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HTAP2 multi-model estimates of premature human mortality due to intercontinental transport of air pollution

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Abstract

Ambient air pollution from ozone and fine particulate matter is associated with premature mortality. As emissions from one continent influence air quality over others, changes in emissions can also influence human health on other continents. We estimate global air pollution-related premature mortality from exposure to $PM_{2.5}$ and ozone , and the avoided deaths from 20% anthropogenic emission reductions from six source regions, North America (NAM), Europe (EUR), South Asia (SAS), East Asia (EAS), Russia/Belarus/Ukraine (RBU) and the Middle East (MDE), three emission sectors, Power and Industry (PIN), Ground Transportation (TRN) and Residential (RES) and one global domain (GLO), using an ensemble of global chemical transport model simulations coordinated by the second phase of the Task Force on Hemispheric Transport of Air Pollution (TF-HTAP2), and epidemiologically-derived concentration-response functions. We build on results from previous studies of the TF-HTAP by using improved atmospheric models driven by new estimates of 2010 emissions, with more source and receptor regions, new consideration of source sector impacts, and new epidemiological mortality functions. We estimate 290,000 (95% CI: 30,000, 600,000) premature O_3-related deaths and 2.8 million (0.5 million, 4.6 million) $PM_{2.5}$-related premature deaths globally for the baseline year 2010. While 20% emission reductions from one region generally lead to more avoided deaths within the source region than outside, reducing emissions from MDE and RBU can avoid more O_3-related deaths outside of these regions than within, and reducing MDE emissions also avoids more $PM_{2.5}$-related deaths outside of MDE than within. In addition, EUR, MDE and RBU have more avoided O_3-related deaths from reducing foreign emissions than from domestic reductions. For six regional emission reductions, the total avoided extraregional mortality is estimated as 10,300 (6,700, 13,400) deaths/year and 42,000 (12,400, 60,100) deaths/year through changes in O_3 and $PM_{2.5}$, respectively. Interregional transport of air pollutants leads to more deaths through changes in $PM_{2.5}$ than in O_3, even though O_3 is transported more on interregional scales, since $PM_{2.5}$ has a stronger influence on mortality. In sectoral emission reductions, TRN emissions account for the greatest fraction (26-53% of global emission reduction) of O_3-related premature deaths in most regions, except for EAS (58%) and RBU (38%) where PIN emissions dominate. In contrast, PIN emission reductions have the greatest fraction (38-78% of global emission reduction) of $PM_{2.5}$-related deaths in most regions, except for SAS (45%) where RES emission dominates. The spread of air pollutant concentration changes across models contributes most to the overall uncertainty in estimated avoided

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deaths, highlighting the uncertainty in results based on a single model. Despite uncertainties, the health benefits of reduced intercontinental air pollution transport suggest that international cooperation may be desirable to mitigate pollution transported over long distances.

1 Introduction

Ozone (O_3) and fine particulate matter with aerodynamic diameter less than 2.5 μm ($PM_{2.5}$) are two common air pollutants with known adverse health effects. Epidemiological studies have shown that both short-term and long-term exposures to O_3 and $PM_{2.5}$ are associated with elevated rates of premature mortality. Short-term exposure to O_3 is associated with respiratory morbidity and mortality (Bell et al., 2005; Bell et al., 2014; Gryparis et al., 2004; Ito et al., 2005; Levy et al., 2005; Stieb et al., 2009) while long-term exposure to O_3 has been associated with premature respiratory mortality (Jerrett et al., 2009, Turner et al., 2016). Short-term exposure to $PM_{2.5}$ has been associated with increases in daily mortality rates from all natural causes, and specifically from respiratory and cardiovascular causes (Bell et al., 2014; Du et al., 2016; Powell et al., 2015; Pope et al., 2011) while long-term exposure to $PM_{2.5}$ can have detrimental chronic health effects, including premature mortality due to cardiopulmonary diseases and lung cancer (Brook et al., 2010; Burnett et al., 2014; Hamra et al., 2014; Krewski et al., 2009; Lepeule et al., 2012; Lim et al., 2012). The Global Burden of Disease Study 2015 (GBD 2015) estimated 254,000 deaths/year associated with ambient O_3 and 4.2 million associated with ambient $PM_{2.5}$ (Cohen et al. 2017). A comparable study using output from an ensemble of global chemistry–climate models estimated 470,000 deaths/year associated with O_3 and 2.1 million premature deaths/year associated with anthropogenic $PM_{2.5}$ (Silva et al. 2013).

Numerous observational and modeling studies have shown that anthropogenic emissions can affect O_3 and $PM_{2.5}$ concentrations across continents (Heald et al., 2006; TF-HTAP, 2010; Leibensperger et al., 2011; Lin et al., 2012; Lin et al., 2017; Liu et al., 2009a; West et al., 2009a; Wild and Akimoto, 2001; Yu et al., 2008). As changes in emissions from one continent influence air quality over others, several studies have estimated the premature mortality from intercontinental transport (Anenberg et al., 2009; Anenberg et al., 2014; Bhalla et al., 2014; Duncan et al., 2008; Im et al., 2017; Liu et al., 2009b; West et al., 2009b; Zhang et al., 2017). In 2005, the Task Force on Hemispheric Transport of Air Pollution (TF-HTAP) was launched under the United Nations Economic Commission for Europe (UNECE) Convention on Long-Range Transboundary Air Pollution (LRTAP). One of its tasks is to investigate the impacts of emission reductions on the intercontinental transport of air pollution, air quality, health,

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ecosystem and climate effects, using a multi-model ensemble to quantify uncertainties due to differences between models (Anenberg et al., 2009; Anenberg et al., 2014; Fiore et al., 2009; Fry et al., 2012; Huang et al., 2017; Stjern et al., 2016; Yu et al., 2013). In the TF-HTAP Phase 1 (TF-HTAP1), human premature mortality due to 20% anthropogenic emission reductions in four large source regions was investigated by Anenberg et al. (2009 and 2014). They found that 20% foreign O_3 precursor emission reductions contribute approximately 30% to >50% of the deaths avoided by reducing precursor emissions in all four regions together (Anenberg et al., 2009). Similarly, reducing emissions in NA and EU was found to avoid more O_3-related premature deaths outside the source region than within (Anenberg et al., 2009), which agrees with other studies that together show for the first time that emission reductions in NA and EU have greater impacts on mortality outside the source region than within (Duncan et al., 2008; West et al., 2009). In contrast, Anenberg et al. (2014) estimate that 93–97 % of $PM_{2.5}$-related avoided deaths from reducing emissions in all four regions occurs within the source region while 3–7 % occur outside the source region from transport between continents. Despite the longer atmospheric lifetime of O_3 and its relatively larger scale of influence, $PM_{2.5}$ was found to cause more deaths from intercontinental transport (Anenberg et al., 2009; 2014). Similarly, an ensemble of regional models in the third phase of the Air Quality Modelling Evaluation International Initiative (AQMEII3) found that a 20% decrease of emissions within the source region avoids 54,000 and 27,500 premature deaths in Europe and the U.S. (from both O_3 and $PM_{2.5}$), while the reduction of foreign emissions alone avoids ~1,000 and 2,000 premature deaths in Europe and the U.S. (Im et al., 2017). Crippa et al (2017) used the TM5-FASST reduced-form model with HTAP2 emissions to estimate a global sensitivity to 20 % emission reductions of $PM_{2.5}$-related premature deaths of 401,000 globally, and 42,000 and 20,000 for Europe and the US respectively.

In addition, several studies have evaluated the relative importance of individual emissions sectors (Barrett et al., 2010; Bhalla et al., 2014; Chafe et al., 2014; Chambliss et al., 2014; Corbett et al., 2007) or multiple sectors (Lelieveld et al., 2015; Silva et al., 2016a) to ambient air pollution–related premature mortality. Lelieveld et al. (2015) estimated that residential energy use such as for heating and cooking has the largest mortality impact globally (for $PM_{2.5}$ and O_3 mortality combined), particularly in South and East Asia. Silva et al (2016) likewise found that residential & commercial emissions are most important for ambient $PM_{2.5}$-related mortality, but also found that land transportation had the greatest impact on O_3-related mortality, particularly in North America, South America, Europe, FSU and the Middle East. Understanding the impact of different sectors on the global burden and the relative importance of each sector among regions can help stimulate international efforts and region-specific air pollution

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control strategies. Nevertheless, those studies were limited by using a single atmospheric model, reflecting a need to understand whether results differ among models.

In this study, we estimate the impacts of interregional transport and of source sector emissions on human premature mortality from O_3 and $PM_{2.5}$, using an ensemble of global chemical transport models coordinated by the Task Force on Hemispheric Transport of Air Pollution Phase 2 (TF-HTAP2) (Galmarini et al., 2016; Huang et al., 2016; Janssens-Maenhout et al., 2015; Stjern et al., 2016). Anthropogenic emissions were perturbed by 20% in six source regions: North America (NAM), Europe (EUR), South Asia (SAS), East Asia (EAS), Russia/Belarus/Ukraine (RBU) and the Middle East (MDE), three emission sectors: Power and Industry (PIN), Ground Transportation (TRN) and Residential (RES), and one worldwide region (GLO). Human premature mortality due to these reductions is calculated using a health impact function based on a log-linear model for O_3 (Jerrett et al. 2009) and an integrated exposure-response model for $PM_{2.5}$ (Burnett et al. 2014), within the six source regions and elsewhere in the world. We conduct a Monte Carlo simulation to estimate the overall uncertainty due to uncertainties in relative risk, air pollutant concentrations (given by the spread of results among different models), and baseline mortality rates.

2 Method

2.1 Modeled O_3 and $PM_{2.5}$ surface concentration

Global numerical modelling experiments initiated by TF-HTAP2, the regional experiments by the Air Quality Model Evaluation International Initiative (AQMEII) over Europe and North America, and the Modelling Intercomparison Study-Asia (MICS-Asia) were coordinated to perform consistent emission perturbation modelling experiments across the global, hemispheric and continental/regional scales (Galmarini et al., 2016). Simulation periods, meteorology, emission inventories, boundary conditions, and model output are also consistent. The Joint Research Centre's (JRC) EDGAR (Emission Data Base for Global Research) team in collaboration with regional emission experts from the U.S. Environmental Protection Agency (US-EPA), European Monitoring and Evaluation Programme (EMEP), Centre on Emission Inventories and Projections (CEIP), Netherlands Organization for Applied Research (TNO), and the MICS-Asia Scientific Community and Regional Emission Activity Asia (REAS) provide a global emission inventory at 0.1^0x0.1^0 resolution for TF-HTAP2 modeling experiments (Janssens-Maenhout et al., 2015). The emissions dataset was constructed for SO_2, NO_X, CO, NMVOC, NH_3, PM_{10}, $PM_{2.5}$, BC and OC and seven emission sectors (shipping, aircraft, land transportation, agriculture, residential, industry and energy) for

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the year 2010 (Fig. S1).

This study uses outputs from 14 global models / model versions (Table S1) participating in TF-HTAP2. Overall, TF-HTAP2 model resolutions are finer than in TF-HTAP1. In TF-HTAP2, each model performed a baseline simulation and sensitivity simulations where the anthropogenic emissions in a defined source region or sector were perturbed (reduced by 20% in most cases). Based on the number of models that simulated different experiments, we choose to focus on emission reductions from six source regions, three emission sectors, and one global domain. More specifically, all anthropogenic emissions are reduced by 20% in the North America (NAM), Europe (EUR), South Asia (SAS), East Asia (EAS), Russia/Belarus/Ukraine (RBU) and the Middle East (MDE) continental regions, in the Power and Industry (PIN), Ground Transportation (TRN) and Residential (RES) emission sectors globally, and in one global domain (GLO) (Fig. S2). Unlike TF-HTAP1 (TF-HTAP, 2010) which defined rectangular regions that included ocean or some sparsely inhabited regions, TF-HTAP2 regions are defined by geopolitical boundaries.

We selected output from the models that provided temporally resolved volume mixing ratios of O_3 and mass mixing ratios of $PM_{2.5}$ ("mmrpm2p5") for the baseline and at least one regional or sectoral emission reduction scenario. Among the 14 models, 11 models reported O_3 and 8 reported $PM_{2.5}$ for regional emission perturbation scenarios, 4 models reported O_3 and 4 reported $PM_{2.5}$ for sectoral emission perturbation scenarios, and 10 models reported O_3 and 8 reported $PM_{2.5}$ for the global emission perturbation. All models used prescribed meteorology for the year 2010, although this meteorology was not uniform across models. Modeled concentrations are processed by calculating metrics consistent with the underlying epidemiological studies to estimate premature mortality. For O_3, we calculate the average of daily 1-h maximum O_3 concentration for the 6 consecutive months with the highest concentrations in each grid cell (Jerrett et al., 2009), for the baseline and each 20% emission reduction scenario. While some models reported hourly O_3 metrics, others only reported daily or monthly O_3. We include these models by first calculating the ratio of the 6-month average of daily 1-h maximum O_3 to the annual average of O_3 in individual grid cells, for models reporting hourly O_3, and then applying that ratio to the annual average of ozone for those models that only report daily or monthly O_3, following Silva et al. (2013; 2016b). For $PM_{2.5}$, we calculate the annual average $PM_{2.5}$ concentration in each cell using the monthly total $PM_{2.5}$ concentrations reported by each model ("mmrpm2p5"). Model results for these two metrics are then regridded from each model's native grid resolution (varying from 0.5°×0.5° to 2.8°×2.8°) to a consistent 0.5°×0.5° resolution used in mortality estimation. We estimate regional and sectoral multi-model averages for each 20% emission reduction scenario in the year 2010, but for each perturbation case, we

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only include models that report both the baseline and perturbation cases.

2.2 Health impact assessment

We use output from the TF-THAP2 model ensemble to estimate annual O_3- and $PM_{2.5}$-related global cause-specific premature mortality and avoided mortality from the 20% regional and sectoral emission reductions, following the same methods used by Silva et al. (2016a; 2016b). The annual O_3- and $PM_{2.5}$-related premature mortality is calculated using a health impact function based on epidemiological relationships between ambient air pollution concentration and mortality in each grid cell: $\Delta M = y_0 \times AF \times Pop$, where ΔM is premature mortality, y_0 is the baseline mortality rate (for the exposed population), $AF=1 - 1/RR$ is the attributable fraction, where RR is relative risk of death attributable to the change in air pollutant concentration ($RR=1$ when there is no increased risk of death associated with a change in pollutant concentration), and Pop is the exposed population (adults aged 25 and older).

For O_3 mortality, we use a log-linear model for chronic respiratory mortality (RESP) from the American Cancer Society (ACS) study (Jerrett et al 2009), following recent studies including the GBD (Cohen et al., 2017), but Turner et al. (2016) recently published new results for chronic ozone mortality, and adoption of these results would lead to more ozone-related deaths overall (Malley et al., 2017). RR is calculated as:

$$RR = e^{\beta \Delta x} \quad (1)$$

where β is the concentration-response factor, Δx corresponds to the change in pollutant concentrations between simulations with perturbed emissions and the baseline simulation, and $RR = 1.040$ (95% Confidence Interval, CI: 1.013-1.067) for a 10 ppb increase in O_3 concentrations. We estimate O_3-related premature deaths due to respiratory disease (RESP) based on decreases or increases in O_3 concentration (i.e. Δx) due to 20% regional and sectoral emission reduction scenarios relative to the baseline. For regional and sectoral reductions, we do not assume a low-concentration threshold below which changes in O_3 have no mortality effects, as there is no clear evidence for such a threshold, following Anenberg et al (2009; 2010) and Silva et al. (2013; 2016a, b). However, we evaluate global O_3 premature mortality for the baseline 2010 simulation, relative to a counterfactual concentration of 37.6 ppb (Lim et al. 2012), for consistency with GBD estimates (Cohen et al., 2017).

For $PM_{2.5}$ mortality, we apply the Integrated Exposure–Response (IER) model, which is intended to better represent the risk of exposure to $PM_{2.5}$ at locations with high ambient concentrations (Burnett et al., 2014). RR is calculated as:

$$\text{For } z < z_{cf},\ RR_{IER}(z) = 1 \quad (2)$$

$$\text{For } z \geqq z_{cf},\ RR_{IER}(z) = 1 + \alpha\{1 - exp[-\gamma(z - z_{cf})^{\delta}]\} \quad (3)$$

where z is the $PM_{2.5}$ concentration in $\mu g/m^3$ and z_{cf} is the counterfactual concentration

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below which no additional risk is assumed (Burnett et al., 2014). The overall $PM_{2.5}$-related cause-specific premature deaths related to ischemic heart disease (IHD), cerebrovascular disease (STROKE), chronic obstructive pulmonary disease (COPD) and lung cancer (LC) are estimated using RRs per age group for IHD and STROKE and RRs for all ages for COPD and LC. A uniform distribution from 5.8 $\mu g/m^3$ to 8.8 $\mu g/m^3$ is used for z_{cf} as suggested by Burnett et al. (2014). We estimate avoided premature mortality in 20% emission perturbation experiments by taking the difference in premature mortality estimates with the 2010 baseline.

For the exposed population, we use the Oak Ridge National Laboratory's Landscan 2011 Global Population Dataset at approximately 1 km resolution (30"x30") (Bright et al., 2012). We use ArcGIS 10.2 geoprocessing tools to estimate the population of adults aged 25 and older from Landscan. We obtained cause-specific baseline mortality rates for 187 countries from the GBD 2010 mortality dataset (IHME, 2013). The population and baseline mortality per age group were regridded to the 0.5°×0.5° grid (Table S2 and Fig. S3). Cause-specific baseline mortality rates vary geographically, e.g. RESP and COPD are relatively more dominant in South Asia, IHD in Europe, STROKE in Russia, and LC in North America.

Finally, we conduct 1,000 Monte Carlo simulations to propagate uncertainty from baseline mortality rates, modeled air pollutant concentrations, and the RRs in health impact functions. We use the reported 95% CIs for cause-specific baseline mortality rates, assuming lognormal distributions. For modeled O_3 and $PM_{2.5}$ concentrations we used the absolute value of the coefficient of variation among models in each grid cell, for each 20% emission perturbation case minus the baseline, assuming a normal distribution. For O_3 RRs, we use the reported 95% confidence intervals (CIs), assuming a normal distribution. For $PM_{2.5}$ RRs, we use the parameter values (i.e. α, γ, δ and z_{cf}) of Burnett et al. (2014) for 1,000 simulations. Given that our 0.5°×0.5° grid cell resolution can capture most population well in a given region, uncertainty associated with population was assumed to be negligible.

3 Results

3.1 Response of O_3 and $PM_{2.5}$ concentrations to 20% regional and sectoral emission reductions

Previous TF-HTAP studies reported area-averaged concentrations to quantify source-receptor relationships averaging concentrations over a region (Doherty et al., 2013; Fiore et al., 2009; Fry et al., 2012; Huang et al., 2017; Stjern et al., 2016;

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Sanderson et al., 2008; Yu et al., 2013). Here, we present the population-weighted concentration over a region, which is more relevant for health. Among six receptor regions, the population-weighted multi-model mean O_3 concentrations range from 48.38±8.05 ppb in EUR to 65.72±10.08 ppb in SAS with a global average of 53.74± 8.03 ppb, while the annual population-weighted multi-model mean $PM_{2.5}$ concentrations range from 9.36±2.62 μg/m^3 in NAM to 39.27±13.50 μg/m^3 in EAS with a global average of 25.98±5.05 μg/m^3 (Table 1 and S3-S4 and Figs.S4-S5).

For 20% perturbation scenarios, in general the impact on the multi-model mean change in surface O_3 and $PM_{2.5}$ concentration is greater within the source region (i.e., domestic region) than outside of it (i.e., foreign region) (Figs. S6-S7). This is also true for individual model results (Figs. S8-S9). Among six source regions, the emission reduction from SAS has the greatest impact on global population-weighted O_3 concentration (Tables 2 and S3), while that from EAS has greatest impact on $PM_{2.5}$ (Tables 3 and S4). The source-receptor pairs with the greatest changes in O_3 and $PM_{2.5}$ concentration reflect the geographical proximity between regions and the magnitude of emissions (Table 2-3) – e.g., EUR→MDE (0.34±0.08 ppb), EUR→RBU (0.34 ppb±0.09), EAS→NAM (0.29±0.14 ppb), EAS→RBU (0.27±0.12 ppb), and NAM→EUR (0.26±0.55 ppb) for O_3, and EUR→RBU (0.26±0.19 μg/m^3), EUR→MDE (0.18±0.08 μg/m^3), MDE→SAS (0.12±0.06 μg/m^3), SAS→EAS (0.08±0.08 μg/m^3), and EAS→SAS (0.08±0.07 μg/m^3) for $PM_{2.5}$. Our ensemble shows similar ozone responses in the western US to emission reductions from EAS (Figs. S6c) as those modeled by Lin et al. (2012 and 2017).

For each receptor region, reducing foreign anthropogenic emissions by 20% (estimated by global minus within-region reductions) can decrease population-weighted O_3 concentrations by 29-74% of the change in O_3 concentration and 8–41 % of the change in $PM_{2.5}$ concentration (Tables 2-3). In some cases, regional emission reductions cause small O_3 concentration increases within the source region or in foreign receptors, reflecting O_3 nonlinear responses (Figs. S8 and S10). For instance, C-IFS_v2 predicts O_3 concentration increases in EUR by 0.04 ppb from domestic emission reductions, which is in agreement with results from TF-HTAP1 (Anenberg et al. 2009). Similarly, CMAchem shows more local O_3 increases, particularly in SAS, than other models (Figs. S8 and S10). The change in O_3 concentration in foreign receptors is broader than for $PM_{2.5}$, reflecting that O_3 has a longer atmospheric lifetime than $PM_{2.5}$.

For sectors, TRN emission reductions cause the greatest decrease in global population-weighted O_3 by 1.13±0.19 ppb, while PIN emission reductions cause the greatest decrease in $PM_{2.5}$ by 1.46±0.56 μg/m^3 globally (Tables 2-3). The 20% emission reductions from individual sectors also have different effects in different regions. Of the three sectors, emission reductions from TRN have the greatest effect on population-

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weighted O_3 in NAM, EUR, SAS, MDE and MDE (40-50% of the global emission reduction) while PIN emission reductions dominate in EAS (57%). Emission reductions from PIN have the greatest effect on population-weighted $PM_{2.5}$ in NAM, EUR, EAS, MDE and MDE (41-84%) while RES emission reductions dominate in SAS (43%). The response of $PM_{2.5}$ concentration to sectoral emission reductions differs significantly across models, which reflects in part the $PM_{2.5}$ species simulated by each model (Table S1). For instance, we found that models that simulate $PM_{2.5}$ nitrate (i.e. CHASER_t42 and GEOSCHEMADJOIN) predict a greater impact on $PM_{2.5}$ concentration from TRN emission reduction than those without nitrate (i.e. GOCARTv5 and SPRINTARS) (Fig S9).

3.2 Global mortality burden associated with anthropogenic air pollution

Table 4 shows the annual multi-model mean O_3- and $PM_{2.5}$-related premature deaths on 6 regions and globally for year 2010 baseline with 95% confidence intervals (CI) based on Monte Carlo sampling. Tables S5-S6 show estimates of premature deaths due to anthropogenic O_3 and $PM_{2.5}$ from individual models. For the ensemble model mean, we estimate 290,000 (30,000, 600,000) premature O_3-related deaths globally using a 37.6 ppb counterfactual concentration, and 2.8 million (0.5 million, 4.6 million) $PM_{2.5}$-related premature deaths using a uniform distribution of counterfactual concentration from 5.8 $\mu g/m^3$ to 8.8 $\mu g/m^3$. Highly populated areas of India and East Asia have the greatest O_3- and $PM_{2.5}$-related deaths, and those regions together account for 82% and 66% of the global total O_3- and $PM_{2.5}$-related deaths. Compared with the GBD 2015 (Cohen et al 2017), our global burden estimates are greater than the 254,000 (97,000, 422,000) premature deaths/year for O_3 from GBD, while less than 4.2 million (3.7 million, 4.8 million) premature deaths for $PM_{2.5}$. Lelieveld et al (2015) estimate 142,000 (CI: 90,000, 208,000) O_3-related deaths and 3.2 million (1.5 million, 4.6 million) $PM_{2.5}$-related premature deaths for 2015. These differences can be explained mainly by exposure estimates. Here we used a multi-model ensemble, whereas Lelieveld et al. (2015) used a single model, and Cohen et al (2017) used a single model for O_3 and a single model combined with surface and satellite observations for $PM_{2.5}$. In addition, Cohen et al (2017) use higher updated baseline mortality rate and population which leads to higher global premature deaths estimate. Our wider range of uncertainty for the global mortality reflects the uncertainty in baseline rates, RRs and spread of air pollutant concentration across models whereas Cohen et al (2017) consider national-level population-weighted mean concentrations and uncertainty of IER function predictions at each concentration and Lelieveld et al. (2015) only account for the statistical uncertainty of the parameters used in the IER functions.

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3.3 Effect of regional reductions on mortality

Reducing global anthropogenic emissions by 20% avoids 47,400 (11,300, 99,000) O_3-related deaths and 290,000 (67,100, 405,000) $PM_{2.5}$-related premature deaths (Tables 5-6 and S7-S8). Most avoided air pollution-related deaths were found within or close to the source region (Figs.1-4). Reducing anthropogenic emissions by 20% from NAM, EUR, SAS, EAS, MDE and RBU can avoid 54%, 54%, 95%, 85%, 21%, and 22% of the global change in O_3-related deaths within the source region, and 93%, 81%, 93%, 94%, 32%, and 82% of the global change in $PM_{2.5}$-related deaths, respectively (Table 5-6). Whereas the most O_3-related premature deaths can be avoided by reducing SAS emissions (20,000 (3,600, 42,200) deaths/year), reducing EAS emissions avoids more O_3-related premature deaths (1,700 (-1,300, 5,400)) outside of the source region than for any other region (500 (180, 870) deaths/year to 1,300 (-1,200, 4,400) deaths/year (Table 5). Similarly, while reducing EAS emissions avoids the most $PM_{2.5}$-related premature deaths (96,600 (3,500, 136,000) deaths/year), reducing EUR emissions avoids more $PM_{2.5}$-related premature deaths (7,400 (930, 9,500) deaths/year) outside of the source region than for any other region (1,400 (-320, 2,300) deaths/year to 5,500 (3,000, 7,800) deaths/year) (Table 6). While emission reductions from one region generally lead to more avoided deaths within the source region than outside, 20% anthropogenic emission reductions from MDE (for both O_3 and $PM_{2.5}$) and RBU (for O_3) can avoid more premature deaths outside of the source region than within. This result for RBU is in agreement with West et al (2009). However, the results for NAM and EUR do not agree with previous studies that found that emission reductions in these regions cause more O_3-related avoided premature deaths outside of the source region than within (Anenberg et al., 2009; Duncan et al., 2008; West et al., 2009). For $PM_{2.5}$, our results are comparable with Anenberg et al. (2014) and Crippa et al. (2017) who found that for most regions, $PM_{2.5}$-related avoided premature deaths are higher within the source region than outside. The difference in results with TF-HTAP1 may be in part because of the definition of regions. Whereas the TF-HTAP2 regions are defined by geopolitical boundaries, the TF-HTAP1 regions are defined by square domains which are larger and include more ocean areas (Anenberg et al., 2009). This could lead more emissions like aviation and shipping emission reduced by TF-HTAP1 experiment, reflecting the higher premature deaths can be avoided in downwind regions. TF-HTAP2 also adds new regions (RBU and MDE) that have strong influences on air quality in adjacent regions.

Using individual models, different conclusions may result for the relative importance of inter-regional transport. For example, for O_3, 8 models predict that NAM emission reductions cause more O_3-related premature deaths within NAM (i.e CAM-Chem, CHASER_T42, CHASER_T106, C-IFS, GEOSCHEMADJOINT, GEOS-

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Chem, GFDL_AM3 and HadGEM2-ES), whereas 2 models predict more deaths outside (i.e. EMEPrv48 and OsloCTM3.v2). 5 models suggest that EUR emission reductions cause more O_3-related premature deaths within EUR (i.e. CAM-chem, CHASER_T42, CHASER_T106, GFDL_AM3 and HadGEM2-ES), whereas 4 show more deaths outside (i.e. C-IFS, GEOSCHEMADJOINT, EMEPrv48 and OsloCTM3.v2). Each individual model shows that emission reductions from SAS and EAS avoid more O_3-related premature deaths within than outside, and that those from MDE and RBU avoid more O_3-related premature deaths outside than within (Fig. S8 and S10). For $PM_{2.5}$, each individual model shows that emission reductions from NAM, EUR, SAS, EAS and RBU avoid more $PM_{2.5}$-related premature deaths within than outside, while for emission reductions from MDE, 3 models (EMEPrv48, GEOSCHEMADJOINT and SPRINARS) show more $PM_{2.5}$-related premature deaths within, while 3 (CHASER_T42 GEOS5 and GOCART) show more $PM_{2.5}$-related premature deaths outside (Fig. S9 and S11). The variation of health effect reflects the differences in processing of natural emissions, atmospheric physical and chemical mechanisms, transport time step etc across models.

For each receptor region, reducing domestic anthropogenic emissions by 20% contributes about 66%, 39%, 84%, 72%, 45% and 25% of the total O_3-related avoided premature mortality (from the global reduction), and 90%, 78%, 87%, 87%, 58% and 66% of the total $PM_{2.5}$-related avoided premature mortality (from the global reduction) in NAM, EUR, SAS, EAS, MDE and RBU, respectively (Table 5-6). Therefore, reducing emissions from foreign regions avoids more O_3 premature deaths in EUR, MDE and RBU than reducing domestic emissions, in agreement with the results for EUR from Anenberg et al (2009). Whereas EAS has the greatest number of avoided O_3-related premature deaths due to foreign emission reduction (3,800 (3,600, 3,900) deaths/year), RBU has the greatest fraction of O_3 mortality from foreign emission reductions (75%) (Table 5). Similarly, for $PM_{2.5}$, while EAS has greatest number of avoided $PM_{2.5}$-related premature deaths due to foreign emission reductions (13,600 (3,500, 18,800) deaths/year), MDE has the greatest fraction of $PM_{2.5}$ mortality from foreign emission reduction (42%) (Table 6).

Overall, adding results from all 6 regional reductions, interregional transport of air pollution from extraregional contributions is estimated to lead to more avoided deaths through changes in $PM_{2.5}$ (42,000 (12,400, 60,100) deaths/year) than in O_3 (10,300 (6,700, 13,400) deaths/year), consistent with Anenberg et al. (2009; 2014). This result is due to the greater influence of $PM_{2.5}$ on mortality, despite the shorter atmospheric lifetime of $PM_{2.5}$ relative to O_3.

We quantify the uncertainties in mortality due to the spread of air pollutant concentrations across models, RRs, and baseline mortality rates, as contributors to the

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overall uncertainty, expressed as a coefficient of variation and compare the result with the Monte-Carlo analysis estimate (Tables S9-S10). For the spread of the model ensemble, we calculate the deterministic mean and standard deviation estimates for mortality with mean RRs and baseline mortality rates. For RRs, we use the 95%CI of RRs reported by Jerrett et al. (2009) for O_3 and upper and lower bound of RRs reported by Burnett et al. (2014) for $PM_{2.5}$ with mean baseline mortality rates to estimate the deterministic mean and standard deviation for mortality. For baseline mortality rates, we use upper and lower bound of baseline mortality rates with mean RRs to estimate the deterministic mean and standard deviation for mortality. For both O_3 and $PM_{2.5}$ mortality, the spread of model results generally contributes most to the overall uncertainty, followed by uncertainty in RRs and in baseline mortality rates, for most source-receptor pairs. The spread of model results is generally wider for $PM_{2.5}$ (14% to 3974% among source-receptor pairs) than for O_3 (13% to 1065%). The uncertainty in RRs for O_3 mortality has constant value (33% to 34%) due to the fixed uncertainty range of RRs from Jerrett et al. (2009), whereas $PM_{2.5}$ mortality leads to a wider range of uncertainty (1% to 247%) in RRs because the uncertainty differs at different $PM_{2.5}$ concentrations (Burnett et al., 2014). Low uncertainty in baseline mortality rate was found for most source-receptor pairs (<20%) except for the response of $PM_{2.5}$ mortality in SAS to 20% reduction from RBU (66%).

3.4 Effect of sectoral reductions on mortality

Reducing global anthropogenic emissions by 20% in 3 sectors (i.e. PIN, TRN and RES) together avoids 48,500 (7,100, 108,000) O_3-related premature deaths and 243,000 (66,800, 357,000) $PM_{2.5}$-related premature deaths globally (Tables 5-6), with the greatest avoided air pollution-related premature deaths located in highly populated areas (e.g., North America, Europe, India, China, etc.) (Figs.1-4). For instance, reducing anthropogenic emissions by 20% in 3 sectors together avoids the highest number of O_3-related deaths in SAS (24,000 (6,000, 49,600) deaths/year) and $PM_{2.5}$-related deaths in EAS (83,400 (29,400, 135,000) deaths/year). We compare our estimates of O_3 and $PM_{2.5}$-related premature deaths attributable to PIN, TRN and RES emissions with previous studies, by multiplying our results for 20% emission reductions by 5, and by combining their sectors to nearly match each of the three sectors in this study (Table 7). Compared with Silva et al (2016a), our estimate of O_3 and $PM_{2.5}$-related premature deaths attributable to PIN and TRN are very comparable, but that to RES is lower here. In comparison with Lelieveld et al (2015), we estimate greater O_3 and $PM_{2.5}$-related premature deaths attributable to PIN and TRN, but less for RES.

Like Silva et al. (2016a) and Lelieveld et al. (2015), different locations show relatively different mortality responses to changes in sectoral emissions. Whereas PIN

emission reductions cause the greatest number of avoided O_3-related premature deaths globally (19,300 (1,400, 45,000) deaths/year), TRN emission reductions cause the greatest fraction of avoided deaths in most of the six regions (26-53% of the global emission reduction), except for EAS (58%) and RBU (38%) where the effect of reducing PIN emissions dominates. In comparison with other studies (Table 7), our conclusion that PIN emissions cause the most O_3-related deaths and TRN emissions cause the greatest fraction of avoided deaths in most regions agrees well with Silva et al (2016a). For $PM_{2.5}$, reducing PIN emissions avoids the most $PM_{2.5}$-related premature deaths globally (128,000 (41,600, 179,000) deaths/year) and in most regions (38-78% of the global emission reduction), except for SAS (45%) where the RES emission dominates. Although these findings differ from those of Lelieveld et al (2015) and Silva et al (2016), who find that Residential emissions have the greatest of impact on $PM_{2.5}$ mortality globally and in most regions, all studies agree that PIN emissions have the greatest impact in NAM. Our result is also comparable with Crippa et al (2017) who find that PIN emissions have the greatest health impact in most countries. Although comparable emission inventories are used (i.e. Lelieveld et al (2015) and this study use EDGAR emissions while Silva et al (2016) use RCP8.5. emissions), our lower mortality estimate for RES emissions may be explained by our 20% reductions relative to the zero-out method, and the different years simulated.

Considering results from individual models, we found that mortality from TRN emission reductions show greater relative uncertainty than from PIN or RES (Table 5-6), reflecting a greater spread of results across models. Regional impacts from individual model also differ from the ensemble mean result - e.g., for O_3, GEOSCHEMADJOINT and OsloCTM3.v2 show that reducing PIN emissions causes the greatest fraction of avoided O_3-related deaths in EUR, while GEOSCHEMADJOINT, HadGM2-ES and OsloCTM3.v2 show that TRN emissions have the greatest fraction of avoided O_3-related deaths in RBU (Figs. S8 and S12). For $PM_{2.5}$, CHASER_t42 and GEOSCHEMADJOINT show that reducing PIN emissions causes the greatest fraction of avoided $PM_{2.5}$-related deaths in SAS (Figs. S9 and S13).

4 Discussion

We aggregate the avoided deaths attributable to 20% reductions from four corresponding source regions (i.e. NAM, EUR, SAS and EAS), and compare with the findings from TF-HTAP1. We estimate that these regional emission reductions are associated with 36,000 (-1,500, 90,300) avoided deaths globally through the change in O_3 and 207,000 (41,500, 304,000) avoided deaths through the change in $PM_{2.5}$, more than those estimated by Anenberg et al. (2009 and 2014) – 21,800 (10,600, 33,400)

deaths for O_3 and 192,000 (146,000, 230,000) deaths for $PM_{2.5}$. This discrepancy might be attributed to different health impact function, emissions data sets, region definitions, updated population or baseline mortality rates. In particular, for O_3 respiratory mortality, we use a log-linear model for chronic mortality (Jerrett et al 2009), instead of the short-term O_3 mortality estimate based on a daily time-series study (Bell et al., 2004) used by Anenberg et al., (2009). For $PM_{2.5}$ mortality, Anenberg et al., (2014) only included the simulated changes in BC, particulate organic matter (POM=primary organic aerosol+secondary organic aerosol), and sulfate for $PM_{2.5}$ concentration, while we use the total reported $PM_{2.5}$ concentration which includes more species for some models. We also apply the Integrated Exposure–Response (IER) model (Burnett et al. 2014) for $PM_{2.5}$, as opposed to the log-linear model of Krewski et al. (2009) used by Anenberg et al., (2014).

For regional reductions, our multi-model average results suggest that NAM and EUR emissions cause more deaths inside of those regions than outside, which disagrees with previous studies (Anenberg et al., 2009; Duncan et al., 2008; West et al., 2009) whereas similar regional impacts are found for EAS and SAS. Also, total avoided deaths through interregional air pollution transport are estimated as 6,000 (-3,400, 18,300) deaths/year for O_3 and 25,000 (8,000, 36,200) deaths/year for $PM_{2.5}$ in this study, in contrast with 7,300 (3,600, 11,200) deaths/year for O_3 and 11,500 (8,800, 14,200) deaths/year for $PM_{2.5}$ in Anenberg et al. (2009; 2014). These differences likely result from different concentration-response functions and the use of 6 regions here vs. 4 by Anenberg et al. (2009; 2014). Overall, whereas O_3 accounts for a higher percentage of the total deaths in foreign regions than $PM_{2.5}$, $PM_{2.5}$ leads to more deaths in general, which agrees well with the results of Anenberg et al. (2009; 2014).

Using regional models in AQMEII3, driven by a single global model (C-IFS_v2), Im et al. (2017) estimated that 20% domestic emission reductions would avoid 54,000 and 27,500 premature deaths (for O_3 and $PM_{2.5}$ combined) in Europe and the U.S., respectively, as opposed to ~1,000 and 2,000 premature deaths due to foreign emission reductions. These results are comparable to our estimates that 32,900 and 19,500 premature deaths result from 20% domestic emission reductions in Europe and the U.S., while 670 and 570 premature deaths result from foreign emission reductions. Although our defined U.S. region is slightly bigger than Im et al. (2017), the majority of U.S. emission sources and population are located within the region defined by Im et al. (2017). This comparison shows that regional and global models show similar impacts on mortality from air pollution transport.

Differences in our estimates of premature mortality attributable to air pollution from three emission sectors (multiplied by 5) may be explained by methodological differences relative to previous studies (Silva et al., 2016; Lelieveld et al., 2015),

565 including our use of 20% emission reductions versus the zero-out method in those
566 studies, different emission inventories, a multi-model ensemble versus single models,
567 and differences in baseline mortality rates, population, and concentration response
568 functions. Our finding that TRN emissions contribute the most avoided deaths for O_3
569 in most regions agrees well with the result by Silva et al (2016a), but differs for $PM_{2.5}$
570 mortality for which we find that PIN emissions cause the most deaths, while both Silva
571 et al (2016a) and Lelieveld et al (2015) find that RES emissions are responsible for the
572 most deaths. This discrepancy may be explained by different $PM_{2.5}$ species included in
573 individual models, as we showed that changes in $PM_{2.5}$ concentration to TRN emission
574 differ across models.
575 By using an ensemble of multi-model results here, we highlight the relative
576 importance of difference source-receptor pairs for mortality in a way that is more robust
577 than using a single model, particularly since some individual models yielded different
578 conclusions than the ensemble mean. The air pollutant concentration changes reported
579 by the HTAP2 models may be different among models, it may result from variety of
580 processes, e.g. atmospheric physical and chemical mechanisms, processing of natural
581 emissions, and transport time step, etc. (Table S1), but not anthropogenic emissions
582 since those were nearly identical among models. In addition, the coarse model
583 resolution used by global models may underestimate health effects by misaligning peak
584 concentration and population, particularly in urban areas and for $PM_{2.5}$ (Punger and
585 West, 2013), but it is not known how model resolution would affect the relative
586 contributions of extraregional and intraregional health benefits. Future research should
587 explore the possible bias from using coarse global models for extraregional and
588 intraregional mortality estimates in metropolitan regions by comparing with finer-
589 resolution chemical transport models.
590 Another uncertainty in this paper (and other global studies) lies in applying the
591 same RRs worldwide, because of lack of long-term records of the chronic influences of
592 ambient air pollution on mortality outside of North America and Europe. We consider
593 only the population of adults ≥25 years old, ignoring possible mortality effects on the
594 younger population, and consequently we may underestimate premature mortality
595 overall. Likewise, the effects of air pollution on several morbidity endpoints are omitted.
596 We assume that all $PM_{2.5}$ is equally toxic, for lack of clear evidence for greater toxicity
597 of some species. Inter-regional transport may also change the toxicity of $PM_{2.5}$ by
598 changing the size distribution or chemical composition, where transport likely causes
599 particles to become more oxidized (West et al., 2016). Future research on $PM_{2.5}$-related
600 mortality should include estimating health effects for different $PM_{2.5}$ chemical
601 components.
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5 Conclusions

We estimate O_3- and $PM_{2.5}$-related premature mortality from simulations with 14 global CTMs participating in the TF-HTAP2 multi-model exercise for the year 2010. An estimate of 290,000 (30,000, 600,000) global premature O_3-related deaths and 2.8 million (0.5 million, 4.6 million) global $PM_{2.5}$-related premature deaths is obtained from the ensemble for the year 2010 in the baseline case. We focus on model experiments simulating 20% regional emission reductions in 6 regions, 3 sectors and 1 global domain. For regional scenarios, source emission reductions cause 84% of the global avoided O_3-related premature deaths within the source region, ranging from 21 to 95% among 6 regions, and 16% (5 to 79%) outside of the source region. For $PM_{2.5}$, 89% of global avoided $PM_{2.5}$-related premature deaths are within the source region, ranging from 32 to 94% among 6 regions, and 11% (6 to 68%) outside of the source region. While most avoided mortality generally occurs within the source region, we find that emission reductions from RBU (only for O_3) and MDE (for both O_3 and $PM_{2.5}$) can avoid more premature deaths outside of these regions than within. Considering the effects of foreign emissions on receptor regions, 20% foreign emission reductions lead to more avoided O_3-related premature deaths in EUR, MDE and RBU than domestic reductions. Reductions from all six regions in the transport of air pollution between regions are estimated to lead to more avoided deaths through changes in $PM_{2.5}$ (42,000 (12,400, 60,100) deaths/year) than for O_3 (10,300 (6,700, 13,400) deaths/year). Overall, the spread of modeled air pollutant concentrations contributes most to the uncertainty in mortality estimates, highlighting that using a single model may lead to erroneous conclusions and may underestimate uncertainty in mortality estimates.

For sectoral emission reductions, reducing anthropogenic emissions by 20% in 3 sectors together avoids 48,500 (7,100, 108,000) O_3-related premature deaths and 243,000 (66,800, 357,000) $PM_{2.5}$-related premature deaths globally. Of the 3 sectors, TRN had the greatest fraction (26-53%) of O_3-related premature deaths globally and in most regions, except for EAS (58%) and RBU (38%) where PIN emissions dominate. For $PM_{2.5}$ mortality, PIN emissions cause the most deaths in most regions (38-78%), except for SAS (45%) where the TRN emissions dominate.

In this study, we have gone beyond previous TF-HTAP1 studies that quantified premature mortality from interregional air pollution transport, by using more source regions, analyzing source emission sectors, and using updated atmospheric models and health impact functions. Despite uncertainties, our results suggest that reducing pollution transported over a long distance would be beneficial for health, with impacts from all foreign emission reductions combined that may be comparable to or even exceed the impacts of emission reductions within a region. Additionally, actions to

reduce emissions should target specific sectors within world regions, as different sectors dominate the health effects in different regions. This work highlights the importance of long-range air pollution transport, and suggests that estimates of the health benefits of emission reductions on local, national, or continental scales may underestimate the overall health benefits globally, when interregional transport is accounted for. International cooperation to reduce air pollution transported over long distances may therefore be desirable.

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Supporting information A detailed description of the models participating in the ensemble, a map of six priority regions used in this analysis, and additional results can be found in the Supporting Information.

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Table 1. Population-weighted multi-model mean O_3 (ppb) and $PM_{2.5}$ concentration (μg/m^3) for the 2010 baseline, for the 6-month O_3 season average of 1-hr. daily maximum O_3 and annual average $PM_{2.5}$, shown with the standard deviation among models.

Scenarios	Receptor regions						
	NAM	EUR	SAS	EAS	MDE	RBU	World
O_3 (11 models)	56.51±9.40	48.38±8.05	65.72±10.08	59.10±10.46	61.11±9.79	46.79±7.53	53.74±8.03
$PM_{2.5}$ (8 models)	9.36±2.62	10.75±3.87	37.05±8.74	39.27±13.50	34.49±17.64	11.61±3.52	25.98±5.05

Table 2. Population-weighted multi-model mean change in O_3 (ppb) in receptor regions due to 20% regional (NAM, EUR, SAS, MDE and RBU), sectoral (PIN, TRN and RES) and global (GLO) anthropogenic emission reductions, for the 6-month O_3 season average of 1-hr. daily maximum. The diagonal, showing the effect of each region on itself, is underlined. All numbers are rounded to the nearest hundredth, and are shown with standard deviations among models.

Source	Receptor region						
regions/sectors	NAM	EUR	SAS	EAS	MDE	RBU	World
NAM	-1.88±0.06	-0.26±0.55	-0.04±0.14	-0.11±0.06	-0.23±0.12	-0.21±0.09	-0.19±0.07
EUR	-0.08±0.04	-0.80±0.55	0.01±0.14	-0.10±0.06	-0.34±0.08	-0.34±0.09	-0.14±0.07
SAS	-0.05±0.02	-0.04±0.02	-3.65±0.94	-0.08±0.04	-0.11±0.04	-0.04±0.03	-0.90±0.22
EAS	-0.29±0.14	-0.25±0.13	-0.09±0.22	-1.96±1.10	-0.23±0.12	-0.27±0.12	-0.58±0.25
MDE	-0.04±0.02	-0.05±0.01	-0.07±0.15	-0.03±0.01	-1.23±0.66	-0.11±0.01	-0.09±0.04
RBU	-0.05±0.04	-0.13±0.05	0.03±0.16	-0.08±0.06	-0.10±0.07	-0.45±0.38	-0.05±0.06
PIN	-1.13±0.28	-0.70±0.19	-1.43±0.18	-1.58±0.88	-1.09±0.45	-0.69±0.31	-1.11±0.25
TRN	-1.26±0.42	-0.81±0.34	-2.05±0.32	-0.73±0.32	-1.40±0.17	-0.71±0.19	-1.13±0.19
RES	-0.24±0.09	-0.21±0.04	-1.19±0.44	-0.62±0.10	-0.23±0.06	-0.18±0.03	-0.57±0.14
GLO	-2.86±0.77	-1.98±0.66	-4.40±1.04	-2.77±1.21	-2.84±0.70	-1.76±0.52	-2.82±0.53

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Table 3. Population-weighted multi-model annual average change in $PM_{2.5}$ concentrations (μg/m^3) in receptor regions due to 20% regional (NAM, EUR, SAS, MDE and RBU), sectoral (PIN, TRN and RES) and global (GLO) anthropogenic emission reductions. The diagonal, showing the effect of each region on itself, is underlined. All numbers are rounded to the nearest hundredth, and are shown with standard deviations among models.

Source	Receptor region						
regions/sectors	NAM	EUR	SAS	EAS	MDE	RBU	World
NAM	-1.33±0.66	-0.03±0.02	0.00±0.01	-0.02±0.02	-0.01±0.01	-0.01±0.01	-0.08±0.04
EUR	-0.01±0.00	-1.17±0.87	-0.01±0.01	-0.02±0.01	-0.18±0.08	-0.26±0.19	-0.13±0.09
SAS	<-0.01	<-0.01	-4.86±2.17	-0.08±0.08	-0.03±0.02	<-0.01	-1.16±0.51
EAS	-0.03±0.01	-0.02±0.01	-0.08±0.07	-6.19±3.08	<-0.01	-0.04±0.02	-1.45±0.71
MDE	<-0.01	-0.03±0.01	-0.12±0.06	-0.01±0.02	-0.91±0.38	-0.05±0.03	-0.08±0.03
RBU	<-0.01	-0.07±0.05	-0.01±0.02	-0.04±0.02	-0.03±0.02	-0.78±0.50	-0.05±0.03
PIN	-0.61±0.18	-0.57±0.26	-1.73±0.71	-2.75±0.99	-0.92±0.14	-0.58±0.19	-1.46±0.56
TRN	-0.27±0.20	-0.38±0.41	-0.82±0.88	-0.54±0.43	-0.09±0.06	-0.15±0.16	-0.40±0.37
RES	-0.20±0.05	-0.27±0.12	-1.93±0.40	-1.70±0.28	-0.08±0.02	-0.20±0.05	-1.17±0.31
GLO	-1.47±0.72	-1.52±1.04	-5.40±2.31	-6.76±3.29	-1.55±0.75	-1.19±0.73	-3.49±1.51

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Table 4. Annual multi-model empirical mean O_3- and $PM_{2.5}$-related premature deaths with 95% CI from Monte-Carlo simulations in parenthesis (including uncertainty in baseline mortality rates, RRs and air pollutant concentration across models) in year 2010 baseline. All numbers are rounded to three significant figures or the nearest 100 deaths.

	Receptor region						
	NAM	EUR	SAS	EAS	MDE	RBU	World
O_3 (11 models)	15,000 (900－30,000)	13,000 (600－28,000)	136,000 (23,000－277,000)	100,000 (3,900－213,000)	3,200 (300－7,000)	2,900 (100－6,600)	291,000 (30,000－596,000)
$PM_{2.5}$ (8 models)	72,000 (1,500－158,000)	203,000 (2,700－463,000)	732,000 (328,000－1,110,000)	1,120,000 (159,000－1,720,000)	79,000 (600－133,000)	177,000 (2,700－358,000)	2,770,000 (514,000－4,640,000)

931 Table 5. Annual avoided multi-model empirical mean O_3-related premature respiratory deaths with 95% CI from Monte-Carlo simulations in
932 parenthesis due to 20 % regional (NAM, EUR, SAS, MDE and RBU), sectoral (PIN, TRN and RES) and global (GLO) anthropogenic emission
933 reductions in each region and worldwide. The diagonal, showing the effect of each region on itself, is underlined. All numbers are rounded to three
934 significant figures or the nearest 10 deaths.

Source regions/sectors	Receptor region						
	NAM	EUR	SAS	EAS	MDE	RBU	World
NAM	1,500 (-170—4,000)	330 (10—780)	170 (-250—690)	500 (-910—2,200)	30 (0—80)	70 (0—170)	2,800 (-1,300—8,400)
EUR	60 (-80—240)	930 (-70—2,400)	-80 (-880—670)	490 (-1,100—2,300)	50 (10—110)	110 (10—250)	1,700 (-490—4,900)
SAS	40 (-40—130)	50 (-30—160)	19,000 (4,000—42,000)	420 (-340—1,400)	20 (0—40)	10 (-10—40)	20,000 (3,600—42,200)
EAS	230 (-50—630)	310 (-50—850)	450 (-1,300—2,400)	9,700 (-2,000—26,400)	30 (0—100)	80 (-10—230)	11,400 (-3,300—31,800)
MDE	30 (-30—120)	60 (-50—190)	310 (-90—910)	160 (-120—520)	180 (-10—480)	30 (0—70)	870 (-330—2,600)
RBU	40 (-60—170)	150 (-50—440)	-200 (-1,700—1,200)	420 (-620—1,700)	20 (-10—60)	140 (-60—420)	640 (120—1,300)
PIN	900 (100—2,100)	850 (40—2,100)	7,400 (1,800—15,400)	7,800 (3,100—20,900)	140 (30—330)	210 (-100—650)	19,300 (1,400—45,000)
TRN	1,000 (-20—2,600)	970 (-270—2,800)	10,600 (2,600—22,000)	3,500 (-420—9,300)	210 (50—440)	200 (20—490)	18,800 (3,000—41,600)
RES	200 (-20—510)	250 (40—550)	6,000 (1,600—12,200)	3,000 (670—6,300)	30 (0—80)	60 (10—120)	10,400 (2,700—21,100)
GLO	2,300 (80—5,600)	2,400 (250—5,400)	22,600 (6,200—46,000)	13,500 (1,500—30,300)	400 (80—940)	550 (80—1,210)	47,400 (11,300—99,000)

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936 Table 6. Annual avoided multi-model empirical mean $PM_{2.5}$-related premature deaths (IHD+STROKE+COPD+LC) with 95% CI from Monte-
937 Carlo simulations in parenthesis due to 20 % regional (NAM, EUR, SAS, MDE and RBU), sectoral (PIN, TRN and RES) and global (GLO)
938 anthropogenic emission reductions in each region and worldwide. The diagonal, showing the effect of each region on itself, is underlined. All
939 numbers are rounded to three significant figures or the nearest 10 deaths.

Source regions/sectors	Receptor region NAM	EUR	SAS	EAS	MDE	RBU	World
NAM	18,000 (630－28,300)	640 (80－1,100)	10 (-210－80)	200 (-300－370)	10 (0－30)	250 (90－420)	19,400 (310－30,600)
EUR	60 (20－110)	31,900 (4,500－53,900)	120 (-60－190)	390 (-20－550)	400 (30－1,400)	2,700 (680－8,000)	39,400 (5,500－63,400)
SAS	50 (-10－90)	110 (0－200)	47,900 (30,000－68,500)	1,400 (-70－2,100)	40 (0－150)	40 (10－110)	51,300 (32,300－73,300)
EAS	340 (40－510)	400 (20－690)	900 (590－1,400)	91,100 (440－128,700)	10 (0－30)	800 (0－1,300)	96,600 (3,500－136,000)
MDE	30 (0－60)	420 (90－850)	1,400 (740－2,400)	180 (-610－460)	1,600 (240－4,500)	640 (30－1,600)	5,000 (1,900－11,100)
RBU	40 (10－60)	2,200 (300－3,700)	90 (-220－190)	810 (330－1,100)	80 (10－220)	17,600 (390－25,700)	21,500 (900－31,000)
PIN	9,300 (940－13,000)	15,700 (1,900－24,700)	21,000 (8,400－30,700)	47,310 (22,600－69,700)	2,200 (200－6,100)	14,300 (0－24,100)	128,000 (41,600－179,000)
TRN	3,600 (-320－7,000)	8,900 (130－17,400)	6,200 (-12,800－14,400)	6,800 (-6,400－12,200)	230 (10－770)	3,100 (0－5,400)	31,900 (-16,500－58,300)
RES	2,900 (110－4,400)	6,900 (210－11,300)	25,000 (15,100－40,700)	29,300 (13,200－52,900)	200 (10－520)	4,600 (0－8,100)	83,400 (41,700－120,000)
GLO	19,900 (710－31,300)	40,900 (4,900－68,100)	55,300 (36,500－78,300)	105,000 (4,000－147,000)	2,800 (330－8,400)	26,700 (2,300－36,000)	290,000 (67,100－405,000)

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941 Table 7. Comparison of O_3 and $PM_{2.5}$-related premature deaths attributable to PIN,
942 TRN and RES emissions with previous studies. Results from this study (for 20%
943 reductions) are multiplied by 5. For Silva et al. (2016), we combine results for "Energy"
944 and "Industry" to represent PIN, and use "Land transportation" to represent TRN and
945 "Residential & Commercial" to represent RES. For Lelieveld et al. (2015), we combine
946 the "Power generation" and "Industry" sectors to represent PIN, and use "Land Traffic"
947 to represent TRN, and "Residential Energy" to represent RES.

Emission source sector	This study	Silva et al. (2016)	Lelieveld et al. (2015)
PIN	O_3: 96,500 (7,000, 225,000) $PM_{2.5}$: 640,000 (208,000, 895,000)	O_3 : 111,000 (23,200, 240,000) $PM_{2.5}$:613,000 (422,000, 816,000)	$O_3 + PM_{2.5}$ (692,000)
TRN	O_3: 94,000 (15,000, 208,000) $PM_{2.5}$: 160,000 (-82,500, 292,000)	O_3: 80,900 (17,400, 180,000) $PM_{2.5}$: 212,000 (114,000, 292,000)	$O_3 + PM_{2.5}$ (165,000)
RES	O_3: 52,000 (13,500, 106,000) $PM_{2.5}$:417,000 (209,000, 600,000)	O_3: 53,700(12,300, 116,000) $PM_{2.5}$:675,000 (428,000, 899,000)	$O_3 + PM_{2.5}$ (1,020,000)

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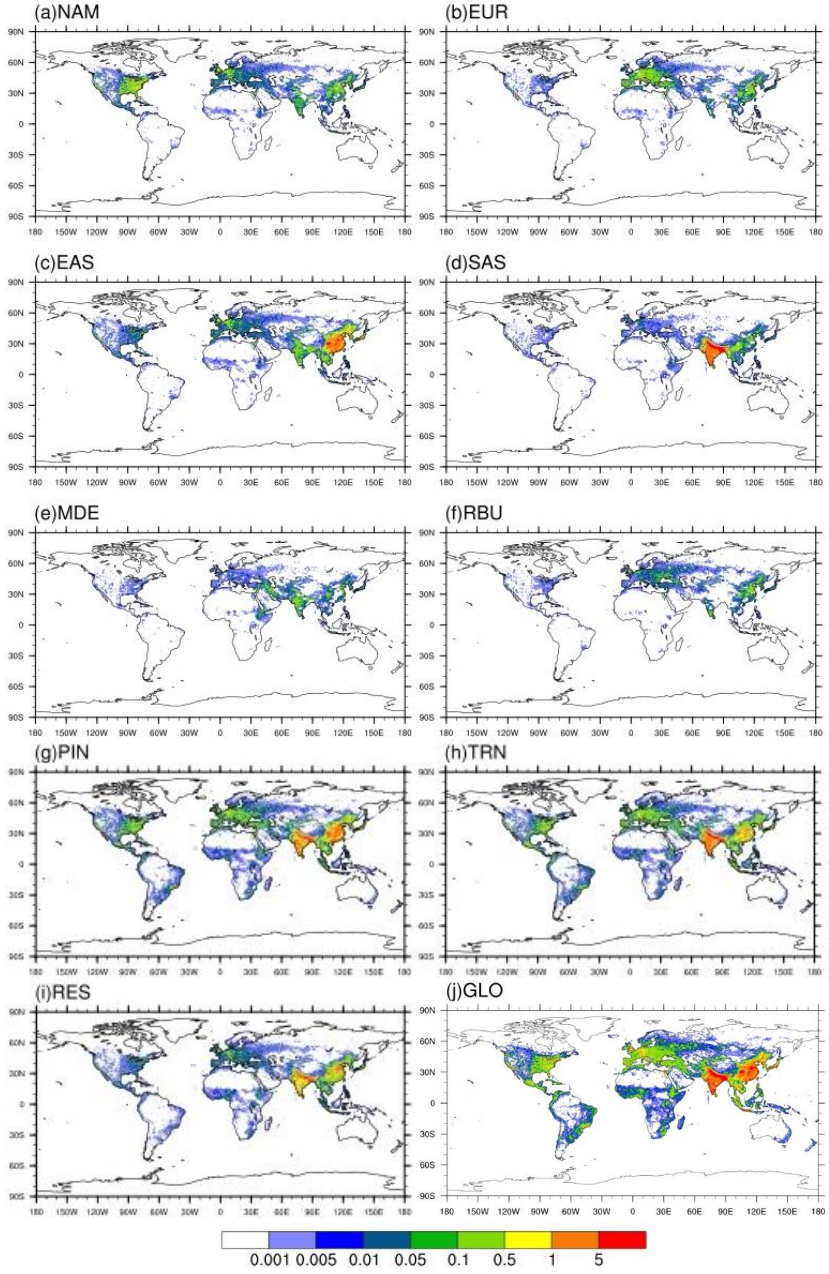

Figure 1. Annual avoided O_3-related premature deaths in 2010 per 1,000 km^2 due to 20 % emission reduction scenarios relative to the base case in a) North America (NAM), b) Europe (EUR), c) East Asia (EAS), d) South Asia (SAS), e) Middle East (MDE), f) Russia/Belarus/Ukraine (RBU), g) Power and Industry (PIN), h) Transportation (TRN), i) Residential (RES) and j) Global (GLO).

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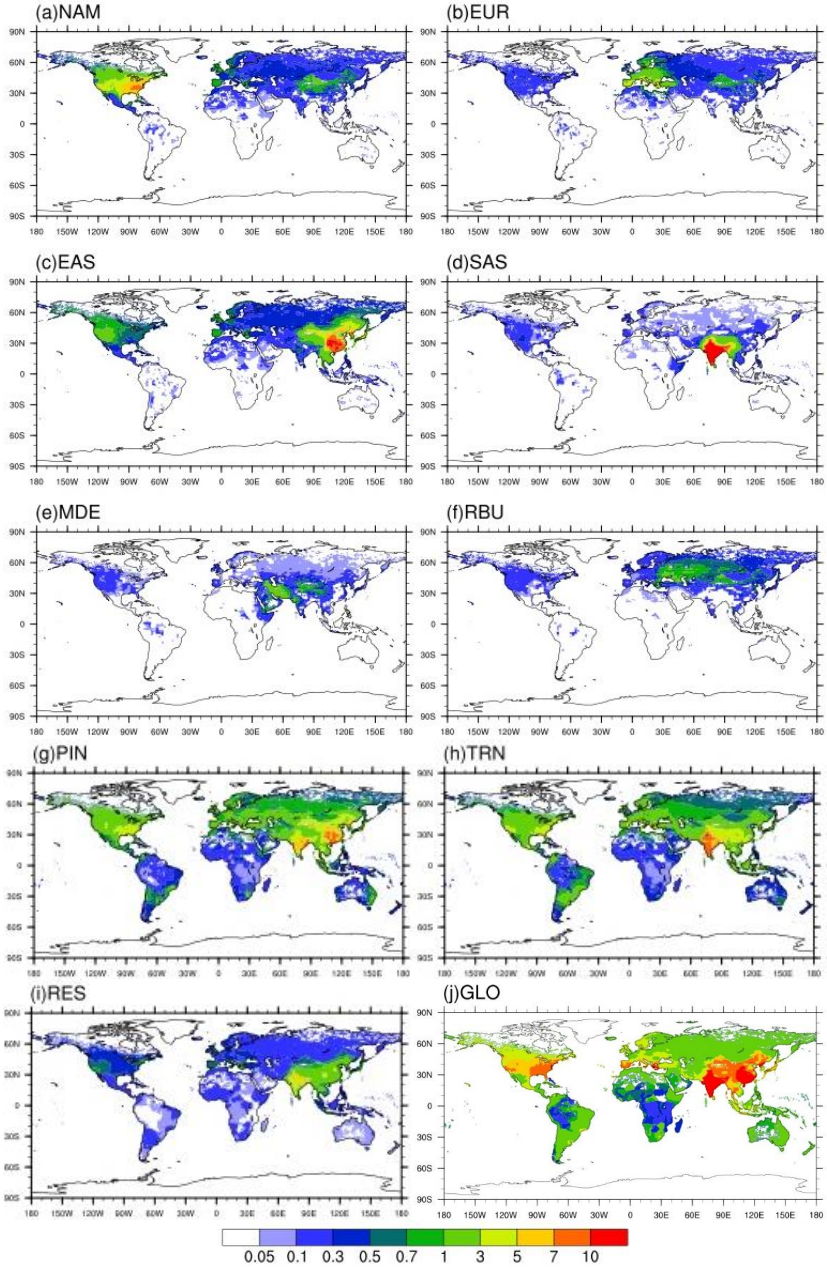

Figure 2. Annual avoided O_3-related premature deaths in 2010 per million people due to 20 % emission reduction scenarios relative to the base case in a) North America (NAM), b) Europe (EUR), c) East Asia (EAS), d) South Asia (SAS), e) Middle East (MDE), f) Russia/Belarus/Ukraine (RBU), g) Power and Industry (PIN), h) Transportation (TRN), i) Residential (RES) and j) Global (GLO)

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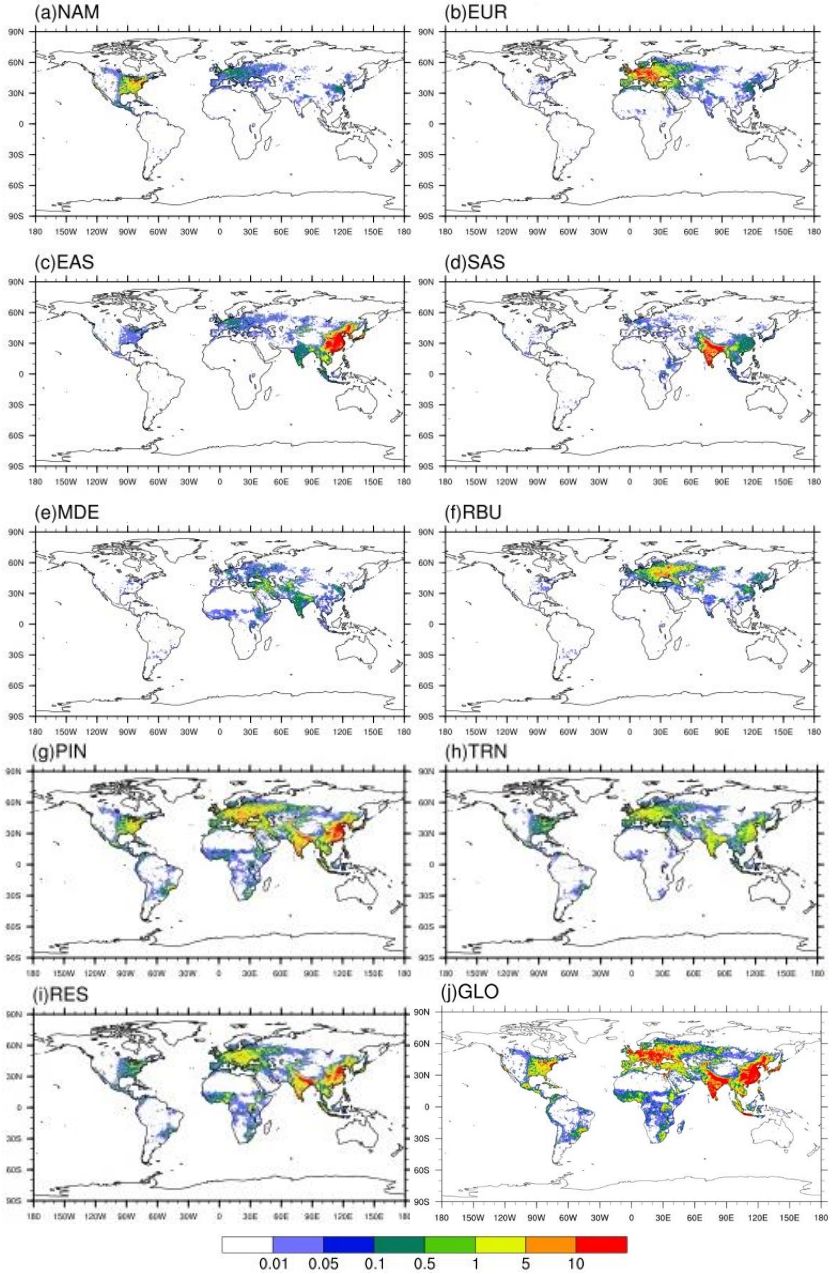

Figure 3. Annual avoided $PM_{2.5}$-related premature deaths in 2010 per 1,000 km^2 due to 20 % emission reduction scenarios relative to the base case in a) North America (NAM), b) Europe (EUR), c) East Asia (EAS), d) South Asia (SAS), e) Middle East (MDE), f) Russia/Belarus/Ukraine (RBU), g) Power and Industry (PIN), h) Transportation (TRN), i) Residential (RES) and j) Global (GLO).

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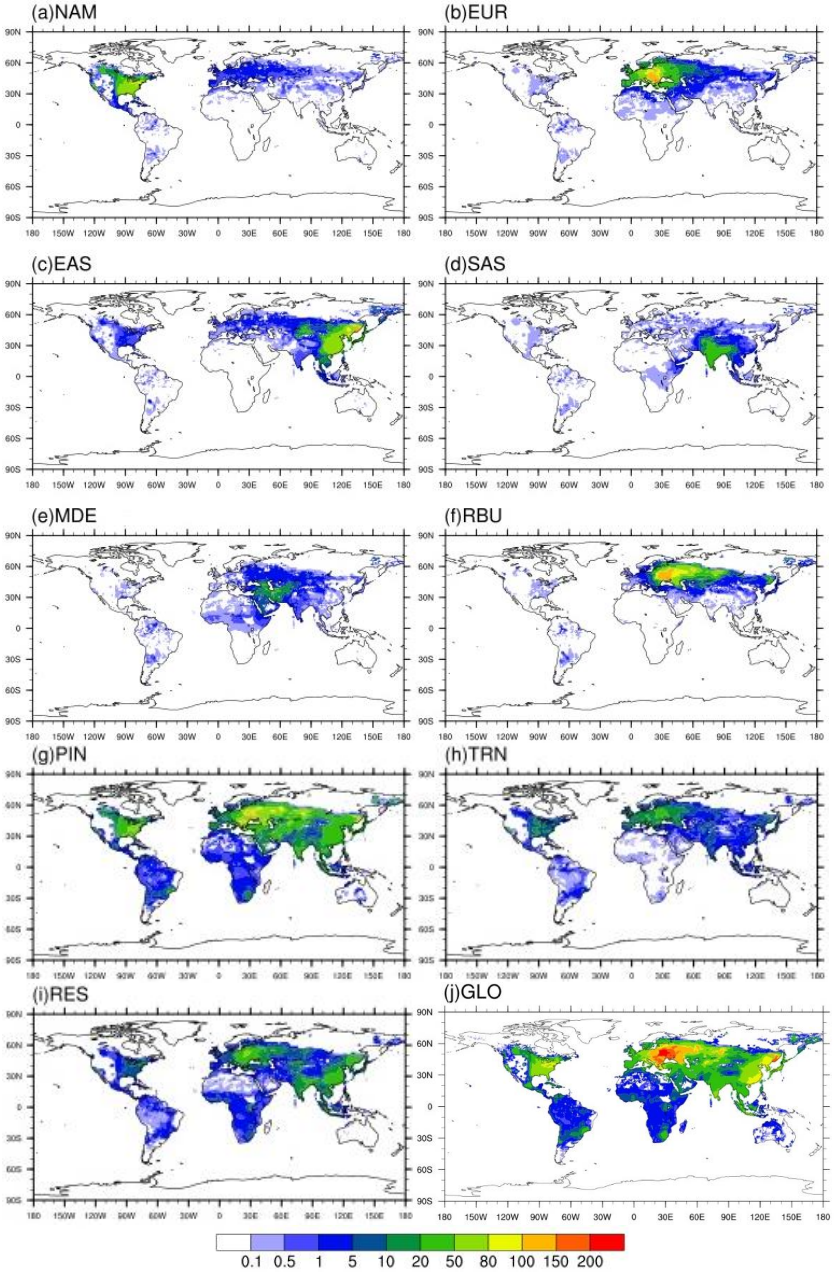

Figure 4. Annual avoided $PM_{2.5}$-related premature deaths in 2010 per million people due to 20 % emission reduction scenarios) relative to the base case in a) North America (NAM), b) Europe (EUR), c) East Asia (EAS), d) South Asia (SAS), e) Middle East (MDE), f) Russia/Belarus/Ukraine (RBU), g) Power and Industry (PIN), h) Transportation (TRN), i) Residential (RES) and j) Global (GLO).