Article

Premature mortality related to United States cross-state air pollution

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Outdoor air pollution adversely affects human health and is estimated to be responsible for five to ten per cent of the total annual premature mortality in the contiguous United States[1–3]. Combustion emissions from a variety of sources, such as power generation or road traffic, make a large contribution to harmful air pollutants such as ozone and fine particulate matter ($PM_{2.5}$)[4]. Efforts to mitigate air pollution have focused mainly on the relationship between local emission sources and local air quality[2]. Air quality can also be affected by distant emission sources, however, including emissions from neighbouring federal states[5,6]. This cross-state exchange of pollution poses additional regulatory challenges. Here we quantify the exchange of air pollution among the contiguous United States, and assess its impact on premature mortality that is linked to increased human exposure to $PM_{2.5}$ and ozone from seven emission sectors for 2005 to 2018. On average, we find that 41 to 53 per cent of air-quality-related premature mortality resulting from a state's emissions occurs outside that state. We also find variations in the cross-state contributions of different emission sectors and chemical species to premature mortality, and changes in these variations over time. Emissions from electric power generation have the greatest cross-state impacts as a fraction of their total impacts, whereas commercial/residential emissions have the smallest. However, reductions in emissions from electric power generation since 2005 have meant that, by 2018, cross-state premature mortality associated with the commercial/residential sector was twice that associated with power generation. In terms of the chemical species emitted, nitrogen oxides and sulfur dioxide emissions caused the most cross-state premature deaths in 2005, but by 2018 primary $PM_{2.5}$ emissions led to cross-state premature deaths equal to three times those associated with sulfur dioxide emissions. These reported shifts in emission sectors and emission species that contribute to premature mortality may help to guide improvements to air quality in the contiguous United States.

Long-term exposure to fine particulate matter ($PM_{2.5}$) and ozone leads to an increased risk of premature death[7–12]. Indeed, $PM_{2.5}$ and ozone are the most prominent known causes of early deaths associated with outdoor air pollution, resulting in more than 90% of total air-pollution-related mortalities[8,11]. For this reason, $PM_{2.5}$ and ozone have become the predominant pollutants for quantifying air quality[2]. These pollutants form mainly through atmospheric chemical reactions following the release of precursor emissions. $PM_{2.5}$, which consists of particles and liquid droplets, forms from gaseous precursor emissions of nitrogen oxides (NO_x), sulfur oxides (SO_x), ammonia (NH_3), and others. $PM_{2.5}$ can also be emitted directly, as in the case of black carbon. Ozone forms from gaseous precursor emissions of NO_x and volatile organic compounds (VOCs). The adverse health impacts due to exposure to $PM_{2.5}$ and ozone can therefore be attributed to the precursor emissions that lead to their formation. Such attribution is useful, as it is these emissions that can be directly controlled, rather than the exposure that results from them.

Combustion emissions constitute the largest source of anthropogenic emissions in the USA, and therefore contribute to the formation of $PM_{2.5}$ and ozone[2]. The health impacts attributable to these emissions have been estimated in various studies[6,13,14], with estimates varying between 90,000 and 360,000 early deaths per year. In the context of the Environmental Protection Agency (EPA) Cross-State Air Pollution Rule (CSAPR) and individual state regulation, measures to further reduce the health impacts of pollution would benefit from a greater understanding of which sectors and which states are responsible for the health impacts in every other state.

Prior studies have investigated parts of this problem. One study[6] estimated the sources of US $PM_{2.5}$ pollution impacts on a fine scale, with other work focusing on the roles of individual emission sectors[15]

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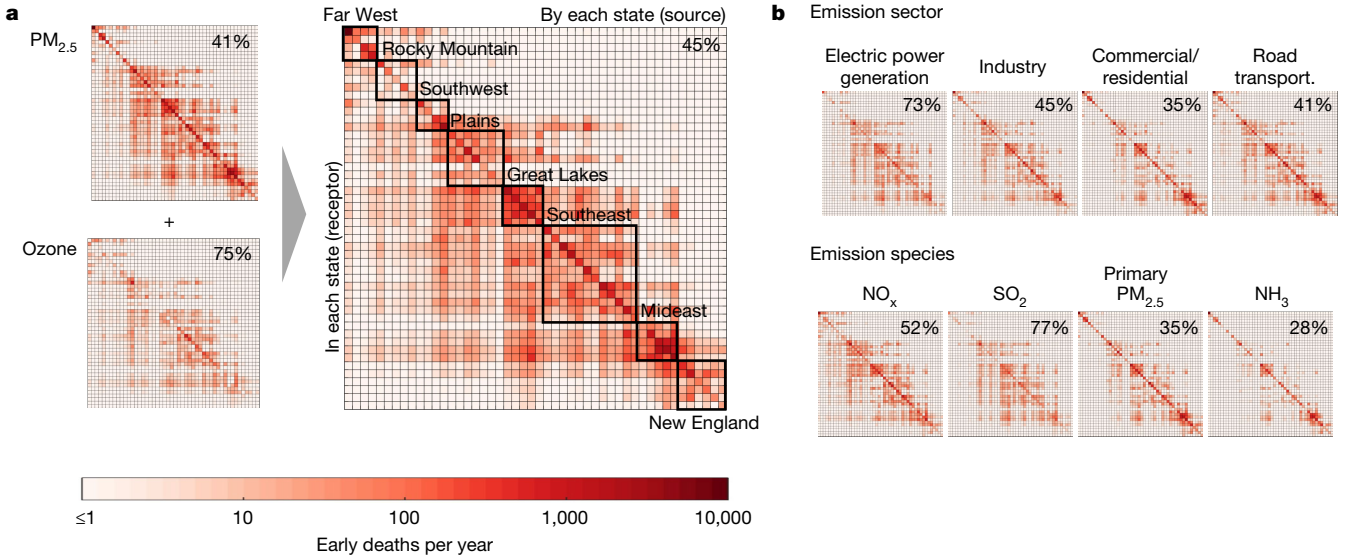

Fig. 1 | Early-death source–receptor matrices for 2011. a, Source–receptor matrix showing total early deaths per year for 48 × 48 states (right), and its breakdown into $PM_{2.5}$- and ozone-attributable impacts (left). **b**, Source–receptor early-death attribution to emission sectors (top) and emission species (bottom) that lead to the formation of $PM_{2.5}$ and/or ozone. States are grouped into US Bureau of Economic Analysis regions[24] and ordered west (left) to east (right) (ordering presented in Extended Data Fig. 1). Boxed percentages represent the fraction of impacts that occur out of the state that caused the corresponding emissions. Obtaining the summarized matrices shown using conventional approaches ('forward difference') would require 433-year-long simulations. Extended Data Fig. 2 presents corresponding matrices for 2005 and 2018.

or species[16,17] for either pollutant. Numerous other studies have focused on examining the roles of different emission sectors[13,18] or species[19,20], without quantifying the aspect of pollution exchange. Variations in time have also been discussed[21]. In all cases these studies have focused on only one or two of the dimensions of the problem (emission sector, emission species, pollutant and exchange), and no previous work has integrated these aspects together into a single study. As such, to date there has been no assessment of cross-state pollution exchange that quantifies the influence, by sector and chemical species, of each state on every other state's health risk, using detailed chemistry-transport modelling and including both $PM_{2.5}$ and ozone.

In this work, we estimate the pollution exchange between the 48 contiguous US states, and form source–receptor relationships between them for combustion emissions from seven sectors: electric power generation; industry; commercial/residential; road transportation; marine; rail; and aviation. The commercial/residential sector includes residential combustion (for example, of biomass), nonindustrial commercial and institutional processes, and waste treatment, among other sources. This analysis yields estimates for the number of early deaths due to $PM_{2.5}$ (primary and secondary, excluding secondary organic aerosols) and ozone exposure in every state, with attribution of impacts to each sector and each emitted chemical species from every state. We estimate combustion emissions for the seven sectors for 2005 (based on the 2005 National Emissions Inventory (NEI)), 2011 (based on NEI2011) and 2018 (based on the NEI2011 forecast), and present these findings in Extended Data Table 1. Lists of the specific sources that are grouped in each sector are included in the associated data repository (see Methods). The impacts of these emissions on each state's air quality are then quantified using receptor-oriented atmospheric sensitivities from the adjoint of the GEOS-Chem chemistry-transport model[22] (see Methods).

We calculate the pollution exchange between every state pair for the contiguous US for every combination of emission sector, $PM_{2.5}$ or ozone precursor emission species, and year. The 2011 source–receptor relations for the two pollutants and the total impacts are summarized in Fig. 1a. Matrices for different sectors and emission species are presented in Fig. 1b. Source–receptor matrices for all three years are presented in Extended Data Fig. 2.

The relative percentage of total impacts that occurred outside of the emitting state decreased with time, from 53% in 2005, to 45% in 2011 and 41% in 2018, meaning that there has been a declining relative magnitude of cross-state impacts. This fraction varies substantially between sectors. Electric power generation is the only sector that is regulated by the CSAPR, and has the highest out-of-state impacts as a fraction relative to in-state impacts: on average, approximately 70% of early deaths from this sector occur outside of the state that caused the emissions. However, with reductions in emissions from electric power generation, by 2018 there were 70% fewer out-of-state early deaths (approximately 13,000 fewer early deaths) by comparison with 2005. Road transportation, industry and commercial/residential emissions resulted in higher cross-state early deaths in 2018 than electric power generation (by 28%, 42% and 74% respectively), but are not regulated by the CSAPR at present. Although $PM_{2.5}$ and ozone impacts can vary by +125% to −65% depending on the specific choice of concentration-response function (see Methods), this disagreement does not affect the net pollution exchange between states and the impacts attributable to each sector.

The results presented in Fig. 1a, b reflect both $PM_{2.5}$- and ozone-attributable early deaths. Although the number of early deaths per additional unit of emission is approximately eight times higher for $PM_{2.5}$ than for ozone (not accounting for nonlinear interactions; see Methods), ozone impacts are typically transported farther. The fraction of $PM_{2.5}$ impacts that happen out of the state that caused them was approximately 41% for 2011, compared with approximately 75% for ozone for the same year. The full source–receptor matrices for each sector–year and species–year combination are included in the data repository (see Methods).

The fact that the source–receptor matrices, presented in Fig. 1, are not symmetric about the diagonal implies that there is a net imbalance in the exchange of early deaths between the US states. Figure 2 presents this exchange in terms of the air-quality-related early deaths per capita because of emissions from each state (Fig. 2a) and occurring within each state (Fig. 2b), as well as the net exchange between states (Fig. 2c). A positive value in Fig. 2c indicates that a given state is a net 'exporter' of early deaths—that is, that emissions in that state cause

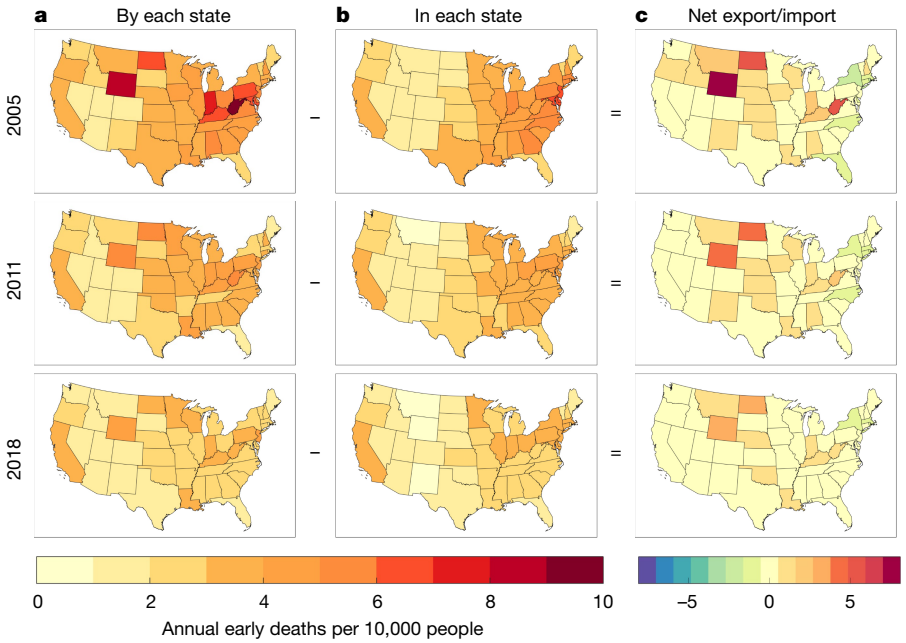

Fig. 2 | Total annual early deaths caused per 10,000 people for 2005, 2011 and 2018. The left plots (**a**) show the total aggregate early deaths caused by emissions in each state, divided by the population of the emitting state. The middle plots (**b**) show the total early deaths caused in each state, divided by the population of the state. The right plots (**c**) show the total early deaths exported by each state, divided by the population of the state (that is, the difference between plots **a** and **b**). These impacts are based on summed contributions from each emitted species (see Fig. 3).

more early deaths outside of the state, than are caused within that state by emissions from elsewhere. A negative value indicates the opposite: that the state is a net importer of early deaths.

Three broad patterns are visible. First, the largest exporters are in the northern midwest, owing to low local populations, high emissions, and large downwind populations. Wyoming was the highest exporter on a per capita basis in 2005, with North Dakota and West Virginia following. While these states remained some of the largest per-capita exporters in 2018, their exported impacts fell by roughly 50% over this period (see the examples in Extended Data Table 2). Second, a cluster of states in the northeast are consistent net importers of impacts. New York was the highest net importer of early deaths in all three years, on both a per-capita and an absolute basis. For 2011, the approximately 2,800 deaths incurred in New York because of New York emissions represent 60% of the total deaths caused by New York emissions, and approximately 40% of the total air-quality-attributable deaths in the state. This implies that around 60% of deaths in New York are imported from other states. Finally, states on the west coast have a net exchange of around 0, owing to a combination of no upwind emissions (attributable to any state), relatively sparse population downwind, and large local populations. We present examples of state-level sectoral contributions in Extended Data Figs. 3–5.

Figures 3a, b present the US-wide early-death impacts for each sector and each chemical species, respectively. Impacts from all sectors decrease over the studied period, with the exception of commercial/residential and aviation (landing and take-off only). Impacts due to commercial/residential emissions increase by 31% between 2005 and 2011, but remain steady (within approximately 5%) from 2011 to 2018. Aviation landing and take-off impacts increase by approximately 60% between 2005 and 2018, but contribute around 0.3% to the summed 2018 impacts. Impacts from electric power generation reduce from 22% of total summed impacts in 2005 to 11% in 2018. We estimate that reductions in emissions from electric power generation have led to around 15,900 avoided early deaths in 2018 and, interpolating linearly, to approximately 137,000 avoided early deaths integrated over the 14 years analysed here. Because of these changes, electric power generation changes from being the second most important emission sector to the fourth, while commercial/residential emissions go from fourth to first, responsible for 37% of the summed early deaths attributable to combustion emissions in 2018.

In terms of speciated impacts—that is, emissions species that contribute to the formation of, and exposure to, $PM_{2.5}$ and/or ozone—primary $PM_{2.5}$ emissions had the greatest impact in all three model years. They also stayed relatively consistent, with a 13% reduction in health impacts from 2005 to 2018. SO_2—which was the third-greatest contributor to impacts in 2005, making up 19% of the summed impacts—was contributing less than 6% by 2018. This was due to an approximately 80% reduction in SO_2 emissions.

Ammonia-attributable impacts increased by around 21% between 2005 and 2018. This difference was driven by an increase in the sensitivity of $PM_{2.5}$ exposure with respect to a unit of ammonia emissions between 2005 and 2011. Owing to the decline in the importance of SO_2, ammonia impacts went from being the fourth-greatest to the third-greatest contributor to total impacts over this period, increasingly close to the contribution of NO_x species. NO_x remained the second-greatest contributor to impacts from 2005 to 2018. Despite the roughly 50% reduction in total NO_x emissions between 2005 and 2018, impacts attributable to NO_x reduced by only around 35% between the two years. This is largely due to the increased sensitivity of $PM_{2.5}$ formation to NO_x emissions between 2005 and 2011, as noted previously[23].

On the basis of a linear combination of impacts by sector, we estimate US combustion emissions in 2005, 2011 and 2018 to have resulted in 111,200 (95% confidence interval 78,100–144,800), 93,700 (65,600–121,800) and 76,500 (53,300–99,600) early deaths, respectively. However, the total impact of all US anthropogenic emissions is different to the combined effect of each individual sector or species, owing to nonlinear interactions between the emitted chemicals (Fig. 3c). These interactions reduce the total impacts attributable to $PM_{2.5}$ by 30–34%. Impacts attributable to ozone instead increase by a factor of 2.4 to 2.8 (with the nonlinearity underlying this shown in Extended Data Fig. 6), raising the fraction of total early deaths attributable to ozone exposure from roughly 10% to around 30%.

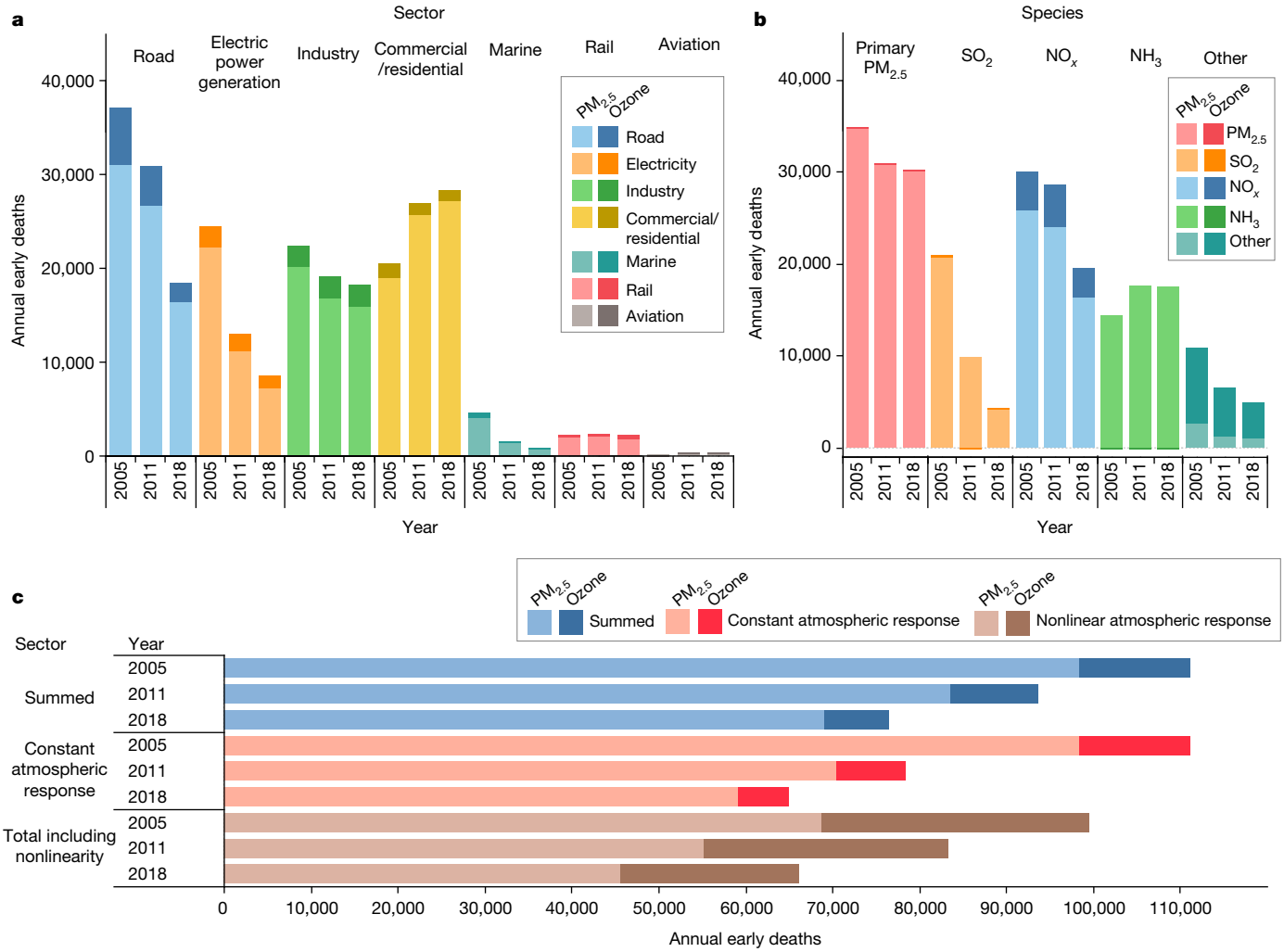

Fig. 3 | Total annual early deaths attributable to emission sector, emission species and in total. a, Total annual early deaths attributable to each emission sector. **b**, Total annual early deaths attributable to each emission species that leads to the formation of $PM_{2.5}$ and/or ozone. **c**, Total annual early deaths. Data are shown for 2005, 2011 and 2018, and for $PM_{2.5}$ and ozone. In **c**, three totals are presented: the sum of all sectors/species ('Summed'), which does not account for nonlinear interactions between species; the sum of all sectors/species with varying emissions, but constant (2005) atmosphere ('Constant atmospheric response'); and the total impacts after accounting for nonlinear interactions between species. Tabulated results are presented in Extended Data Table 3.

Taking these nonlinearity effects into account results in total US combustion emissions impacts of 96,600 (95% confidence interval 74,200–125,000), 83,300 (62,400–104,200) and 66,100 (49,300–82,900) early deaths for 2005, 2011 and 2018, respectively. This effect highlights the difference in expected changes in population exposure that result from marginal changes by comparison with larger-scale emissions increases or reductions. An explanation of this effect and its quantification is given in the Methods. The atmospheric nonlinearity is also reflected in our computed sensitivity differences between 2005 and 2011. Thus, a 1% reduction in 2011 emissions would lead to roughly 940 avoided early deaths. Had the atmospheric response to a unit of emissions remained constant between 2005 and 2011 (in terms of meteorology and background concentrations), the same emissions reduction would have led to around 780 avoided early deaths. The changing atmospheric composition thus increases the early deaths attributable to a unit of emission. These three effects are displayed in Fig. 3c.

Overall, we have found that more than 40% of the combustion-emissions-related early deaths cross state lines. This highlights the need for a cooperative approach between states for reaching air-quality targets or targeting problematic areas, as underlined by the introduction of EPA's CSAPR[5]. We find that the electric power generation sector is of declining importance to air quality, by comparison with the increasing importance of commercial/residential emissions. A 10% decrease in emissions from the commercial/residential sector would have 3.3 times greater benefit than a further 10% decrease in emissions from electric power generation. This is reflected in the declining relative importance of SO_2, and the increasing relative importance of primary $PM_{2.5}$ and ammonia. A 10% decrease in primary $PM_{2.5}$, NO_x and ammonia emissions would now have 7, 4.5 and 4 times the benefit, respectively, compared with a further 10% decrease in SO_2 emissions. These changing relative sectoral and speciated influences provide room to advance air-quality mitigation efforts in the US.

Online content

Any methods, additional references, Nature Research reporting summaries, source data, extended data, supplementary information, acknowledgements, peer review information; details of author contributions and competing interests; and statements of data and code availability are available at https://doi.org/10.1038/s41586-020-1983-8.

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Methods

We present here the data and models used in calculating the cross-state early-deaths caused by combustion emissions. We first estimate the speciated emissions for each combustion sector. We then use the adjoint of a chemistry-transport model to estimate the impact of changes in emissions on population exposure. Finally, we relate increases in population exposure to public health impacts (early deaths) using epidemiologically derived concentration-response functions. These steps, intercomparison of our results against existing literature, and the limitations of our approach are outlined below.

Combustion emissions

Emissions are attributed to each of the six nonaviation sectors in the US using SMOKE and the EPA National Emissions Inventory (NEI) for the year of 2005 (previously used in ref. [13]), 2011 (NEI2011v6 version 1) and the 2011-based forecast for 2018 (refs. [25–27]). These are generated on a 12 km × 12 km or 36 km × 36 km grid, and regridded to the 0.5° × 0.666° (latitude × longitude) grid of the nested GEOS-Chem adjoint model. The full list of individual sources (and corresponding EPA source classification code (SCC) identifiers) that comprise each sector are provided in the data repository noted in the Methods section 'Data and code availability'. For road transportation for 2011 and 2018, we use the EPA MOtor Vehicle Emissions Simulator (MOVES)-processed emissions[28]. For aviation emissions we use the Aviation Environmental Design Tool (AEDT) inventories for 2006, 2010, 2012 and 2015 (ref. [29]). When referring to each of 2005, 2011 and 2018 aviation impacts, we imply impacts from 2006, the average of 2010 and 2012, and 2015 emissions respectively, owing to the absence of more recent datasets. Only aviation emissions that occur within 1 km of the surface (landing and take-off emissions) are taken into account. These have been shown to capture roughly one-third of total aviation-emissions-attributable early deaths in the US[30,31]. We account for the underrepresentation of EPA's point-source oil and gas sector (pt_oilgas) in NEI2011v6 version 1, by distributing the underrepresented NO_x (the difference in pt_oilgas NO_x between version 3 and version 1) to the industry sector NO_x emissions on a state level, assuming the existing spatial distribution[27]. When calculating state source–receptor matrices for the marine sector, we only consider marine emissions within state boundaries and within, on average, around 25 km off the coast over the sea (where applicable). Besides the marine sector, which does not necessarily fall within state boundaries, we do not account for the impacts of emissions that occur outside of this domain and might contribute to US early deaths. Further details on emissions modelling are provided in the data repository.

Air-quality modelling

We use the adjoint of the GEOS-Chem chemistry-transport model[22] to calculate the sensitivities of the aggregate population exposure in each of the 48 contiguous US states with respect to the various emission species in the North American domain. The resolution of the horizontal grid is 0.5° × 0.666° (roughly 55 km × 55 km) (latitude × longitude), with 47 vertical layers up to 80 km. This horizontal resolution is adequate for capturing state-wide impacts[32–34]. Boundary conditions for the nested domain are obtained from the global GEOS-Chem model run at 4° × 5° resolution, driven by corresponding global meteorological data. Each of the 48 sensitivities quantifies the effect that any emission species in any location in the contiguous US and at any time will have on the population exposure to $PM_{2.5}$ or ozone in each corresponding state. We define $PM_{2.5}$ as the mass sum of nitrates, sulfates, ammonium, black carbon and organic carbon, capturing both primary and secondary $PM_{2.5}$ concentrations. Secondary organic aerosols are not captured. We perform an annual simulation for each of $PM_{2.5}$ and ozone state-level exposure, in each contiguous US state, for 2006 and 2011, resulting in 192 annual adjoint simulations in total (48 × 2 × 2). We use GEOS assimilated meteorological data from the Global Modelling and Assimilation Office (GMAO) at the NASA Goddard Space Flight Center. The year 2006 was climatologically warm in the US, with the annual average temperature being 0.55 °C higher than the 1995–2015 mean, whereas 2011 was climatologically average with an average temperature 0.04 °C lower than the 1995–2015 mean[35]. For 2018 we use the 2011 atmospheric response. Given the change between 2005 and 2011 (comparing the 'Summed' and 'Constant atmospheric response' in Fig. 3), we expect that this approximation will result in a maximum error of around 15% (as there were larger emissions changes between 2005 and 2011 than between 2011 and 2018). Total impacts across all sectors are calculated using additional 'forward' runs, described at the end of this section.

The GEOS-Chem baseline emissions are from EPA's NEI for 2005 and 2011 accordingly[26,27]. Previous studies have found that the NEI 2011 road transportation NO_x emissions are overestimated by around 50% in the southeast and nationally[36,37]. The effects of this are not included here as they are, as of the time of writing, not incorporated in EPA's NEI. An overestimation of 50% in the road transportation NO_x emissions in 2011 implies that results presented here overestimate road transportation early deaths by around 7,500 (95% confidence interval 5,200–9,700) early deaths per year. Other emissions sources, both natural and anthropogenic, are simulated using the standard GEOS-Chem nested North American domain datasets. The Electronic Data Gathering, Analysis and Retrieval (EDGAR) global anthropogenic emissions inventory drives the global model (from which the boundary conditions for the nested simulations are generated)[38]. This is replaced by regional emissions inventories where available (for example, NEI). Biogenic emissions are from the Model of Emissions of Gases and Aerosols from Nature (MEGAN) inventory[39], and lighting NO_x emissions are calculated on the basis of ref. [40].

We estimate the impacts of each sector by performing an inner (Hadamard) product of the sensitivities with the gridded emissions for each of the seven sectors, and calculate the corresponding population exposure impacts. This linear approach was used and validated in refs. [19,20,41–43] against the forward model difference method.

When calculating the total impacts from all sectors combined, we use a different approach to take into account nonlinear interactions between the sectors. Total impacts are calculated by comparing the surface concentrations in forward GEOS-Chem simulations with and without all US anthropogenic emissions. These forward model simulations allow us to quantify nonlinearity in the response of US air quality. Sets of seven forward simulations are conducted for both 2005 and 2011 to quantify this nonlinearity. Extended Data Fig. 6 shows how the simulated, population-weighted concentrations of ozone and $PM_{2.5}$ respond to large changes in emissions ('Average sensitivity'). Compared with the sensitivities used for single-sector and speciated impact calculations ('Marginal sensitivity'), the full, nonlinear $PM_{2.5}$ response to removal of all emissions is found to be 30–34% smaller, while the ozone response is found to be 2.4–2.8 times greater, implying greater nonlinearity effects for ozone by comparison with $PM_{2.5}$. This is because ozone sensitivities are larger when ozone concentrations are low, owing to the greater ozone-production efficiencies in a clean background atmosphere[44]. For $PM_{2.5}$, the response nonlinearity is driven by competition between SO_4 (from emitted SO_2) and NO_3 (from emitted NO_x) for ammonia[23,45].

Total impacts for 2018 are estimated by scaling the 2011 response. The scaling factor is calculated as the total growth in US population, multiplied by the ratio of the linearized response to 2018 and 2011 emissions.

Health impacts

We quantify air-quality impacts in terms of early deaths (premature mortalities). The toxicity of different $PM_{2.5}$ species is assumed to be equal, consistent with EPA practice. As with any study of air pollution impacts, our results are sensitive to the specific choice of concentration–response function (CRF). To calculate the effects of $PM_{2.5}$ exposure, we apply the American Cancer Society (ACS) cohort study

log-linear response estimate of 6% (range 4–8%) increased risk of all-cause mortality per 10 μg m^{-3} increase in annually averaged $PM_{2.5}$ exposure, derived for 1999–2000 exposures using the random-effects Cox model, and adjusted for 44 individual-level and 7 ecological covariates[7]. This estimate is linearized and applied here for adults over the age of 30 years old. This CRF has been applied in a number of estimates of US pollution impacts[46–48]; it is consistent with the results of a global meta-analysis of epidemiological literature, which also found a 6% (range 4–8%) increase in risk per 10 μg m^{-3} (ref. [9]).

Using a different risk estimate would result in a change in the total estimated impact. An expert elicitation performed by the EPA indicated a 1% (range 0.4–1.8%) increase in all-cause mortalities per 1 μg m^{-3} of exposure[2]. This would imply a roughly 70% increase in calculated early deaths, although all relative comparisons would remain the same. Another alternative based on the US medicare cohort would imply a roughly 18% increase in the calculated early deaths for $PM_{2.5}$, when applied to the same 30-plus population (again with all relative comparisons staying the same, but with the caveat that this was derived in a 65-plus cohort)[49]. Extended Data Table 4 shows how the estimate of total impacts, accounting for nonlinearity of the atmospheric response, is affected by the estimated relative risk, including the previously cited studies[2,7,12], refs. [49–51] and the results of a meta-analysis of epidemiological literature[9]. Although we cannot directly apply a nonlinear CRF, using the mean 2011 US concentration of $PM_{2.5}$ in the global exposure mortality model (GEMM)[12], we estimate a 35% increase in calculated early deaths.

For ozone, we apply the respiratory disease mortality CRF of ref. [52]; this is based on US exposure data from the same ACS study as above[7]. Impacts are calculated using the 8-hour maximum daily average ozone over the entire year, and applied to the same population. However, as with $PM_{2.5}$, there is disagreement regarding the correct exposure response curve to use. Extended Data Table 4 also includes estimates of ozone impacts, accounting for nonlinearity of the atmospheric response, using different ozone exposure response curves from the literature[50,52,53]. Using the all-cause mortality CRF of ref. [52] would result in a 110% increase in total mortality due to ozone exposure. Applying the all-cause mortality CRF of ref. [50] to quantify ozone health impacts would instead result in a roughly 17% increase in the reported early deaths due to ozone exposure. We note that the CRF of ref. [50] is based on mean summertime ozone exposure, whereas we measure annual-average exposure to 8-hour maximum ozone. However, ref. [52] showed that the response of respiratory mortality to chronic ozone exposure is similar when using either annual average (12% increase per 10 ppbv) or warm season (10% per 10 ppbv) exposure.

Population data are obtained from the global rural urban mapping project (GRUMP)[54] and LandScan[55] databases. For 2018, we scale the 2011 population to match the 2017 US Census totals[56]. State population fractions over the age of 30 years old are obtained from the US Census Bureau for 2011 (ref. [57]). The US baseline all-cause and respiratory disease incidence rates are obtained from the WHO for 2012 (ref. [58]). For both $PM_{2.5}$ and ozone, the early-deaths confidence intervals reflect the reported uncertainty range for the CRF. Uncertainty in the summed $PM_{2.5}$ and ozone impacts is calculated by performing a Monte Carlo simulation with 10^6 independent draws of each CRF, applying a triangular distribution to both.

Intercomparison with other studies

Pollution exchange on an intercontinental scale has previously been estimated for ozone[59–61], $PM_{2.5}$ (refs. [62–65]), and both[66], highlighting the influence of emissions from cross-continental sources. Regional studies have focused on individual species or species and pollutants—for example, the NO_x to ozone effect between EU countries[67] and between US states[17], sources of black-carbon impacts in parts of the US[16], and fine-scale monetized US $PM_{2.5}$ impacts of different sectors[6], in addition to other studies not using detailed chemistry-transport model (CTM) approaches.

The main contribution of our work is the breakdown of both air-pollution causes and impacts in the US, and there are no studies to which direct comparisons at the level of disaggregation in our work can be made. However, the aggregate results of this study compare well with those in the existing literature. Ref. [68] reports a roughly 25% decrease in $PM_{2.5}$-attributable early deaths in the US between 2005 and 2014, which is similar to the roughly 22% found here (interpolating for these two years). Our estimated total early deaths fall within the uncertainty ranges of recent studies, for example, the 79,300 (95% confidence interval 39,700–113,000) non-agriculture-related 2015 US early deaths reported in ref. [69]; the 88,400 (66,800–115,000) 2015 US $PM_{2.5}$-attributable early deaths reported in ref. [70]; and the central estimate of 107,000 total 2011 US $PM_{2.5}$-attributable deaths (of which around 85,600 correspond to non-agriculture- and non-fire-related deaths) reported in ref. [6]. As in these studies, our 2011 estimates are higher than the 2010 estimates of ref. [4] (around 37,400 US early deaths for non-natural and non-agriculture-related deaths). In addition, refs. [4,69] report different sectoral attributions, probably owing to the different emissions inventory used (EDGAR versus NEI). Our sectoral and speciated relative attribution is similar (for 2005) to that of ref. [19] (with the absolute values being different because of the different health-impacts function applied).

We also compare our estimated changes in population exposure to data obtained from monitor sites. We find that, between 2005 and 2011, the simulated population exposure to $PM_{2.5}$ and ozone (taking into account nonlinearities) fell by roughly 20% and 8.6% respectively. For the same two years, EPA's annual trends from nationwide monitor sites show a decrease of 24% and 8% for $PM_{2.5}$ and ozone concentrations respectively[71].

Limitations

In terms of air-quality modelling, even though the 0.5° × 0.666° (roughly 55 km × 55 km) (latitude × longitude) resolution is sufficient for capturing state-level regional impacts, it may underestimate primary $PM_{2.5}$ impacts and misrepresent ozone impacts in densely populated urban areas. This is in part due to the instantaneous dilution of the emissions, and, for ozone, to the highly nonlinear relationship between ozone formation and background VOC and NO_x concentrations. The EPA NEIs that are used here, and in policy assessments, are also only an approximation, with some known issues that we do not explicitly account for[36,37]. This could affect both the baseline calculation of the sensitivity and the absolute impacts attribution. In addition, the emissions presented for 2018 are forecasted from the NEI2011 inventory. Such forecasts are inherently uncertain[72–74]. Finally, previous studies have shown a tendency for GEOS-Chem simulations to overestimate nitrates[75,76]. This may result in artificially increased $PM_{2.5}$ formation in response to combustion emissions.

In estimating health impacts, the choice of CRF is critical for early-death calculations. Here we apply the all-cause CRF for $PM_{2.5}$ from the ACS cohort study[7] because of the large and nationally representative cohort it is based on, and because of its wide application in $PM_{2.5}$-attributable health-impact estimates in the literature. This CRF was derived for pre-2000 concentrations, and we thus assume no heterogeneity in effect estimates over time (as concentrations change). An analysis of the level of disagreement between different CRFs, and the effect on our estimated impacts, is presented in the 'Health impacts' section above.

We assume equal toxicity between different $PM_{2.5}$ species, consistent with EPA's practice. However, epidemiological work on differential toxicity has provided estimates for mortality predictors based on exposure to individual $PM_{2.5}$ constituents[77]. Sulfates and black carbon have specifically been highlighted because of their suspected higher toxicity amongst $PM_{2.5}$ constituents[9,78].

Here we choose to quantify all-cause and respiratory-disease mortality for long-term exposure to $PM_{2.5}$ and ozone respectively, but note that human exposure to $PM_{2.5}$ and ozone has been correlated with a variety of specific health endpoints, such as neurological diseases[79], various forms of cancer[80], low birth weight[81], and others. Short-term exposure to $PM_{2.5}$ and ozone has also been found to correlate causally with an increased likelihood of early death[82,83], and is not included here. Nonfatal (morbidity) effects attributable to $PM_{2.5}$ and ozone exposure—including acute respiratory symptoms, exacerbated asthma, days of work and school lost, upper and lower respiratory symptoms, nonfatal heart attacks, acute bronchitis, and hospital and emergency-department visits—are also not captured. In addition, given the aggregate nature of the adjoint objective function, we present results for the aggregate state-level population. Air-pollution-related health impacts, however, have been known to disproportionally affect different races, ages and socioeconomic backgrounds[84,85]. These are not broken down here.

We also note that this work quantifies the pollution exchange between the contiguous US states, and does not take into account sources outside of this domain (for example, Mexico, Canada and intercontinentally[65,86]). In addition, while changes in emissions are probably the largest driver of changes in the cross-state, sectoral and speciated patterns between the years, effects of meteorological changes can also contribute, and are not specifically decoupled here. Finally, for simultaneous, large changes in multiple pollutant emissions, there may be nonlinear interactions. These interactions could change the total impact relative to that calculated for individual sectors here, where independent changes are assumed. For this reason, and as discussed above, we calculate and present total impacts (aggregated across all sectors) using forward simulations in which all emissions are reduced simultaneously.

Data availability

The cross-state source–receptor matrices generated and analysed here, together with sector definitions, are available in the 4TU.ResearchData repository at https://doi.org/10.4121/uuid:edfc5304-39ed-4556-a95a-f8b3313f7cfc.

Code availability

The atmospheric modelling code used is publicly available; instructions for download are given at http://wiki.seas.harvard.edu/geos-chem/index.php/GEOS-Chem_Adjoint.

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Author contributions I.C.D. and S.R.H.B. planned the research. I.C.D. performed the emissions modelling and the air quality modelling $PM_{2.5}$ simulations. S.D.E. and E.M. performed the air quality modelling ozone simulations. I.C.D. and S.D.E. performed results analysis. I.C.D. drafted the manuscript with the help of S.D.E. and S.R.H.B. All authors provided feedback on the manuscript.

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Additional information

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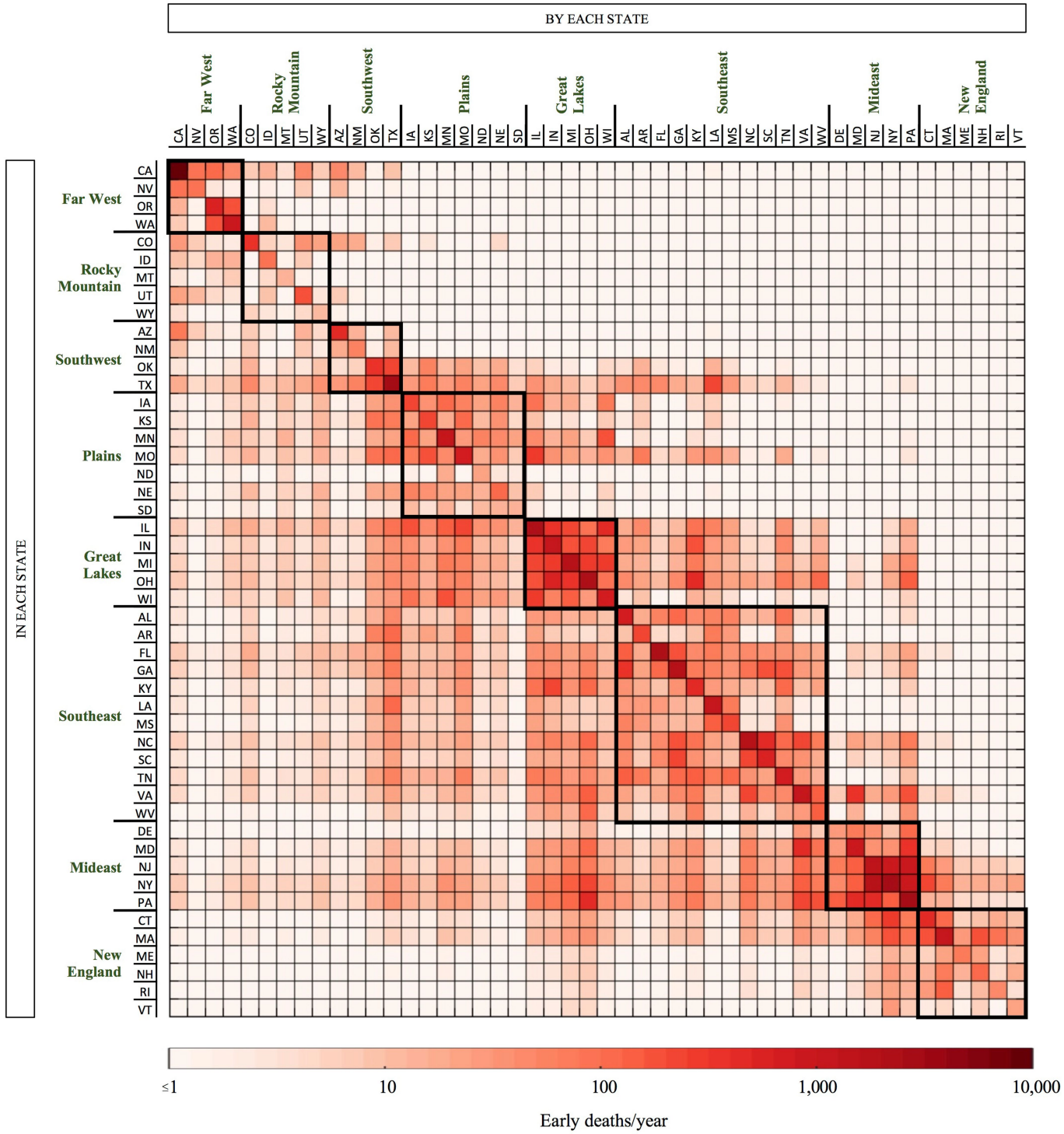
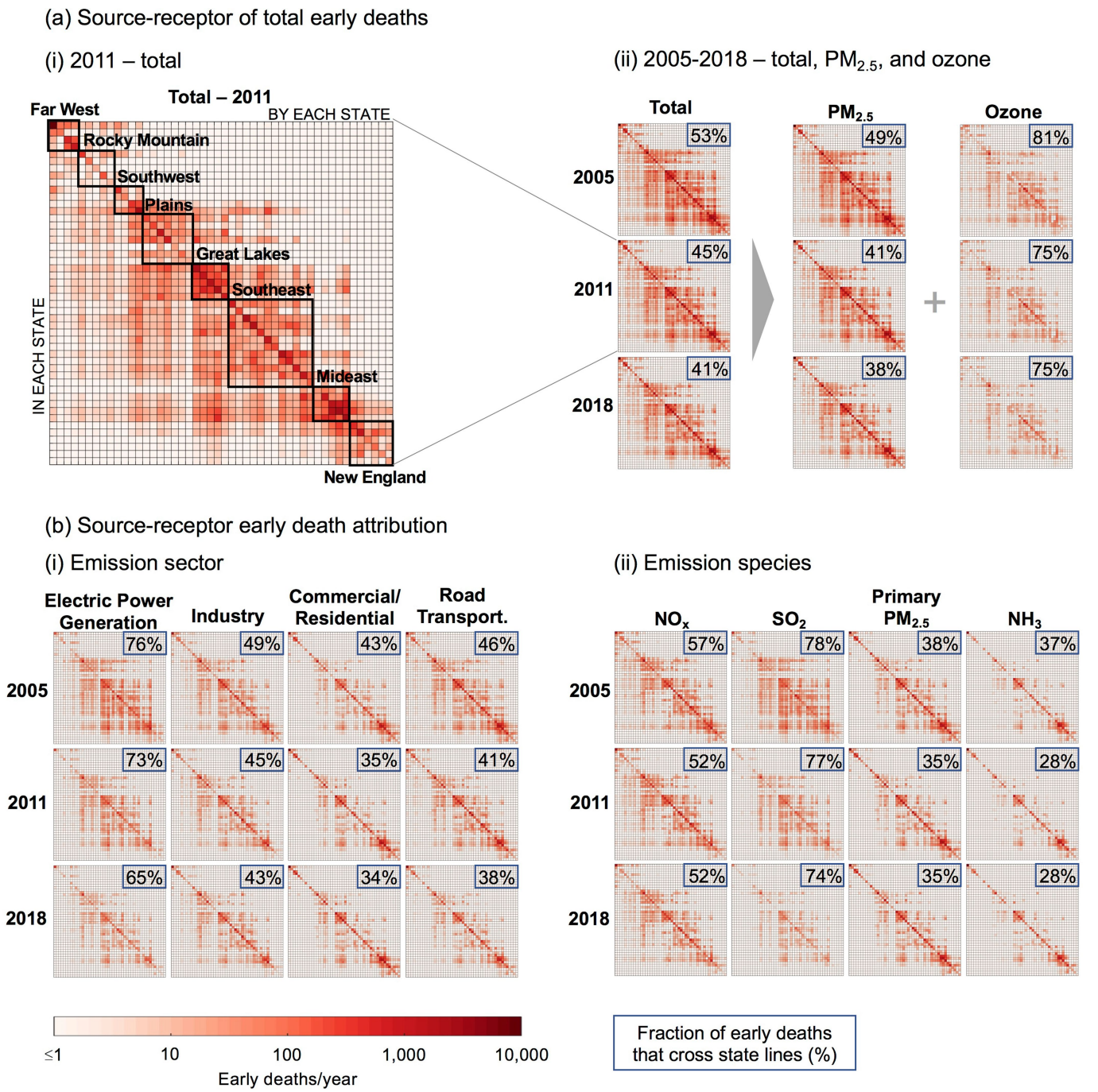
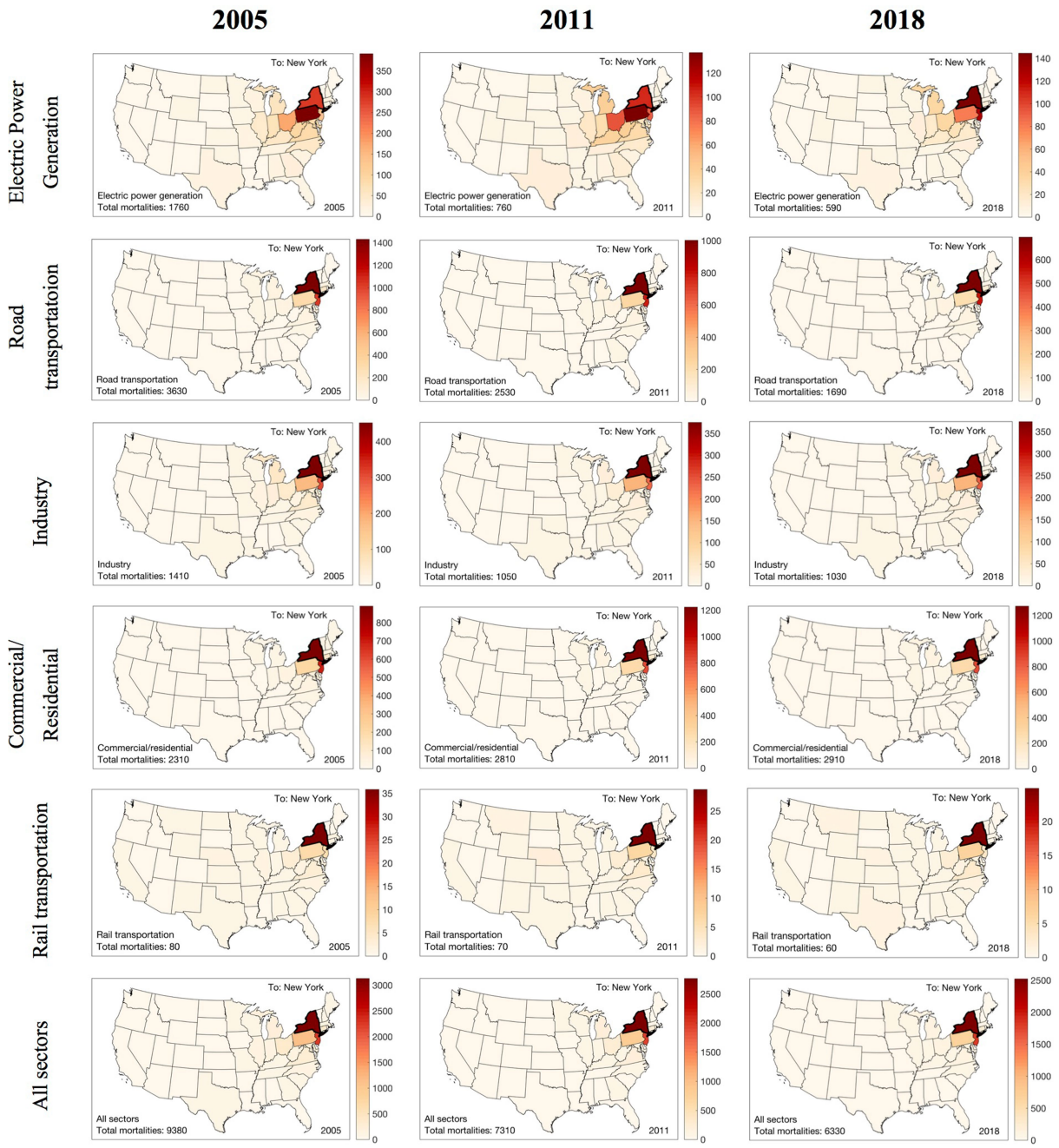


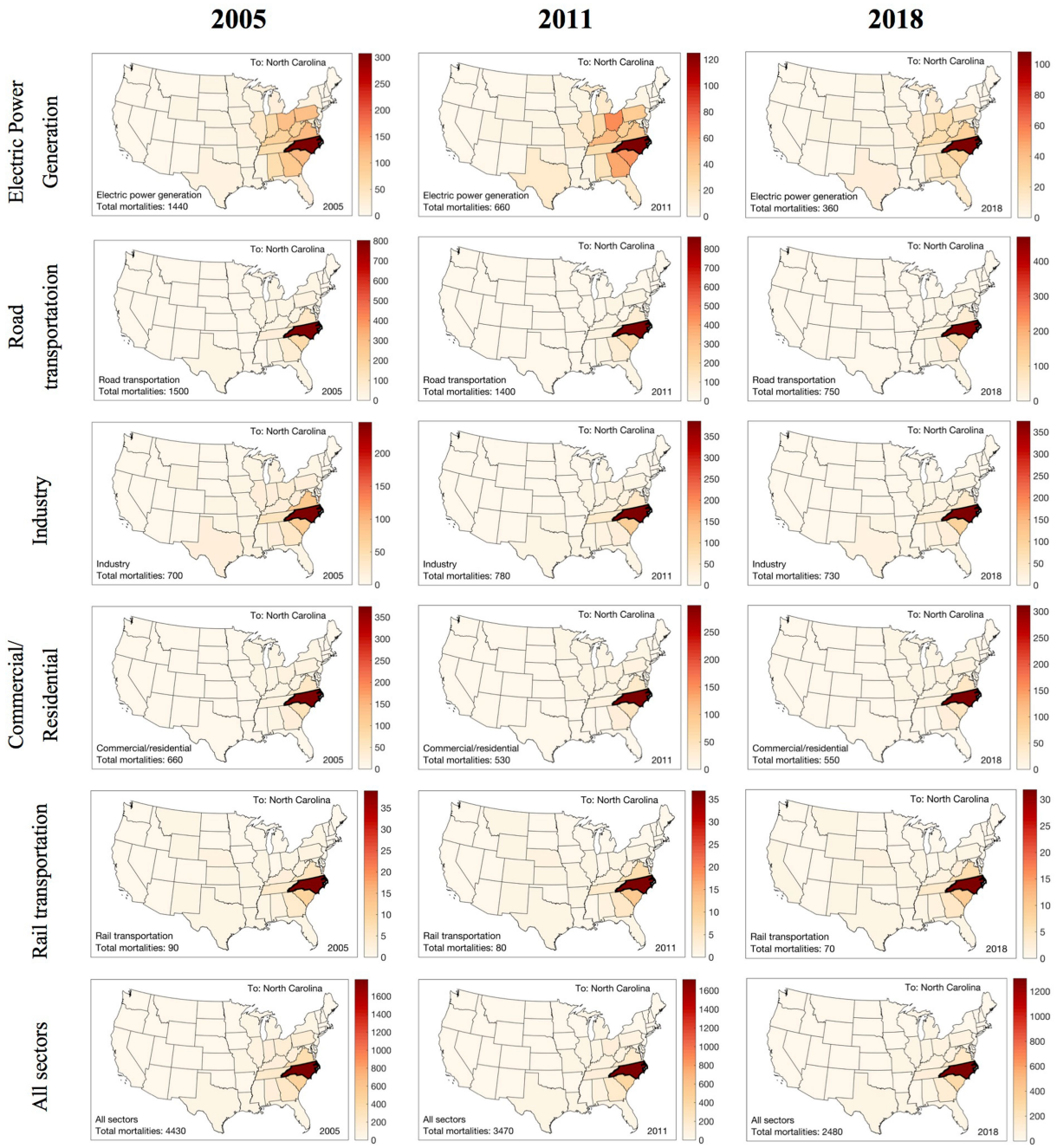
Extended Data Fig. 1 | Source–receptor matrix showing total impacts in 2011 for the contiguous US. 'By each state' indicates sources; 'in each state' indicates receptors. The matrix is annotated with state abbreviations and their regional grouping.



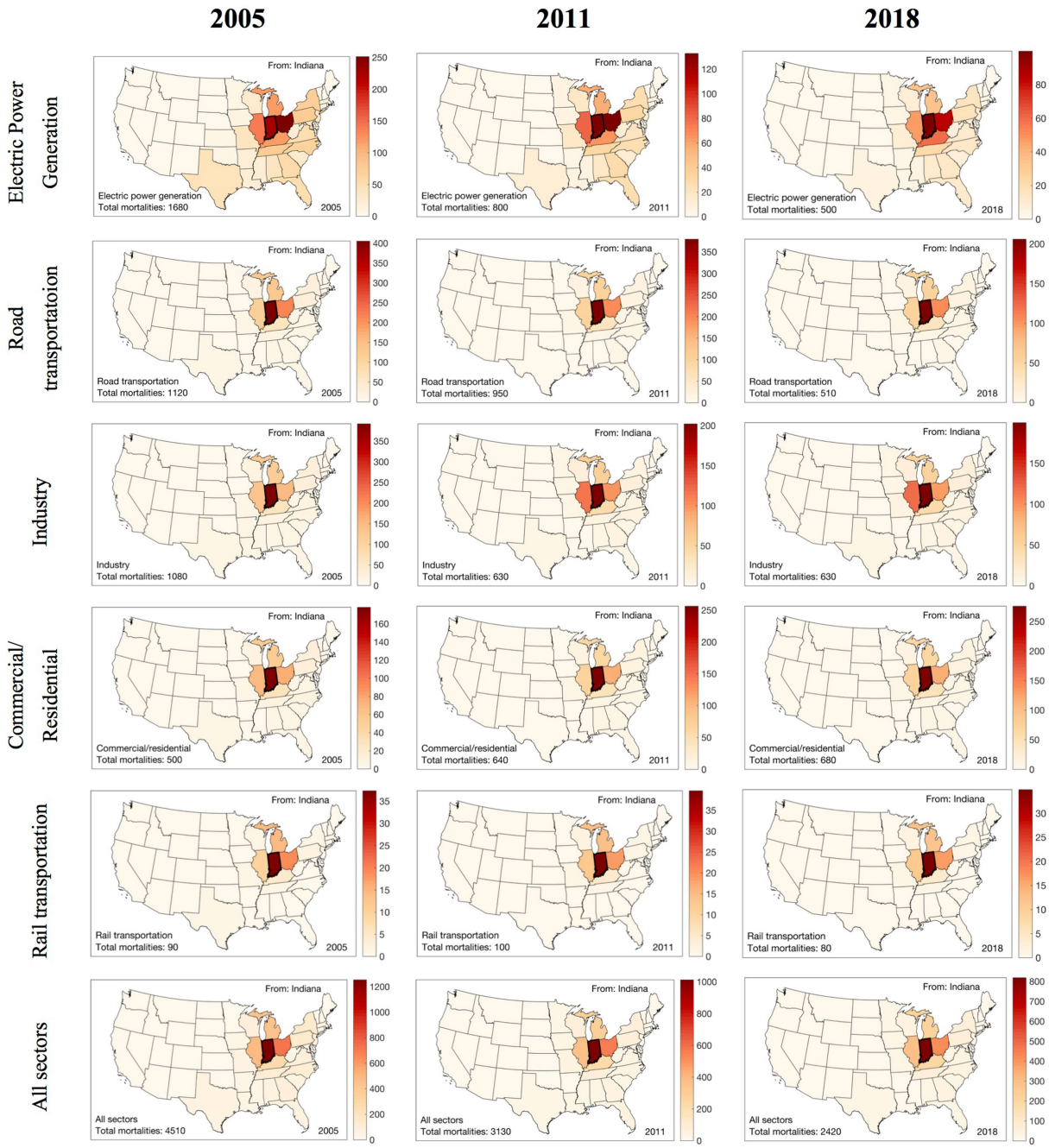
Extended Data Fig. 2 | Annual early-death source–receptor matrices for 2005, 2011 and 2018 for the contiguous US. Each matrix comprises 48 × 48 states. **a** (**i**), The total source–receptor matrix for 2011. **a** (**ii**), Its breakdown to $PM_{2.5}$-attributable and ozone-attributable impacts for all three years. **b**, Source–receptor early-death attribution to emission sectors (**i**) and emission species that lead to the formation of $PM_{2.5}$ and/or ozone (**ii**). States are grouped in regions defined by the Bureau of Economic Analysis[20] (labelled in **a**) and ordered from west (left) to east (right). The ordering of individual states is presented in Extended Data Fig. 1. Boxed percentages represent the fraction of impacts that occur out of the state that caused the corresponding emissions. We note that to obtain these summarized source–receptor matrices using conventional modelling approaches ('forward difference simulations') would have required around 1,300 simulations.



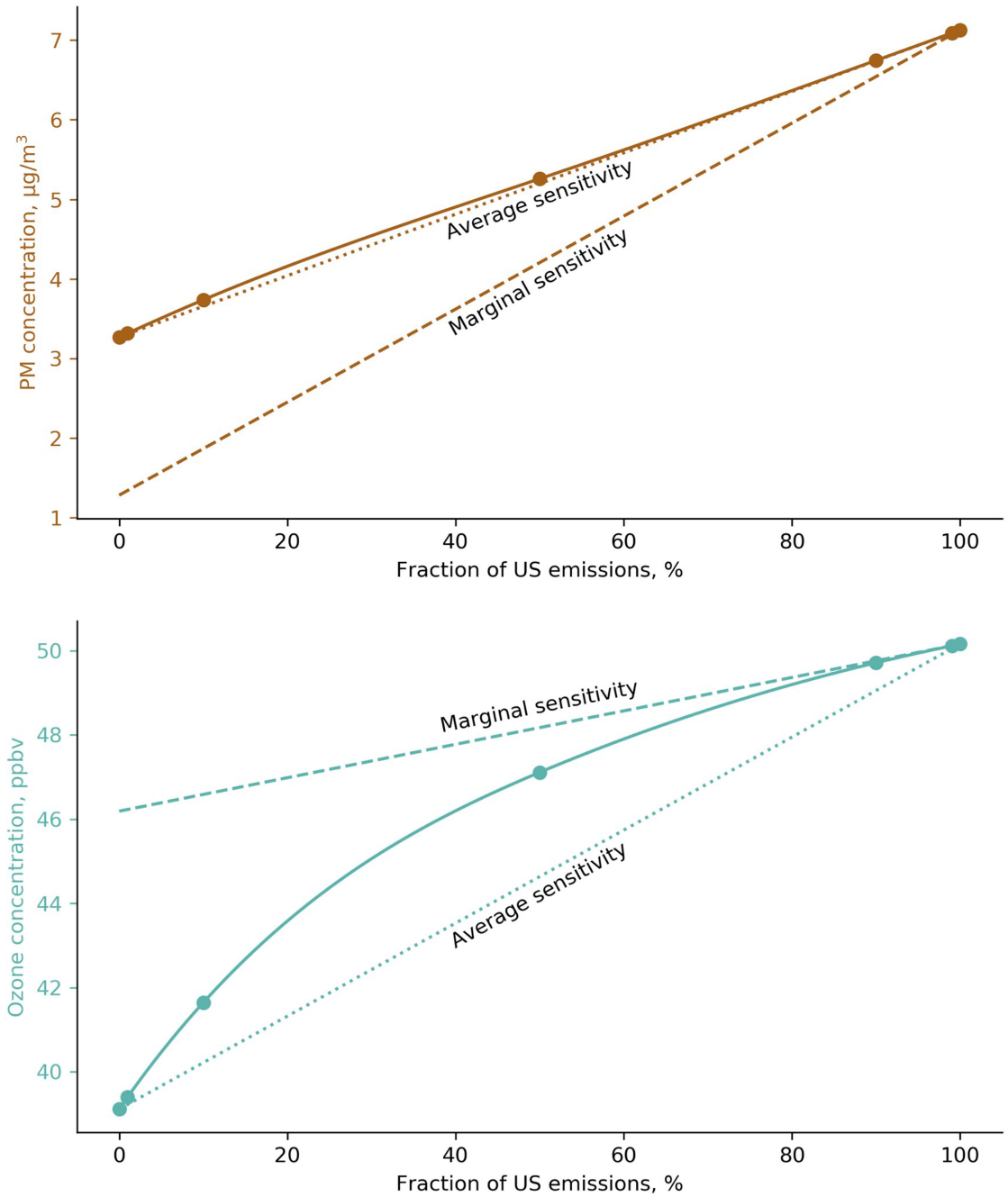
Extended Data Fig. 3 | Origins of New York annual early deaths, for 2005, 2011 and 2018, for five sectors and in total. Each state is coloured according to the annual early deaths that emissions from that state cause in the state of New York, for each sector–year combination. The total early deaths occurring in New York (that is, the sum of all states' values) for each sector–year combination is displayed at the bottom left of each panel.



Extended Data Fig. 4 | Origins of North Carolina annual early deaths, for 2005, 2011 and 2018, for five sectors and in total. Each state is coloured according to the annual early deaths that emissions from that state cause in the state of North Carolina, for each sector–year combination. The total early deaths occurring in North Carolina (the sum of all states' values) for each sector–year combination is displayed at the bottom left of each panel.



Extended Data Fig. 5 | Receptors of annual early deaths due to emissions in Indiana for 2005, 2011 and 2018, for five sectors and in total. Each state is coloured according to the annual early deaths that occur in that state because of emissions in Indiana, for each sector–year combination. The total early deaths caused by Indiana emissions (that is, the sum of all states' values) for each sector–year combination is displayed at the bottom left of each panel.



Extended Data Fig. 6 | Changes in the response of surface-population-weighted $PM_{2.5}$ and ozone concentrations to US emissions. Data points show the results of a series of forward simulations, in which the input conditions of the simulation (the total US anthropogenic emissions of all species) are reduced, joined by a cubic spline fit. The 'average sensitivity' lines indicate the gradient implied when impacts due to all sectors combined are calculated—that is, when the effects of atmospheric nonlinearity are taken into account—and thus the total results are scaled to match this. The 'marginal sensitivity' lines indicate the gradient of the response obtained by our GEOS-Chem adjoint simulation, and are used for calculations of individual sector and species impacts (where individual perturbations are of smaller size). The difference between the zero intercept of the two lines constitutes the 'interaction' effect. All values are population-weighted means for 2011.

Extended Data Table 1 | Primary $PM_{2.5}$, NO_x and SO_x emissions totals for 2005, 2011 and 2018

	2005				
	$PM_{2.5}$	NO_x	SO_x	NH_3	CO
Electric power generation	0.46	3.42	9.46	0.02	0.57
Industry	0.57	2.75	2.55	0.13	3.03
Commercial/ Residential	0.69	0.76	0.49	0.04	4.82
Road transportation	0.27	8.17	0.16	0.14	39.3
Marine	0.07	1.30	0.45	0.00	0.18
Rail	0.03	1.01	0.07	0.00	0.11
Aviation (LTO %)	0.003 (15%)	0.60 (13%)	0.06 (15%)	--	0.26 (44%)

	2011				
	$PM_{2.5}$	NO_x	SO_x	NH_3	CO
Electric power generation	0.18	1.78	4.10	0.02	0.69
Industry	0.41	2.52	1.20	0.09	2.49
Commercial/ Residential	0.62	0.61	0.22	0.12	3.67
Road transportation	0.19	5.10	0.03	0.11	23.1
Marine	0.02	0.60	0.05	0.00	0.11
Rail	0.02	0.77	0.01	0.00	0.12
Aviation (LTO %)	0.003 (13%)	0.58 (13%)	0.05 (13%)	--	0.17 (40%)

	2018				
	$PM_{2.5}$	NO_x	SO_x	NH_3	CO
Electric power generation	0.19	1.42	1.32	0.04	0.74
Industry	0.52	2.15	0.85	0.09	2.42
Commercial/ Residential	0.64	0.60	0.12	0.12	3.82
Road transportation	0.11	2.38	0.01	0.08	14.0
Marine	0.01	0.37	0.002	0.00	0.09
Rail	0.02	0.67	0.001	0.00	0.14
Aviation (LTO %)	0.003 (12%)	0.63 (12%)	0.05 (12%)	--	0.15 (33%)

Emissions expressed in teragrams per year for each sector for 2005, 2011 and 2018. Emissions for all sectors apart from aviation are derived from EPA's NEI. Aviation emissions are taken from the AEDT inventory[29] (for years 2006, 2010/2012 and 2015) and include emissions that occurred over the contiguous US. The percentages of aviation emissions that occur within around 1 km of altitude (landing and take-off emissions) are given in parentheses, and are the aviations emissions included in our analysis.

Extended Data Table 2 | Five states with the greatest reduction in annual early deaths between 2005 and 2018

By each state				In each state			
State	2005	2018	Δ(2005-18) (%)	State	2005	2018	Δ(2005-18) (%)
WV	1,740 [1,240-2,250]	745 [540-950]	1,000 (57%)	AL	2,080 [1,520-2,640]	990 [730-1,260]	1,080 (53%)
AL	2,640 [1,920-3,350]	1,280 [950-1,610]	1,350 (51%)	ME	330 [240-430]	170 [120-220]	160 (48%)
TN	2,370 [1,730-3,020]	1,210 [880-1,550]	1,160 (49%)	MS	970 [710-1,220]	510 [380-640]	460 (47%)
MD	3,240 [2,250-4,230]	1,660 [1,140-2,180]	1,580 (49%)	VA	3,760 [2,680-4,840]	2,010 [1,440-2,590]	1,750 (46%)
KY	2,750 [1,940-3,560]	1,430 [1,020-1,850]	1,320 (48%)	WV	790 [570-1,010]	420 [310-540]	370 (46%)

Data are given in terms of early deaths caused by emissions from each state and in each state. Values in square brackets show 95% confidence intervals.

Extended Data Table 3 | Early deaths attributable to each sector and species (that lead to $PM_{2.5}$ and/or ozone formation) for 2005, 2011 and 2018

(a)

Sector	2005	2011	2018
Electric power generation	24,400 [16,900-31,800]	12,800 [9,100-16,600]	8,500 [6,000-10,900]
Industry	22,400 [15,600-29,100]	19,100 [13,400-24,700]	18,200 [12,900-23,600]
Commercial/residential	20,400 [14,100-26,800]	26,800 [18,300-35,400]	28,200 [19,200-37,300]
Road transportation	37,000 [26,500-47,600]	30,800 [21,800-39,800]	18,400 [12,900-23,800]
Marine Transportation	4,600 [3,200-5,900]	1,500 [1,100-2,000]	810 [590-1,000]
Rail transportation	2,300 [1,600-2,900]	2,400 [1,700-3,100]	2,100 [1,500-2,800]
Aviation	130 [80-200]	210 [140-270]	220 [150-290]
Non-linear $PM_{2.5}$ interactions	*-29,500*	*-28,400*	*-23,500*
Non-linear ozone interactions	*+17,900*	*+18,000*	*+13,100*
Total	96,600 [74,200-125,000]	83,300 [62,400-104,200]	66,100 [49,300-82,900]

(b)

Species	2005	2011	2018
NO_x	30,000 [21,300-38,700]	28,600 [20,500-36,800]	19,600 [14,000-25,200]
SO_2	21,000 [14,100-28,000]	9,900 [6,600-13,100]	4,300 [2,900-5,800]
Primary $PM_{2.5}$	34,800 [23,200-46,400]	31,000 [20,600-41,300]	30,200 [20,100-40,200]
NH_3	14,400 [9,600-19,200]	16,700 [11,800-23,500]	17,400 [11,600-23,200]
Other	10,900 [8,000-13,800]	6,500 [4,700-8,300]	4,800 [3,500-6,200]
Non-linear $PM_{2.5}$ interactions	*-29,500*	*-28,400*	*-23,500*
Non-linear ozone interactions	*+17,900*	*+18,000*	*+13,100*
Total	96,600 [74,200-125,000]	83,300 [62,400-104,200]	66,100 [49,300-82,900]

Values in square brackets are 95% confidence intervals. Only the mean effect is reported for the nonlinear interaction terms.

Extended Data Table 4 | Alternative CRF application to 2011 early deaths for all sectors, for $PM_{2.5}$ and ozone

Pollutant	Study	Mortality end-points	Early deaths
$PM_{2.5}$	*Ref. 7*	*All-cause*	*55,200 [36,800-73,600]*
	Ref. 2	All-cause	94,300 [37,700-169,700]
	Ref. 50	All-cause	66,800 [64,900-68,600]
	Ref. 9	All-cause	55,200 [36,800-73,600]
	Ref. 49	All-cause	124,200 [62,100-195,100]
	Ref. 51	All-cause	55,200 [9,200-101,200]
	Ref. 12	NCD+LRI*	75,000 [65,500-85,300]**
	Ref. 7	Cardiopulmonary	38,600 [29,700-48,200]
	Ref. 9	Cardiovascular	31,200 [14,200-45,400]
	Ref. 49	Cardiovascular	69,100 [37,200-106,300]
	Ref. 51	Cardiovascular	87,500 [54,000-123,500]
Ozone***	*Ref. 52*	*Respiratory (MDA8, annual avg.)*	*28,100 [18,700-37,400]*
	Ref. 52	All-cause (MDA8, annual avg.)	59,500 [29,800-119,100]
	Ref. 50	All-cause (24-hr avg. warm season)	32,900 [29,900-35,900]
	Ref. 53	Respiratory (MDA1, warm season)	9,700 [2,400-16,300]

Atmospheric nonlinearity is taken into account. The CRFs used to calculate the estimates in the main text are shown in italics. As in the main text, we apply these to the 30-plus population, using corresponding data for disease-specific baseline incidence rates from the WHO for 2012. Uncertainty intervals (in square brackets) reflect the 95% confidence intervals for each CRF.

*The GEMM model health end-point is all nonaccidental deaths, almost all of which are due to noncommunicable diseases (NCDs) and lower respiratory infections (LRIs). We use the all-cause mortality incidence rate from the WHO, excluding all injury-related deaths.

**To estimate the early deaths from the GEMM model, we use the parameters provided in ref.12 for more than >25 years, excluding the Chinese male cohort study, and use the mean population-weighted concentration of $PM_{2.5}$ in the US to determine the local relative risk per unit increase in exposure. The uncertainty intervals here reflect one standard error in parameter θ of the model.

***Note that the different CRF studies compared here assume different measures of ozone exposure (annual mean 8-hour maximum in ref. [52]; warm-season (April–September) mean in ref. [50]; and warm-season 1-hour daily maximum in ref. [53]). We apply all of these using the annual mean 8-hour maximum ozone exposure. MDA8, maximum daily 8-hour average.