

Intercontinental Impacts of Ozone Pollution on Human Mortality

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Ozone exposure is associated with negative health impacts, including premature mortality. Observations and modeling studies demonstrate that emissions from one continent influence ozone air quality over other continents. We estimate the premature mortalities avoided from surface ozone decreases obtained via combined 20% reductions of anthropogenic nitrogen oxide, nonmethane volatile organic compound, and carbon monoxide emissions in North America (NA), East Asia (EA), South Asia (SA), and Europe (EU). We use estimates of ozone responses to these emission changes from several atmospheric chemical transport models combined with a health impact function. Foreign emission reductions contribute approximately 30%, 30%, 20%, and >50% of the mortalities avoided by reducing precursor emissions in all regions together in NA, EA, SA, and EU, respectively. Reducing emissions in NA and EU avoids more mortalities outside the source region than within, owing in part to larger populations in foreign regions. Lowering the global methane abundance by 20% reduces mortality most in SA, followed by EU, EA, and NA. For some source–receptor pairs, there is greater uncertainty in our estimated avoided mortalities associated with the modeled ozone responses to emission changes than with the health impact function parameters.

Introduction

Ground-level ozone (O₃) causes deleterious impacts to human health, including cardiovascular and respiratory mortality (e.g., 1–4). O₃ is photochemically produced in the troposphere by oxidation of methane (CH₄), nonmethane volatile organic compounds (NMVOCs), and carbon monoxide (CO) in the presence of nitrogen oxides (NO_x = NO + NO₂). Observations and modeling studies demonstrate that O₃ produced in polluted regions can be transported long distances and that transport of precursors can enhance O₃ production in remote regions, impacting air quality on a global scale (5, 6). Understanding the impacts of O₃ precursor emissions from one region on health in distant regions may inform future air pollution mitigation strategies.

The impacts of O₃ on human mortality are influenced by demographic characteristics, including population density and baseline mortality rates. We calculate the premature mortalities within the entire Northern Hemisphere (NH) and within four major industrial regions—North America (NA), East Asia (EA), South Asia (SA), and Europe (EU)—that could be avoided by decreasing O₃ precursor emissions in each region. We use results from the Task Force on Hemispheric Transport of Air Pollution (TF HTAP, www.htap.org) multimodel study which showed that surface O₃ in one region decreases following 20% reductions of anthropogenic NO_x, NMVOCs, and CO emissions in any of the foreign regions, and that the O₃ decrease is similar to that from a 20% reduction in anthropogenic CH₄ emissions in the same foreign region (5, 6). We estimate avoided premature mortalities using these simulated long-range O₃ responses and a health impact function.

Methods

The TFHTAP coordinated an effort to quantify source–receptor relationships for four regions (NA, EA, SA, and EU; Figure S1), using multiple chemical transport models (CTMs) (6). We use the resulting multimodel mean surface O₃ responses for 20% reductions in NO_x, NMVOC, and CO emissions in

each region (SR6) and a 20% reduction in the global CH₄ mixing ratio (SR2), relative to the base case (SR1). Fourteen models participated in the SR6 vs SR1 cases and 17 models participated in the SR2 vs SR1 case, all with horizontal resolution ranging from 5° × 5° to 1° × 1° (Table S1), meteorology and emissions from 2001, and a constant CH₄ mixing ratio (1760 ppb for base case). All models have fixed meteorology, and do not model changes in meteorology due to atmospheric composition. For the 20% reductions in NO_x, NMVOC, and CO emissions, we do not account for long-term changes in O₃ due to the resulting change in CH₄ (6–8), which is estimated to be small for this scenario (6). Compared with surface O₃ observations, the model ensemble mean captured seasonal cycles in the northern midlatitude regions, except for a 10–20 ppb summertime positive bias over the eastern United States (US) and Japan, which did not correlate with estimates of the O₃ response to foreign emission reductions. Surface O₃ concentrations (monthly averages) from the individual models are regridded to a common 0.5° × 0.5° grid, and the ensemble average concentration is calculated for each grid cell and perturbation scenario.

Following previous studies using one global CTM (9–12), avoided premature mortalities resulting from each perturbation scenario are calculated using a health impact function based on a log-linear relationship between O₃ concentration and relative risk (RR) (1). RR is used to calculate the attributable fraction (AF), the fraction of the disease burden attributable to the risk factor (eq 1). When RR > 1, O₃ exposure increases risk of mortality.

$$AF = \frac{RR - 1}{RR} = 1 - \exp^{-\beta\Delta X} \quad (1)$$

Here, β is the concentration–response factor (CRF) and ΔX is the change in O₃ concentration. AF is multiplied by the baseline mortality rate (y_0) and exposed population (Pop) to yield avoided mortalities due to the O₃ concentration change (eq 2).

$$\Delta Mort = y_0(1 - \exp^{-\beta\Delta X})Pop \quad (2)$$

We apply eq 2 in each grid cell for each month using the corresponding population and baseline mortality rates, and sum the results to yield annual avoided premature mortalities (“avoided mortalities”).

CRFs are from a daily time-series study of the average relative risk of mortality associated with short-term ambient O₃ concentrations in 95 US cities (1). For each 10 ppb increase in 24-h average O₃, total nonaccidental (includes cardiovascular and respiratory) mortality increased by 0.52% (95% posterior interval (PI), 0.27–0.77%) and cardiovascular and respiratory mortality in particular increased by 0.64% (95% PI, 0.31–0.98%). These values are relatively low compared with other studies in the US (13), and we assume that they are valid globally, as similar results have been demonstrated in Europe and in developing nations (13, 14). Compared with estimates of avoided nonaccidental mortalities, estimates of avoided cardiopulmonary mortalities may be less influenced by differences in mortality causes around the world. We present results for both cardiopulmonary and nonaccidental mortalities, but emphasize the cardiopulmonary results, which alone may underestimate the impact of decreased O₃ on mortality by excluding other causes of death potentially associated with O₃.

Epidemiology studies relating O₃ and mortality may be subject to confounding by correlated copollutants, weather, and other factors, such as demographics and health status. As recommended previously (15), we use data from Bell et al. (1), who controlled for known confounders, and examine the sensitivity of our results to RR estimates from meta-

TABLE 1. Population-Weighted Reduction in Annual Mean O₃ Concentration (ppb) in Receptor Regions Following 20% NO_x, NMVOC, and CO Emission Reductions in Each Source Region, Based on 2006 Population (billions, *italics*) (17)

source region	receptor region			
	NA	EA	SA	EU
	<i>(0.481)</i>	<i>(1.67)</i>	<i>(1.55)</i>	<i>(1.02)</i>
NA	0.960	0.183	0.149	0.349
EA	0.184	1.200	0.146	0.168
SA	0.059	0.112	1.890	0.066
EU	0.150	0.221	0.165	0.589

analyses of single-city time-series studies (2–4). While little evidence exists for a low-concentration threshold below which O₃ causes no adverse impacts (15), we calculate results with and without a low-concentration threshold of 35 ppb, as recommended for European analyses (16).

We map the gridded global population in 2006 (17, NH total is 5.71 billion) to the coarser grid of the multimodel mean O₃ concentrations. Baseline cardiopulmonary and nonaccidental mortality rates are from the World Health Organization for 14 world regions (18) and 66 countries (19). For countries with no available data, we back-calculate population-weighted average mortality rates from regional rates. We use a Geographic Information System to calculate area-weighted average rates for grid cells that overlap multiple countries and to calculate avoided mortalities within US borders.

Our estimates of O₃ responses are subject to uncertainty due to the coarse resolution of global models. While global models are currently necessary to estimate long-range transport, their coarse resolution influences O₃ production within the source region, and our results may differ from those of a fine-resolution regional model. Errors due to resolution are expected to be smaller for long-range transport, as changes in concentration are more spatially uniform (20). Errors for mortality estimates may be greatest within source regions, particularly in urban regions with strong population gradients.

Results

Regional NO_x, NMVOC, and CO Emission Reductions. For simultaneous 20% reductions of anthropogenic NO_x, NMVOC, and CO emissions in each of the four regions, the largest impact on the multimodel mean change in surface O₃ occurs in the “domestic” region (i.e., where emissions are reduced) (Table 1). We note that “domestic” impacts include transport between metropolitan regions, states, and neighboring nations. Emission reductions in SA yield the greatest disparity between the domestic and “foreign” (i.e., within the three regions outside of the source region) population-weighted O₃ response, with a ratio of the domestic change to the change in each foreign region of 17–32. EA emission reductions also cause a large domestic vs foreign difference (ratio of domestic to foreign response of 6.5–8.2), while this ratio is smaller for NA (2.8–6.4) and EU (2.7–3.9).

Regardless of where emission reductions occur, avoided mortalities are concentrated in highly populated areas (e.g., Northern India and China; Figure 1, left). The greatest rates of avoided mortalities per million people occur near the source region (Figure 1, right), except for EU, where NO_x reductions increase O₃ during the winter (6), increasing domestic mortalities. For each receptor region, reducing domestic emissions is more effective at decreasing mortalities than reducing emissions in any of the three foreign regions (Tables 2 and S2). In response to domestic emission reductions, more avoided mortalities are calculated in EU

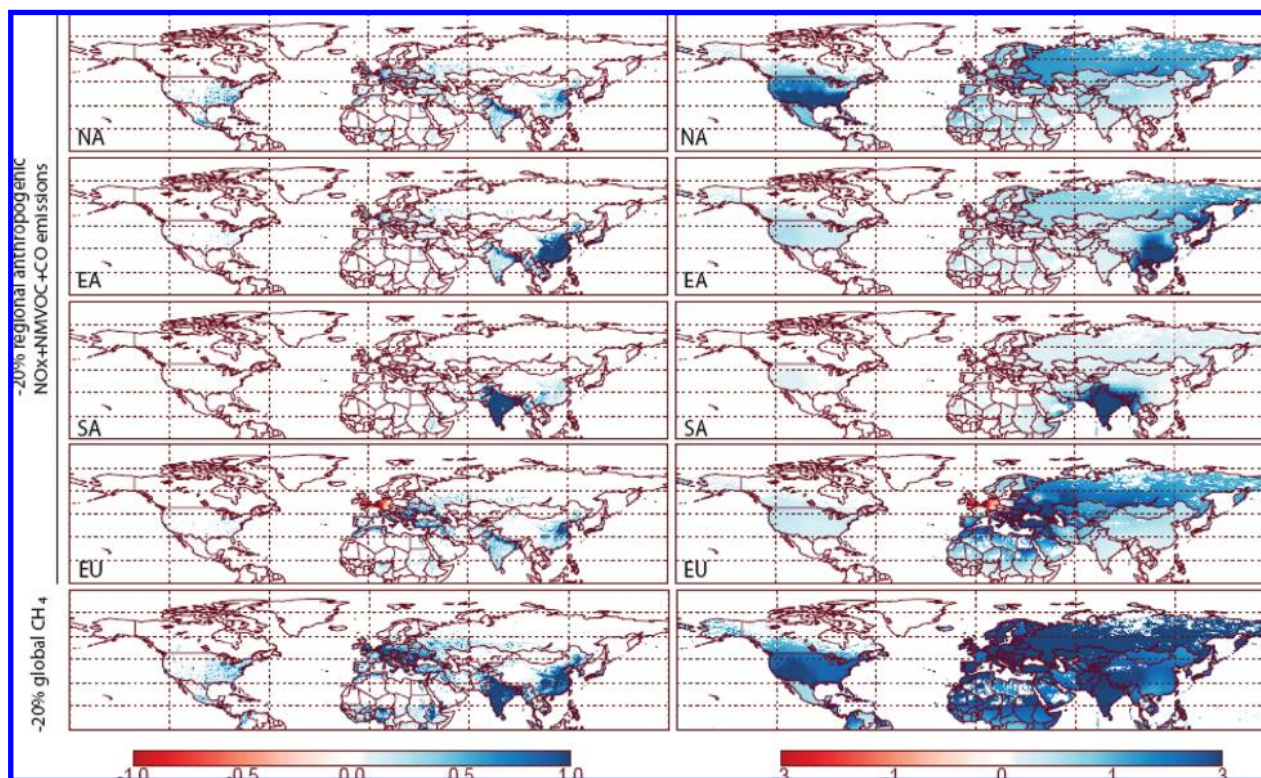


FIGURE 1. Annual avoided cardiopulmonary mortalities per 1000 km² (left) and per million people (right) resulting from 20% NO_x, NMVOC, and CO emission reductions in the region shown and a 20% global CH₄ mixing ratio reduction, assuming no low-concentration threshold.

TABLE 2. Annual Avoided Cardiopulmonary Mortalities (Hundreds) Following 20% NO_x, NMVOC, and CO Emission Reductions in Each Region, Assuming No Concentration Threshold (bold) and Assuming a Concentration Threshold of 35 ppb (normal font)^a

source region	receptor region				
	NA	EA	SA	EU	NH
NA	9 (4 – 13)	7 (3 – 10)	6 (3 – 9)	11 (5 – 17)	36 (18 – 55)
	9 (4 – 14)	4 (2 – 6)	5 (3 – 8)	6 (3 – 9)	27 (13 – 41)
EA	2 (1 – 3)	43 (21 – 66)	6 (3 – 9)	5 (3 – 8)	59 (29 – 91)
	1 (1 – 2)	40 (19 – 61)	5 (2 – 8)	3 (1 – 4)	49 (24 – 76)
SA	1 (0 – 1)	4 (2 – 6)	76 (37 – 117)	2 (1 – 3)	85 (41 – 130)
	0 (0 – 1)	3 (1 – 4)	66 (32 – 101)	1 (0 – 2)	71 (34 – 108)
EU	2 (1 – 3)	8 (4 – 12)	6 (3 – 10)	17 (8 – 26)	38 (18 – 58)
	1 (0 – 1)	6 (3 – 8)	6 (3 – 9)	25 (12 – 38)	40 (19 – 61)

^a Confidence intervals (95%) reflect uncertainty in the CRF only (1).

with a threshold than without, since the wintertime domestic mortality increase often occurs when O₃ concentrations are lower than 35 ppb.

Reducing anthropogenic precursor emissions by 20% in all regions together avoids 21,800 (95% confidence interval (CI), 10,600–33,400) cardiopulmonary mortalities in the NH annually, assuming no threshold (Table 2), corresponding to about 6% of global cardiopulmonary mortalities attributable to O₃ (21) and about 0.03% of mortalities of all causes globally. Avoided nonaccidental mortalities are about 1.7 times higher than the cardiopulmonary results (Table S2). Foreign emission reductions contribute about 30% of the total avoided mortalities in NA (63–72% of these in the US) and EA, 20% for SA, and >50% for EU, indicating that more mortalities would be avoided in EU by reducing emissions in the three foreign regions compared with reducing domestic emissions.

Focusing on the impacts of each source region, 64–76% of the total annual NH avoided mortalities following NA emission reductions occur outside of NA (the range reflects different causes of mortality and threshold assumptions).

Without a threshold, 55–58% of the total annual NH avoided mortalities following EU emission reductions occur outside of EU, though this conclusion reverses when a threshold of 35 ppb is applied (38–40% outside of EU). These findings agree with recent studies indicating that emission reductions in NA and EU have greater impacts on mortality outside the source region than within (11, 12). NA is the only region where reducing emissions avoids more mortalities in a foreign receptor region (EU) than domestically, reflecting higher population and baseline mortality rates in EU. However, this conclusion does not hold when a threshold is applied (Tables 2 and S2). Emission reductions in SA yield the most annual avoided mortalities overall, but influence foreign regions the least, (90% of the resulting NH avoided mortalities are in SA) due to its large population and minor influence of emissions on O₃ in the three foreign regions (Table 1). Emission reductions in EA also result in more avoided mortalities within the region (about 70% of the total NH avoided mortalities) than in the rest of the NH.

We analyze the sensitivity of the avoided nonaccidental mortality results to the uncertainty in the O₃ responses

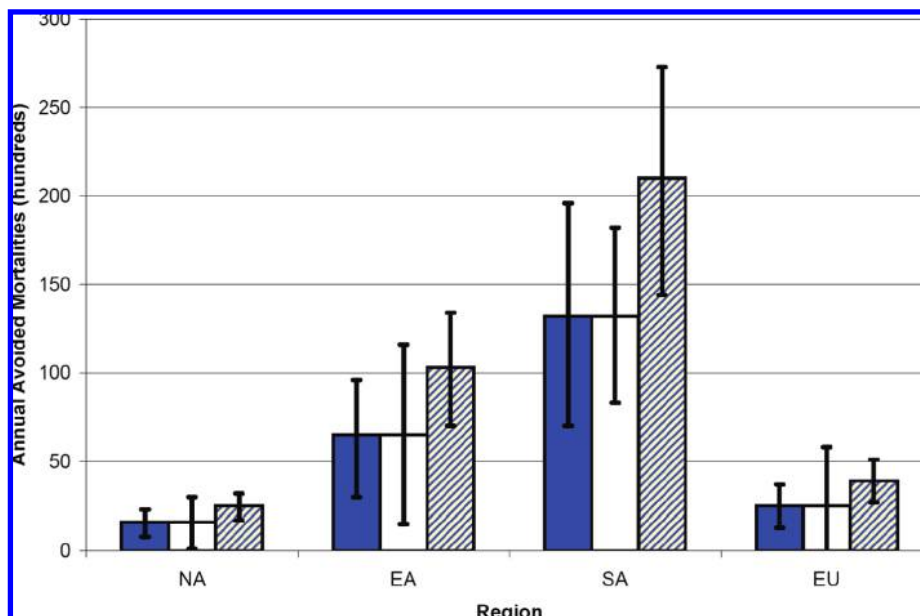


FIGURE 2. Annual avoided nonaccidental mortalities (hundreds) in each region from 20% NO_x, NMVOC, and CO emission reductions in the same region using the CRF and confidence interval (95%) from Bell et al. (1) (solid bars), using the CRF from Bell et al. (1) and confidence intervals (68%) from ± 1 standard deviation of the model ensemble O₃ perturbation in each grid cell (6) (white bars), and using the mean and confidence intervals (95%) of the CRFs from three meta-analyses of O₃ mortality (2–4) (striped bars). We convert the CRFs of Ito et al. (3) and Levy et al. (4) for 1-h maximum O₃ concentrations to 24-h mean using a ratio of 1-h maximum to 24-h mean equal to 2 (4).

simulated by the model ensemble for each grid cell (Table S3 and Figure 2). Different resolutions, O₃ precursor emissions, and representations of chemical and transport processes in the individual models contribute to a large standard deviation among O₃ responses, particularly within the source region (6). The range of avoided mortalities given by the 68% CI (± 1 standard deviation) in the modeled O₃ responses is similar in magnitude to the 95% CI in the CRF from Bell et al. (1) for foreign source–receptor pairs, but is larger for each region in response to domestic emission reductions, except for SA. This large range in O₃ response to domestic emission reductions influences the relative importance of source–receptor pairs for mortality. For example, whereas using the ensemble mean O₃ concentration for NA emission reductions results in similar annual avoided mortalities in NA and EU, using the mean minus 1 standard deviation leads to more avoided mortalities in EU than in NA, and using the mean plus 1 standard deviation leads to more avoided mortalities in NA than in EU (Table S3). A similar effect occurs for foreign vs domestic avoided mortalities from EU emission reductions.

We also examine the sensitivity of the avoided nonaccidental mortalities to the mean CRF from three meta-analyses of single-city daily time-series studies (Table S3 and Figure 2), which have generally consistent results (2–4) and do not report CRFs for cardiopulmonary mortalities. The mean CRF from the meta-analyses is larger than the CRF from Bell et al. (1); consequently, avoided mortality estimates are 1.6–1.9 times larger. While the range in modeled O₃ responses to emission changes contributes more to uncertainty in our estimated avoided mortalities than does the uncertainty associated with the CRF from Bell et al. (1) for some regions, using the mean CRF from the meta-analyses yields avoided mortality estimates that are often near or higher than the upper end of the range from the uncertainty in modeled O₃ concentrations (Figure 2). Therefore, source–receptor relationships for mortality are highly sensitive to both the CRF and modeled O₃ responses to emission reductions.

Global CH₄ Mixing Ratio Reduction. Reducing the global CH₄ mixing ratio by 20% decreases O₃ fairly uniformly around

TABLE 3. Population-Weighted Reduction in Annual Mean Surface O₃ (ppb) and Annual Avoided Cardiopulmonary and Total Non-Accidental Mortalities (Hundreds) in Each Region and the Entire NH Following a 20% Reduction in the Global Mean CH₄ Abundance, Assuming No Concentration Threshold (Confidence Intervals (95%) Reflect Uncertainty in the CRF) only (1)

receptor region	population-weighted ΔO_3 (ppb)	avoided cardiopulmonary mortalities (hundreds)	avoided nonaccidental mortalities (hundreds)
NA	1.11	11 (5 – 17)	19 (10 – 29)
EA	1.08	38 (19 – 59)	58 (30 – 86)
SA	1.19	48 (23 – 73)	83 (43 – 123)
EU	1.23	39 (19 – 59)	58 (30 – 86)
NH	1.12	160 (77 – 244)	271 (141 – 401)

the world, so that the population-weighted changes in O₃ concentration in all receptor regions are comparable and slightly higher in SA and EU (Table 3), in agreement with previous studies (8, 9, 23). About 80% of the 16,000 (95% CI, 7700–24,400) NH annual avoided cardiopulmonary mortalities (assuming no concentration threshold) occur in the four regions: 8% of which occur in NA (57–82% of these in the US), 28% in EA, 35% in SA, and 29% in EU. Since the concentration change in each region is similar, the differences in avoided mortalities are largely driven by population and baseline mortality rates. Avoided nonaccidental mortalities are about 1.7 times the cardiopulmonary results, and both are 0.6–0.8 times the results with a threshold of 35 ppb (Table S4). These results are similar to a previous estimate of about 30,000 avoided nonaccidental mortalities in 2030 due to 20% global anthropogenic CH₄ emission reductions (9), when accounting for differences in steady-state assumptions, modeled CH₄ reductions, and future population growth. CH₄ responds to emission reductions over decades, during which population growth increases the ultimate health benefits of CH₄ reductions.

Following methods used by Fiore et al. (6), we infer the contribution of 20% anthropogenic CH₄ emission reductions

TABLE 4. Population-Weighted Reduction in Annual Mean Surface O₃ (ppb) and Annual Avoided Cardiopulmonary and Total Non-Accidental Mortalities (Hundreds) in the NH Following a 20% Reduction in Anthropogenic CH₄ Emissions in Each Region Relative to the Base Simulation, Assuming No Concentration Threshold (Confidence Intervals (95%) Reflect Uncertainty in the CRF Only (1))

source region	population-weighted ΔO ₃ (ppb)	avoided cardiopulmonary mortalities (hundreds)	avoided nonaccidental mortalities (hundreds)
NA	0.145	21 (10 – 31)	35 (18 – 52)
EA	0.166	24 (11 – 36)	40 (21 – 59)
SA	0.150	21 (10 – 33)	36 (19 – 54)
EU	0.140	20 (10 – 31)	34 (18 – 50)

in each region to the simulated O₃ response from reducing the global CH₄ mixing ratio by 20% (see Supporting Information). We find about equivalent resulting reductions in population-weighted O₃ concentrations and avoided mortalities in the NH (Table 4), with the greatest mortality decrease in SA and smallest in NA (Figure S5). Without a threshold, reducing anthropogenic CH₄ emissions by 20% in all source regions collectively avoids 8600 (4100–13,100) cardiopulmonary mortalities annually. Avoided nonaccidental mortalities are about 1.7 times the cardiopulmonary results, and both are about 0.7 times the results with a threshold of 35 ppb (Table S5). The domestic impacts of reducing regional CH₄ emissions are 0.1–0.3 times those from reducing domestic NO_x, NMVOC, and CO emissions by the same percentage, due to the large domestic effects of these precursors (Table 2 and Figure S5). However, for each region, reducing emissions of NO_x, NMVOC, and CO in the three foreign regions has impacts on mortality similar to those of an equivalent percentage decrease in CH₄ emissions (Table 2 and Figure S5).

Foreign vs Domestic Influences within North America.

Foreign anthropogenic NO_x, NMVOC, and CO emission reductions decrease mortalities in NA throughout the year, but compared with domestic emission reductions, cause fewer avoided mortalities in the summer and more in the winter due to the seasonality of O₃ in response to domestic NO_x emission reductions (Figures 3 and S6). We compare foreign and domestic impacts in NA using Health Import Sensitivity (HIS), the ratio of the summed avoided mortalities following NO_x, NMVOC, and CO emission reductions in the three foreign regions to the avoided mortalities following

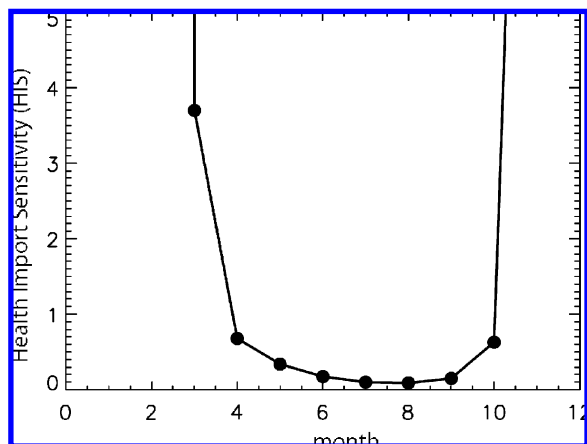


FIGURE 3. Seasonality of Health Import Sensitivity (the ratio of foreign vs domestic impacts on cardiopulmonary mortalities) in NA, assuming no threshold.

emission reductions in NA only. When HIS < 1, the avoided mortalities due to domestic emission reductions are greater than the sum of those from emission reductions in the three foreign regions. The annual HIS for NA is 0.56, indicating that reducing domestic emissions is more effective than reducing foreign emissions for avoiding mortalities. The HIS for NA is lowest in the summer due to the large influence of domestic vs foreign emissions on mortality, and it approaches infinity in the winter when foreign emission reductions decrease O₃ but domestic emission reductions increase O₃ (Figure 3).

Discussion

We estimate the intercontinental impacts of O₃ on human mortality using a health impact function and multimodel estimates of the surface O₃ response to precursor emission reductions in four large industrial regions. Reducing O₃ precursor emissions by 20% within each receptor region (“domestic”) avoids more mortalities than does reducing emissions in any of the three foreign regions. However, for all regions, emission reductions in the three foreign regions contribute significantly to the avoided mortalities resulting from emission reductions in all regions combined (30% for NA and EA, 20% for SA, and >50% for EU). For EU, the larger foreign impact is due to the influence of NA emissions.

Intercontinental health impacts of O₃ are influenced by the contribution of foreign emissions to O₃ in each region and by regional population and baseline mortality rates. Using the mean O₃ responses from the multimodel ensemble, more mortalities are avoided outside the source region than within following emission reductions in NA (64–76% of resulting NH annual avoided mortalities occur outside the source region) and EU (55–58%, assuming no threshold). The opposite is true for EA (about 70% within the source region) and SA (about 90%). Due to large populations, reducing emissions in any of the regions avoids many mortalities in EA and SA. Similarly, lowering the global CH₄ abundance by 20% reduces mortality most in SA, followed by EU, EA, and NA.

The relative importance of source–receptor pairs for mortality is strongly influenced by the accuracy and consistency of global CTMs in estimating O₃ responses to domestic and foreign precursor emission reductions. The substantial intermodel variation, particularly in the domestic O₃ response, causes uncertainty that influences our conclusions about the relative numbers of domestic vs foreign avoided mortalities. We expect that the coarse resolution of global models captures long-range transport, but may cause error particularly for domestic emission reductions. In assessing mortalities, systematic positive biases in the model ensemble mean O₃ should not affect our results when no threshold is assumed, but would when we assume a threshold, as the number of days above the threshold is affected. Future research should explore the possible bias in using coarse global models for health impact assessments, considering the relationships between concentration and population in metropolitan regions, by comparing with regional models, and should increasingly use finer-resolution or nested CTMs.

Our results focus solely on O₃-related mortality and do not account for possible effects on particulate matter mortality from the same changes in emissions. We examine only the short-term impacts of O₃ on mortality, for which years of life saved are unknown. We assume that the CRFs found in the US are valid globally, but populations across the world have different health characteristics that may influence O₃ impacts. The CRFs used here are corroborated by meta-analyses of short-term O₃ epidemiology studies in Europe and the developing world, which show a similar association between O₃ and mortality, but are somewhat

inconsistent in the magnitude of the relationship. In addition, epidemiology studies could be subject to confounders, including correlated copollutants, that are as yet unknown.

Our results suggest that emission controls in one region affect O₃ air quality and O₃-related mortality in other world regions, with impacts outside the source region that are comparable to or even exceed the domestic (intra-regional) impacts. Confidence in these estimates would be increased by resolving uncertainties, including domestic O₃ responses to emission reductions and O₃ CRFs around the world. Despite uncertainties, our results point to widespread impacts of emission reductions, suggesting that collective international agreements over larger spatial scales may be needed to address local mortalities due to O₃ pollution (24, 25).

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Supporting Information Available

Participating modeling groups, maps of the four regions, population, and baseline mortality rates, and additional tables of results. This information is available free of charge via the Internet at <http://pubs.acs.org/>.

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