of values and the prediction of each combination is weighted by its performance in predicting health outcomes in the American Cancer Society cohort. To reduce model complexity and computational expense, we use a deterministic version of the relationship, where $\gamma = 0.0478$, $\delta = 6.94$, and $\lambda = 3.37$ are determined using nonlinear regression to predict the expected value of the ensemble prediction. The relationship used here and by Nasari et al. (6) differs from the relationship presented by Burnett et al. (7) in that it is derived from the US-based American Cancer Society cohort rather than from 41 global cohorts.

The term $\mathrm{HR}(C)$ is a nonlinear function; therefore, the impact of a change in concentration depends on the initial concentration. It follows that if a number of emissions sources are consecutively added or subtracted from an area, their health impact per unit emission will depend on the order that they were added or subtracted. We assume that the impact of each unit $PM_{2.5}$ is equal to the average per-unit impact of $PM_{2.5}$ in a given location, as in Eq. **9**:

$$\overline{\mathrm{HR}}_i = \frac{\mathrm{HR}(C_{t,i})}{C_{t,i}}, \quad [9]$$

where $\overline{\mathrm{HR}}_i$ is the average per-unit concentration hazard ratio at location i, and $C_{t,i}$ is the total concentration at location i.

As a sensitivity analysis, we also use three other hazard ratio models based on the work of Krewski et al. (29) and Lepeule et al. (30), which all take the form shown in Eq. **10**:

$$\mathrm{HR}(C) = \exp(\beta \times \max[0, C - C_o]), \quad [10]$$

where β is an empirically determined constant. We use two β values reported by Krewski et al. (29): $\beta = \ln(1.06)/10$ and $\beta = \ln(1.078)/10$. We also use $\beta = \ln(1.14)/10$ as reported by Lepeule et al. (30). C_o represents the lowest observed concentration: 5 $\mu g \cdot m^{-3}$ for Krewski et al. (29) and 8 $\mu g \cdot m^{-3}$ for Lepeule et al. (30); our method assumes that for concentrations below this threshold, the risk of $PM_{2.5}$-caused premature mortality is zero.

Health impact calculation. We calculate the health impacts of air pollution using Eq. **11**:

$$M(C_i) = p_i \sum_c^n I_{o,c} f_{i,c} \overline{\mathrm{HR}}_i, \quad [11]$$

where $M(C_i)$ is the number of mortalities caused by the concentration of pollution (C_i) at location i, p_i is the population count in grid cell i, $I_{o,c,i}$ is the underlying incidence rate for one of n counties (c) overlapping grid cell i, and $f_{i,c}$ is the fraction of grid cell i that overlaps county c. We then calculate the $PM_{2.5}$ health impacts, d, of economic final demand by combining Eqs. **5** and **11** in Eq. **12**:

$$d = M\left(\sum_p \{C_p \hat{\rho}\}\right), \quad [12]$$

where d is a vector with length equaling the number of spatial grid cells and units of [deaths].

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