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Letter

Reducing Mortality from Air Pollution in the United States by Targeting Specific Emission Sources

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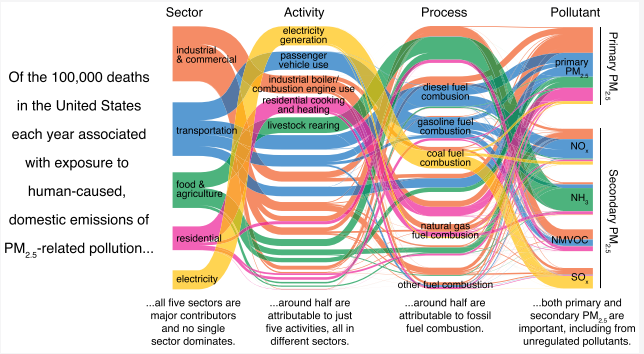
ABSTRACT: Air quality in the United States has dramatically improved, yet exposure to air pollution is still associated with 100000−200000 deaths annually. Reducing the number of deaths effectively, efficiently, and equitably relies on attributing them to specific emission sources, but so far, this has been done for only highly aggregated groups of sources, or a select few sources of interest. Here, we estimate mortality in the United States attributable to all domestic, human-caused emissions of primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors. We present detailed source-specific attributions in four alternate groupings relevant for identifying promising ways to reduce mortality. We find that nearly half of the deaths can be attributed to just five activities, all in different sectors. Around half of the deaths can be attributed to fossil fuel combustion, with the remainder attributable to combustion of nonfossil fuels, agricultural processes, and other noncombustion processes. Both primary and secondary $PM_{2.5}$ are important, including $PM_{2.5}$ from currently unregulated precursor pollutants such as ammonia. We suggest improvements in air quality can be realized by continued reductions of emissions from traditionally important sources and by novel strategies for reducing emissions from sources of emerging relative importance and research focus. Such changes can contribute to improved health outcomes and other environmental goals.



INTRODUCTION

After decades of effective environmental policy, air quality in the United States has dramatically improved, whether compared to historical levels or to the air breathed by the vast majority of people in the world. However, exposure to air pollution remains the greatest environmental health risk factor in the United States, associated with 100000−200000 excess deaths annually,[1,2] substantially more deaths than from murders and car crashes combined.[3]

Although regulatory efforts have focused on attaining air quality standards, reducing the health impacts of air pollution has, to date, been achieved largely by targeting emission sources.[4] Indeed, targeting specific emission sources based on a knowledge of the health impact of every emission source is the key to effective, efficient, and equitable regulation of air pollution. However, traditional methods for attributing health impacts to emission sources are computationally demanding and resource intensive and thus have been used for only highly aggregated groups of sources,[5] or a select few sources of interest.[6]

Here we consider the deaths attributable to all human-caused, domestic emissions of pollutants that contribute to increased atmospheric levels of fine particulate matter ($PM_{2.5}$) in the contiguous United States. We use newly developed models, which have been used in fine-scale pollution damage mapping and the assessment of environmental justice,[1,7] that greatly reduce the time and computational resources needed to trace the causal chain from emission sources to exposures, and to health impacts.[8] Our focus is on $PM_{2.5}$, which is most strongly associated with excess mortality risk from outdoor air pollution in the United States and globally.[9] We exclude emissions from biogenic sources and wildfires, and emissions from Mexico and Canada that affect $PM_{2.5}$ concentrations in the United States but are typically outside the scope of U.S. air quality policy. Furthermore, as we model outdoor $PM_{2.5}$ concentrations, we do not consider indoor or microenvironmental exposures such as from incense and tobacco.

We attribute the deaths in four alternate ways—by sector, activity, process, and pollutant—as follows. We first group all

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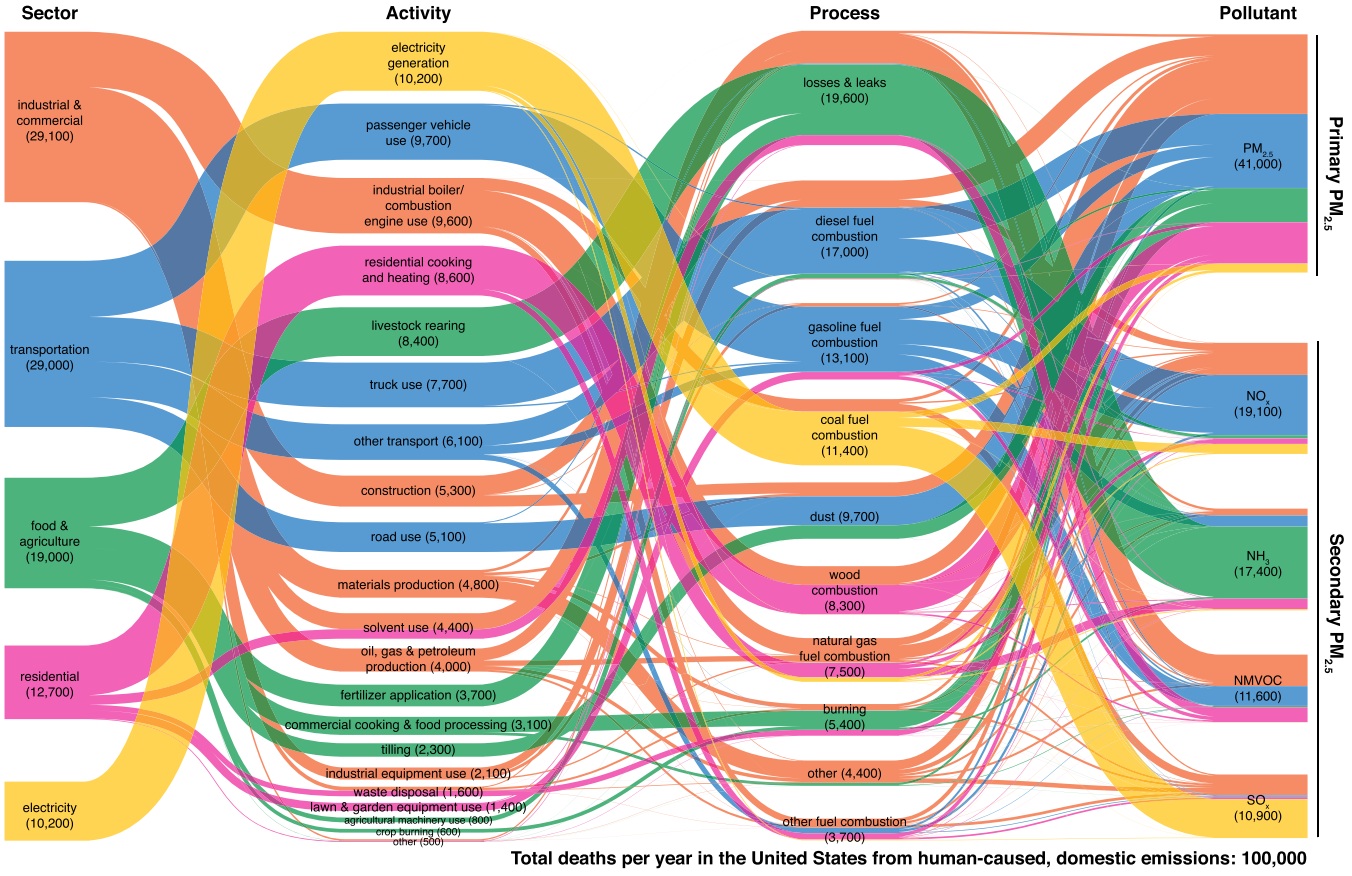

Figure 1. Deaths per year attributed to all human-caused emissions in the United States grouped by sector (leftmost column), activity (left middle column), process (right middle column), and pollutant emitted (rightmost column). The numbers of deaths are given in parentheses after each grouping. Flows are colored by the sector to which the deaths from each flow are attributed: industrial and commercial (orange), transportation (blue), food and agriculture (green), residential (red), and electricity (yellow). Within each column, flows are ordered from most (top) to fewest (bottom) deaths per year. "Other transport" includes railroad use (1800 deaths), marine vessel use (1400 deaths), light commercial vehicle use (1300 deaths), municipal vehicle use (1000 deaths), and aircraft use (700 deaths). "Other fuel combustion" includes fuel oil (1700 deaths). Deaths from wildfire, biogenic, and foreign emission sources are not shown.

human-caused emissions in the United States into five broad sectors (transportation, electricity, food and agriculture, residential, and industrial and commercial) and estimate annual deaths attributable to each sector. Then, we attribute the deaths to 25 activities within the sectors, and also to 10 processes by which pollutants are released. Finally, we attribute the annual deaths to the pollutants primary $PM_{2.5}$ and the four precursors of secondary $PM_{2.5}$: nitrogen oxides (NO_x), sulfur oxides (SO_x), ammonia (NH_3), and non-methane volatile organic compounds (NMVOC). Details of the methods, comparisons between models, and code to reproduce results are provided below and in the Supporting Information.

MATERIALS AND METHODS

Emissions of the five pollutant groups are taken from the U.S. Environmental Protection Agency National Emissions Inventory (NEI) 2014 version 1, as grouped by 5434 Source Classification Codes (SCCs). Emissions are prepared as described by Tessum et al.,[1] including quality control, emissions processing, and scaling to year 2015 using sector-specific scale factors. Here, instead of mapping SCCs to emission sources aggregated by industry for economic analysis, we map SCCs to emission sources aggregated by sector, activity, and process. Definitions clarifying the groups are provided in the Supporting Information.

$PM_{2.5}$ concentrations across the contiguous United States arising from the emission sources are modeled using the Intervention Model for Air Pollution (InMAP), an open-source, reduced-complexity air quality model, which is run using a variable resolution grid (1−12 km) and outputs from a more detailed model simulation to parametrize its chemistry.[10] InMAP modeling assumptions and performance evaluation are described elsewhere,[1,7,8,10] including performance evaluations against other models[8,10] and ground measurements for different $PM_{2.5}$-related species.[7,10] Source−receptor (S−R) matrices built from InMAP[7] were used to estimate changes in annual average concentrations of speciated $PM_{2.5}$ forms [primary $PM_{2.5}$, secondary organic aerosol (SOA), particulate nitrate, particulate ammonium, and particulate sulfate] arising from emissions from each source.

Deaths associated with changes in annual average concentrations of $PM_{2.5}$ species were estimated following Tessum et al.[1] in population counts, baseline mortality rates, and the $PM_{2.5}$ concentration−response relationship described by Nasari et al.[11] and Burnett et al.,[2] parametrized as in Tessum et al.[1] As the concentration−response relationship is nonlinear, the marginal effect of $PM_{2.5}$ concentrations on mortality varies depending on the total $PM_{2.5}$ concentration. To account for this dependence, total deaths in each grid cell are derived from InMAP-predicted total $PM_{2.5}$ concentrations from all emissions

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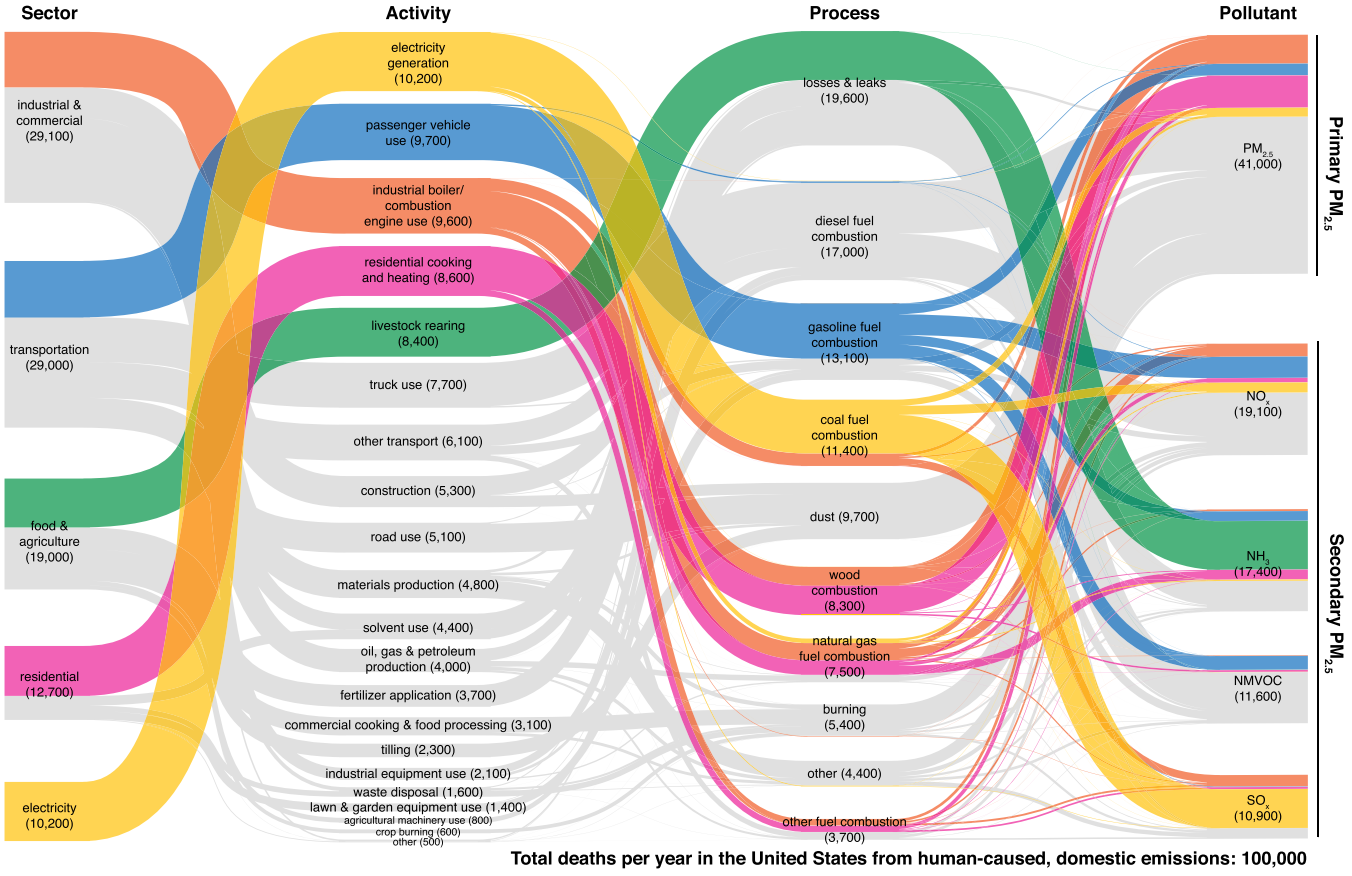

Figure 2. Deaths per year attributed to all human-caused emissions in the United States grouped as in Figure 1. Flows are colored by sector, as in Figure 1, for the five activities with the most attributed excess deaths. Flows colored gray are for excess deaths attributed to activities other than those five.

(including anthropogenic, biogenic and wildfire, and international emissions) used by Tessum et al.[1] The mortality in each grid cell is attributed to emissions from each source in proportion to the contribution those emissions make to the total $PM_{2.5}$ concentration.

The $PM_{2.5}$ concentration−response relationship used here is based on pooled estimates from available cohort studies that use proportional hazard regression survival models to evaluate associations between mortality risk and long-term $PM_{2.5}$ exposures.[1,11] The studies statistically control for various individual risk factors (e.g., age, sex, race, smoking, diet, obesity, and education) or neighborhood-level socioeconomic and other contextual variables. It is possible that observed associations are not causal but are the result of residual confounding or lack of adequate control by some unknown or unmeasured risk factor that is also associated with air pollution. However, consistent and coherent $PM_{2.5}$−mortality associations, especially with cardiorespiratory and lung cancer mortality,[12] have now been observed in diverse cohorts over decades of research and from various locations around the world.[13] Furthermore, these results are consistent with broader epidemiological evidence that includes a wide variety of alternative study designs, including applications of multiple causal analysis methods,[14,15] and a growing literature exploring biological mechanisms linking air pollution exposure to cardiovascular and respiratory morbidity and mortality.

In each grid cell, deaths associated with primary $PM_{2.5}$ concentrations were attributed to primary $PM_{2.5}$ emissions from each SCC. Although precursor pollutant emissions can influence the mass of $PM_{2.5}$ species other than the $PM_{2.5}$ mass they directly form (e.g., NH_3 emissions controls may reduce the level of particulate sulfate formation), here we attribute deaths from secondary $PM_{2.5}$ concentrations only to emissions of precursor pollutants that directly form the $PM_{2.5}$ species concentrations, by mass of $PM_{2.5}$ species formed. Therefore, for each SCC, in each grid cell, deaths attributed to changes in concentrations of SOA, particulate nitrate, particulate ammonium, and particulate sulfate were each attributed to emissions of NMVOC, NO_x, NH_3, and SO_x, respectively, from that SCC, from anywhere in the contiguous United States. Equal concentrations by mass of each $PM_{2.5}$ species were assumed to have equivalent toxicity. Each of the 5434 SCCs was allocated to exactly one sector, activity, and process; the full mapping between SCCs and the groups used here is provided in Data set S1. Figure 1 was visualized using the RAWGraphs platform[16] and InMAP-derived results.

To evaluate robustness, the same tests were performed with two other reduced-complexity air quality models, whose underlying mechanisms use assumptions different from each other and from InMAP: AP2 (Air Pollution Emission Experiments and Policy analysis model version 2) and EASIUR (Estimating Air pollution Social Impacts Using Regression). Their underlying mechanisms differ: AP2 estimates pollutant concentrations using a Gaussian dispersion model, whereas EASIUR uses statistical relationships between changes in emissions and simulation outputs from a chemical transport model. Comparisons among the three models, including their

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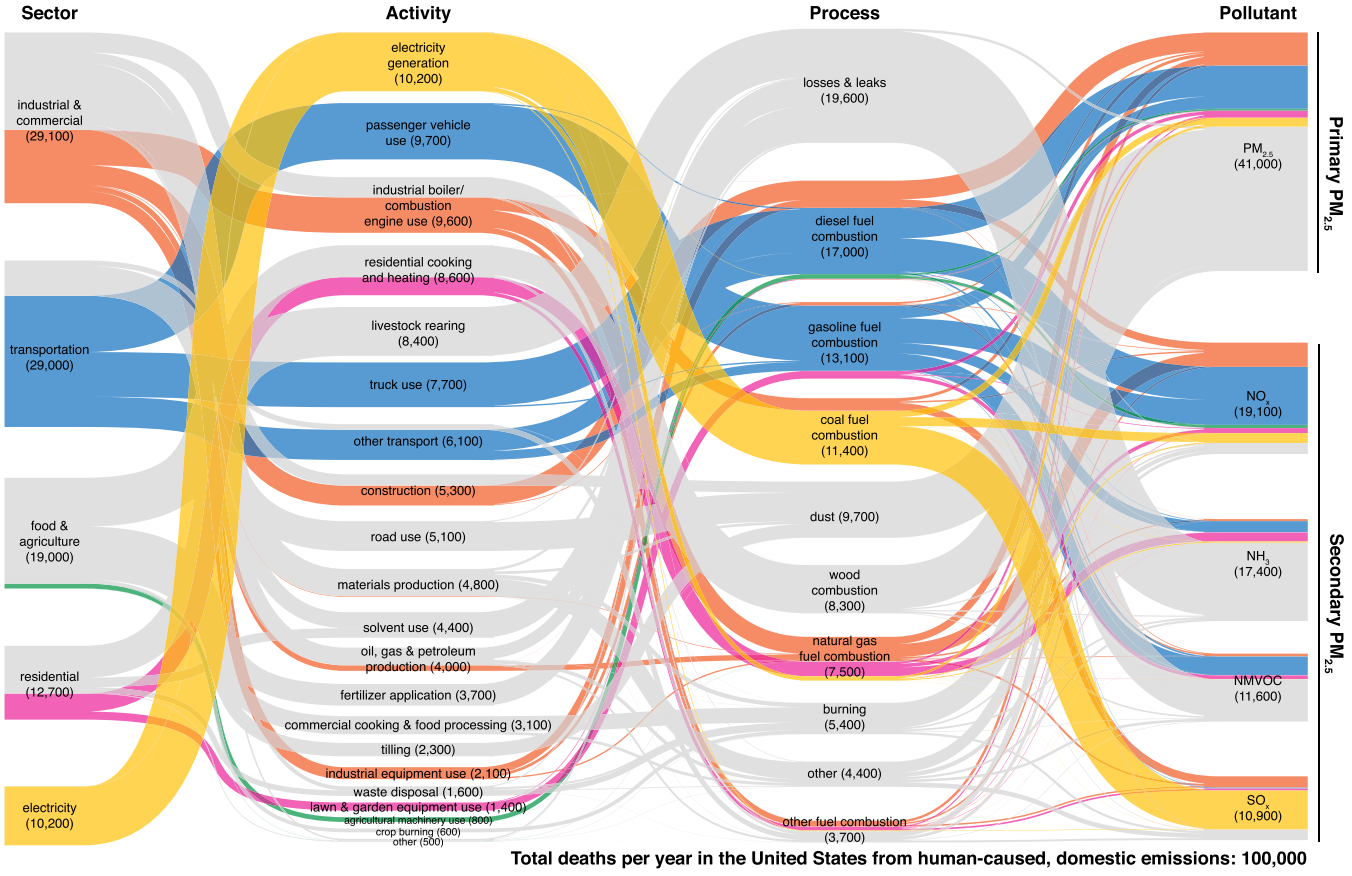

Figure 3. Deaths per year attributed to all human-caused emissions in the United States, grouped as in Figure 1. Flows are colored by sector, as in Figure 1, for fossil fuel combustion. Flows colored gray represent deaths attributed to processes other than fossil fuel combustion. Major fossil fuels are diesel, gasoline, coal, and natural gas, each of which is represented by a separate process. "Other fuel combustion" (3700 deaths) includes deaths attributable to combustion of minor fossil fuels, such as fuel oil, liquefied petroleum gas, jet fuel, and petroleum coke.

underlying assumptions, grid resolutions, and results, are discussed more fully by Gilmore et al.[8]

For EASIUR,[17] S−R matrices have also been built,[18] and matrices corresponding to annual average changes in $PM_{2.5}$ concentrations arising from changes in emissions were used here, using the same emissions, population data, and concentration−response relationship that were used for InMAP. EASIUR estimates $PM_{2.5}$ concentrations from the same pollutants as InMAP but does not include SOA from NMVOC emissions.[8] Rather than leave SOA as missing in the EASIUR results underlying Tables S1 and S2, and to facilitate comparisons among model results, the deaths attributed to SOA, as predicted by InMAP and AP2, were averaged and added to EASIUR totals. The proportion of deaths attributable to SOA is 11% for InMAP, which estimates SOA formation using equilibrium partitioning coefficients,[10] and 19% for AP2, which uses fractional aerosol yield coefficients.[8] Air quality models tend to underpredict SOA,[19] suggesting its importance, though highlighted here, may nevertheless be underestimated.

For AP2, raw county-level emissions from NEI 2014 version 1 were multiplied by the marginal damages by county[20] (Data set S1). The concentration−response relationship underlying the marginal damages is that described by Krewski et al.,[21] which was fitted to the same cohort as the concentration−response relationship underlying InMAP and EASIUR results but has a different functional form. Further discussion about how the choice between these two concentration−response relationships affects results derived from InMAP is given in ref 7.

Results for all three models are listed in Table S1, and code is provided in Data set S1 to reproduce all results. Conclusions, including insights 1−4 below, are supported across all of the models. In particular, the five activities to which the most deaths are attributed are consistent across the choice of models. Table S2 provides the root-mean-square errors and correlation coefficients between the InMAP results and the results from AP2 and EASIUR (with SOA averaged from InMAP and AP2 and added to the EASIUR results), across sectors, activities, processes, and pollutants.

RESULTS AND DISCUSSION

We find 100000 deaths each year (model range of 88000−107000) are attributable to human-caused emissions in the United States. For comparison, in previous work that used the same methods and data sources, 29000 deaths each year are attributable to U.S. emissions from wildfire and biogenic sources and 3000 deaths to emissions from Canada and Mexico.[1] Our estimates are consistent with the range of total deaths using a similar health impact estimation.[2] Four key insights into the current state of air pollution-related deaths in the United States emerge from our analysis.

(1) All **sectors** are major contributors to excess mortality risk, not just those dominated by tailpipes and smokestacks (Figure 1). Thousands more deaths each year are attributable

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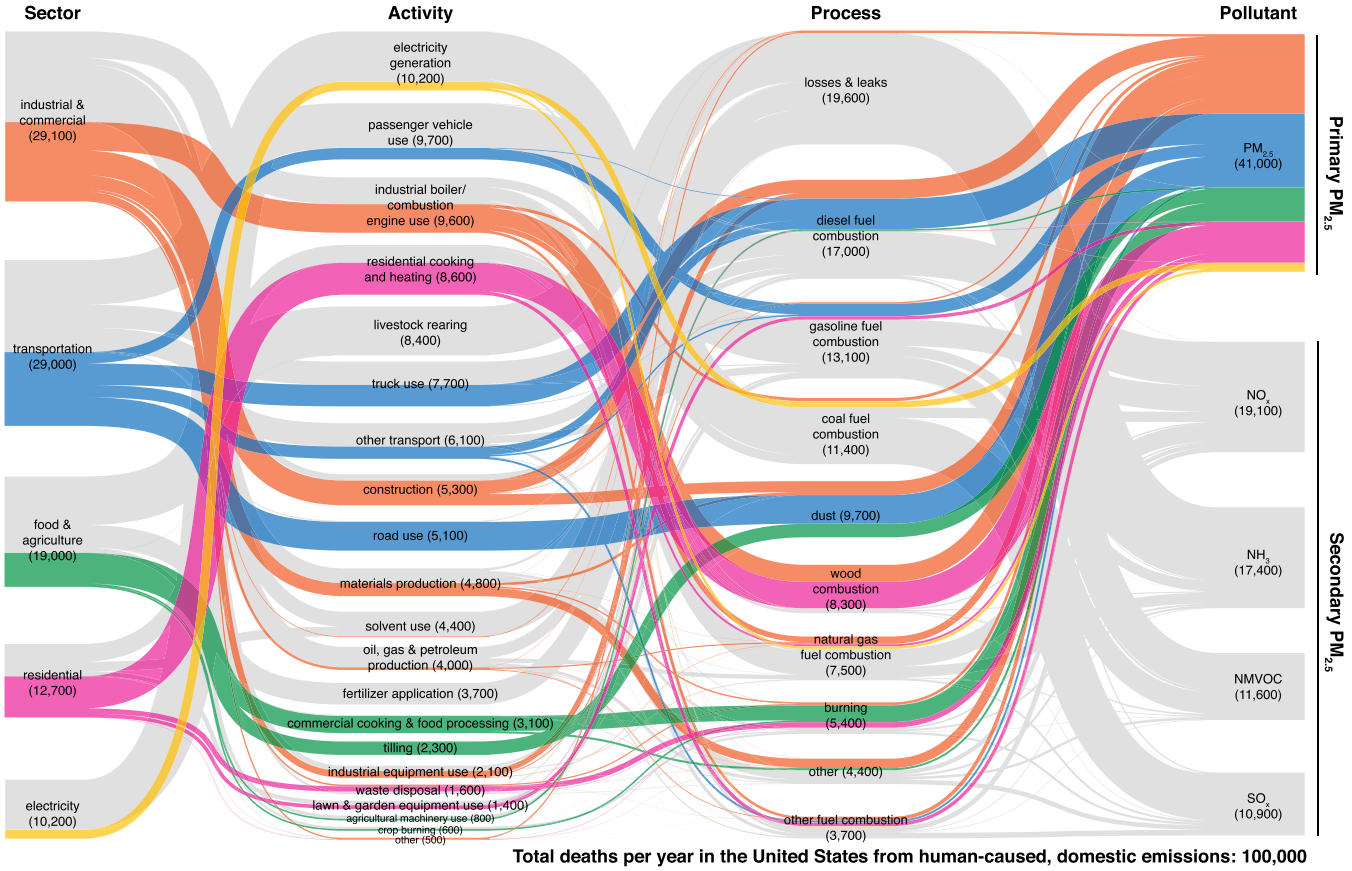

Figure 4. Deaths per year attributed to all human-caused emissions in the United States, grouped as in Figure 1. Flows are colored by sector, as in Figure 1, for primary $PM_{2.5}$ emissions. Flows colored gray represent deaths attributed to emissions of secondary $PM_{2.5}$ precursors NO_x, NH_3, NMVOC, and SO_x.

to the food and agriculture sector than to the electricity sector. Residential emissions are also of major importance.

(2) However, half of air pollution-related deaths are attributable to just five **activities** (Figure 2). These are electricity generation, passenger vehicle use, industrial boiler and combustion engine use, residential cooking and heating, and livestock rearing. These activities are all in different sectors, demonstrating the need for economy-wide strategies.

(3) Approximately half of the deaths are attributable to fossil fuel combustion, but other **processes** also matter (Figure 3). The other half of the deaths are roughly evenly split three ways among non-fossil fuel combustion (e.g., wood), agricultural noncombustion emissions (e.g., NH_3 from fertilizer and livestock), and other noncombustion emissions (e.g., road dust).

(4) Both primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors are important **pollutants**, including secondary $PM_{2.5}$ precursors that have not been heavily regulated (Figure 4). Around a fifth of the deaths are attributable to NH_3, which has been largely unregulated, despite analyses showing that emission controls are cost-effective.[22] NMVOC emissions regulation has been limited, generally only concerned with the hazards of direct NMVOC inhalation, or their potential to increase the level of ozone formation, but NMVOC emissions are major contributors to excess mortality risk through secondary $PM_{2.5}$ formation, including $PM_{2.5}$ formed from low-volatility NMVOC emissions that are generally not addressed by existing ozone regulations.[23]

Overall, we find that coal-powered electricity generation and passenger vehicle use remain among the most important targets for reducing deaths, even after decades of regulation have drastically reduced their emissions. Replacing coal combustion with low-emission electricity sources, such as solar and wind power, should therefore be pursued with continued urgency.[24] Large-scale electrification of the passenger vehicle fleet, with electricity provided from low-emission processes, coupled with incentives to reduce vehicle miles traveled, such as through investment in walkable neighborhoods, cycling infrastructure, and mass public transit, has the potential to further save many lives.[6]

Our analysis also suggests the potential for novel and emerging policies to reduce deaths from air pollution. These include but are not limited to (a) reducing deaths from livestock rearing, such as from manure management techniques that reduce NH_3 emissions,[25] or by substituting the production of animal-sourced foods with plant-sourced foods; (b) tightening emissions standards for nonroad diesel and gasoline combustion sources, including equipment use for construction, agriculture, industry, and residential lawn care and gardening, or switching to electric engines powered by electricity from low-emission processes; (c) reducing NMVOC emissions by improving formulations for residential and industrial solvents, such as cleaning supplies, paints, printing inks, and personal care products; and (d) encouraging more efficient technology, control devices, and fuel switching to reduce pollution from wood combustion, including switching to residential heaters powered by electricity from low-emission processes.

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The analysis here, which links emission sources to deaths rather than simply to mass of pollutants emitted or ambient concentrations formed, is an important step toward identifying promising strategies for improving public health by reducing air pollution. The effectiveness and viability of the strategies can be further investigated using information about the uncertainty in emissions inventories, abatement costs, the regional impacts of different emission sources, the logistical or political feasibility of different interventions, and other social costs and benefits. For example, many strategies for improving public health by reducing air pollution also reduce greenhouse gas emissions.[6,24−26] Detailed and extensive attribution of air pollution-related deaths to emission sources, such as in the analysis here, may be used to support climate policies and other environmental goals, alongside reductions in mortality from outdoor air pollution.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acs.estlett.0c00424.

Detailed information about emissions groups and model results (PDF)

Data set with code for generating results (ZIP)

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Notes

The authors declare no competing financial interest.

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■ REFERENCES

(1) Tessum, C. W.; Apte, J. S.; Goodkind, A. L.; Muller, N. Z.; Mullins, K. A.; Paolella, D. A.; Polasky, S.; Springer, N. P.; Thakrar, S. K.; Marshall, J. D.; et al. Inequity in consumption of goods and services adds to racial−ethnic disparities in air pollution exposure. *Proc. Natl. Acad. Sci. U. S. A.* **2019**, *116* (13), 6001−6006.

(2) Burnett, R.; Chen, H.; Szyszkowicz, M.; Fann, N.; Hubbell, B.; Pope, C. A., III; Apte, J. S.; Brauer, M.; Cohen, A.; Weichenthal, S.; et al. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. *Proc. Natl. Acad. Sci. U. S. A.* **2018**, *115* (38), 9592−9597.

(3) Institute for Health Metrics and Evaluation. Global Burden of Disease compare. https://vizhub.healthdata.org/gbd-compare/ (accessed 2020-01-19).

(4) National Research Council. *Air quality management in the United States*; National Academies Press, 2004.

(5) Lelieveld, J.; Evans, J. S.; Fnais, M.; Giannadaki, D.; Pozzer, A. The contribution of outdoor air pollution sources to premature mortality on a global scale. *Nature* **2015**, *525* (7569), 367−371.

(6) Tessum, C. W.; Hill, J. D.; Marshall, J. D. Life cycle air quality impacts of conventional and alternative light-duty transportation in the United States. *Proc. Natl. Acad. Sci. U. S. A.* **2014**, *111* (52), 18490−18495.

(7) Goodkind, A. L.; Tessum, C. W.; Coggins, J. S.; Hill, J. D.; Marshall, J. D. Fine-scale damage estimates of particulate matter air pollution reveal opportunities for location-specific mitigation of emissions. *Proc. Natl. Acad. Sci. U. S. A.* **2019**, *116* (18), 8775−8780.
(8) Gilmore, E. A.; Heo, J.; Muller, N. Z.; Tessum, C. W.; Hill, J. D.; Marshall, J. D.; Adams, P. J. An inter-comparison of air quality social cost estimates from reduced-complexity models. *Environ. Res. Lett.* **2019**, *14* (7), No. 074016.
(9) Landrigan, P. J.; Fuller, R.; Acosta, J. R.; Adeyi, O.; Arnold, R.; Basu, N.; Baldé, A. B.; Bertollini, R.; Bose-O'Reilly, S.; Boufford, J. I.; et al. The Lancet Commission on pollution and health. *Lancet* **2018**, *391* (10119), 462−512.
(10) Tessum, C. W.; Hill, J. D.; Marshall, J. D. InMAP: A model for air pollution interventions. *PLoS One* **2017**, *12* (4), No. e0176131.
(11) Nasari, M. M.; Szyszkowicz, M.; Chen, H.; Crouse, D.; Turner, M. C.; Jerrett, M.; Pope, C. A., III; Hubbell, B.; Fann, N.; Cohen, A.; et al. A class of non-linear exposure-response models suitable for health impact assessment applicable to large cohort studies of ambient air pollution. *Air Qual., Atmos. Health* **2016**, *9* (8), 961−972.
(12) Pope, C. A., III; Lefler, J. S.; Ezzati, M.; Higbee, J. D.; Marshall, J. D.; Kim, S. Y.; Bechle, M. J.; Gilliat, K. S.; Vernon, S. E.; Robinson, A. L.; et al. Mortality risk and fine particulate air pollution in a large, representative cohort of U.S. adults. *Environ. Health Perspect.* **2019**, *127* (7), No. 077007.
(13) Pope, C. A., III; Coleman, N.; Pond, Z. A.; Burnett, R. T. Fine particulate air pollution and human mortality: 25+ years of cohort studies. *Environ. Res.* **2020**, *183*, 108924.
(14) Goldman, G. T.; Dominici, F. Don't abandon evidence and process on air pollution policy. *Science* **2019**, *363* (6434), 1398−1400.
(15) Deryugina, T.; Heutel, G.; Miller, N. H.; Molitor, D.; Reif, J. The mortality and medical costs of air pollution: evidence from changes in wind direction. *Am. Econ. Rev.* **2019**, *109* (12), 4178−4219.
(16) Mauri, M.; Elli, T.; Caviglia, G.; Uboldi, G.; Azzi, M. RAWGraphs: a visualisation platform to create open outputs. In *Proceedings of the 12th Biannual Conference on Italian SIGCHI Chapter*; Paternò, F., Spano, L. D., Ardito, C., Santoro, C., Eds.; Association for Computing Machinery, 2017; Vol. *28*, pp 1−5.
(17) Heo, J.; Adams, P. J.; Gao, H. Reduced-form modeling of public health impacts of inorganic $PM_{2.5}$ and precursor emissions. *Atmos. Environ.* **2016**, *137*, 80−89.
(18) Heo, J.; Adams, P. J.; Gao, H. O. Public health costs accounting of inorganic $PM_{2.5}$ pollution in metropolitan areas of the United States using a risk-based source-receptor model. *Environ. Int.* **2017**, *106*, 119−126.
(19) Gao, Y.; Wang, H.; Zhang, X.; Jing, S. A.; Peng, Y.; Qiao, L.; Zhou, M.; Huang, D. D.; Wang, Q.; Li, X.; et al. Estimating secondary organic aerosol production from toluene photochemistry in a megacity of China. *Environ. Sci. Technol.* **2019**, *53* (15), 8664−8671.
(20) Muller, N. Z. Boosting GDP growth by accounting for the environment. *Science* **2014**, *345* (6199), 873−74.
(21) Krewski, D.; Jerrett, M.; Burnett, R. T.; Ma, R.; Hughes, E.; Shi, Y.; Turner, M. C.; Pope, C. A., III; Thurston, G.; Calle, E. E.; et al. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. *Res. Rep. - Health Eff. Inst.* **2009**, *140*, 5−136.
(22) Pinder, R. W.; Adams, P. J.; Pandis, S. N. Ammonia emission controls as a cost-effective strategy for reducing atmospheric particulate matter in the eastern United States. *Environ. Sci. Technol.* **2007**, *41* (2), 380−386.
(23) McDonald, B. C.; de Gouw, J. A.; Gilman, J. B.; Jathar, S. H.; Akherati, A.; Cappa, C. D.; Jimenez, J. L.; Lee-Taylor, J.; Hayes, P. L.; McKeen, S. A.; et al. Volatile chemical products emerging as largest petrochemical source of urban organic emissions. *Science* **2018**, *359* (6377), 760−764.
(24) Buonocore, J. J.; Luckow, P.; Norris, G.; Spengler, J. D.; Biewald, B.; Fisher, J.; Levy, J. I. Health and climate benefits of different energy-efficiency and renewable energy choices. *Nat. Clim. Change* **2016**, *6* (1), 100−105.
(25) Petersen, S. O.; Sommer, S. G. Ammonia and nitrous oxide interactions: roles of manure organic matter management. *Anim. Feed Sci. Technol.* **2011**, *166*, 503−513.
(26) Shindell, D.; Faluvegi, G.; Seltzer, K.; Shindell, C. Quantified, localized health benefits of accelerated carbon dioxide emissions reductions. *Nat. Clim. Change* **2018**, *8* (4), 291−295.

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