Assessment and economic valuation of air pollution impacts on human health over Europe and the United States as calculated by a multi-model ensemble in the framework of AQMEII3

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Abstract

The impact of air pollution on human health and the associated external costs in Europe and the United States (U.S.) for the year 2010 is modelled by a multi-model ensemble of regional models in the frame of the third phase of the Air Quality Modelling Evaluation International Initiative (AQMEII3). The modelled surface concentrations of O_3, CO, SO_2 and $PM_{2.5}$ are used as input to the Economic Valuation of Air Pollution (EVA) system to calculate the

resulting health impacts and the associated external costs from each individual model. Along with a base case simulation, additional runs were performed introducing 20% anthropogenic emission reductions both globally and regionally in Europe, North America and East Asia, as defined by the second phase of the Task Force on Hemispheric Transport of Air Pollution (TF-HTAP2).

Health impacts estimated by using concentration inputs from different chemistry and transport models (CTMs) to the EVA system can vary up to a factor of three in Europe (twelve models) and the United States (three models). In Europe, the multi-model mean total number of premature deaths (acute + chronic) is calculated to be 414 000 while in the U.S., it is estimated to be 160 000, in agreement with previous global and regional studies. The economic valuation of these health impacts are calculated to be 300 and 145 billion Euros in Europe and the U.S., respectively. A subset of models that produce the smallest error compared to the surface observations at each time step against an all-models mean ensemble results in increase of health impacts by up to 30% in Europe, while in the U.S., the optimal ensemble mean led to a decrease in the calculated health impacts by ~11%.

A total of 54 000 and 27 500 premature deaths can be avoided by a 20% reduction of global anthropogenic emissions in Europe and the U.S., respectively. A 20% reduction of North American anthropogenic emissions avoids a total premature death of ~1 000 in Europe and 25 000 total premature deaths in the U.S. A 20% decrease of anthropogenic emissions within the European source region avoids a total premature death of 47 000 in Europe. Reducing the East Asian anthropogenic emissions by 20% avoids ~2000 total premature deaths in the U.S. These results show that the domestic anthropogenic emissions make the largest impacts on premature death on a continental scale, while foreign sources make a minor contributing to adverse impacts of air pollution.

1. Introduction

According to the World Health Organization (WHO), air pollution is now the world's largest single environmental health risk (WHO, 2014). Around 7 million people died prematurely in 2012 as a result of air pollution exposure from both outdoor and indoor emission sources (WHO, 2014). WHO estimates 3.7 million premature deaths in 2012 from exposure to outdoor air pollution from urban and rural sources worldwide. According to the Global Burden of Disease (GBD) study, exposure to ambient particulate matter pollution remains among the ten leading risk factors. Air pollution is a transboundary phenomenon with global, regional, national and local sources, leading to large differences in the geographical distribution of human exposure. Short-term exposure to ozone (O_3) is associated with respiratory morbidity and mortality (e.g. Bell et al., 2004), while long-term exposure to O_3 has been associated with premature respiratory mortality (Jerrett et al., 2009). Short-term exposure to particulate matter ($PM_{2.5}$) has been associated with increases in daily mortality rates from respiratory and cardiovascular causes (e.g. Pope and Dockery, 2006), while long-term exposure to $PM_{2.5}$ can have detrimental chronic health effects, including premature mortality due to cardiopulmonary diseases and lung cancer (Burnett et al., 2014). The Global

Burden of Disease Study 2015 estimated 254 000 O_3-related and 4.2 million anthropogenic $PM_{2.5}$-related premature deaths per year (Cohen et al., 2017).

Changes in emissions from one region can impact air quality over others, affecting also air pollution-related health impacts due to intercontinental transport (Anenberg et al., 2014; Zhang et al., 2017). In the framework of the Task Force on Hemispheric Transport of Air Pollution (TF-HTAP), Anenberg et al. (2009) found that reduction of foreign ozone precursor emissions can contribute to more than 50% of the deaths avoided by simultaneously reducing both domestic and foreign precursor emissions. Similarly, they found that reducing emissions in North America (NA) and Europe (EU) has largest impacts on ozone-related premature deaths in downwind regions than within (Anenberg et al., 2009). This result agrees with Duncan et al. (2008), which showed for the first time that emission reductions in NA and EU have greater impacts on ozone mortality outside the source region than within. Anenberg et al. (2014) estimates that 93–97 % of $PM_{2.5}$-related avoided deaths from reducing emissions occurs within the source region while 3–7 % occur outside the source region from concentrations transported between continents. In spite of the shorter lifetime of $PM_{2.5}$ compared to O_3, it was found to cause more deaths from intercontinental transport (Anenberg et al., 2009; 2014). In the frame of the second phase of the Task Force on Hemispheric Transport of Air Pollution (TF-HTAP2; Galmarini et al., 2017), an ensemble of global chemical transport model simulations calculated that 20% emission reductions from one region generally lead to more avoided deaths within the source region than outside (Liang et al., 2017).

Recently, Lelieveld et al. (2015) used a global chemistry model and calculated that outdoor air pollution led to 3.3 million premature deaths globally in 2010. They calculated that in Europe and North America, 381 000 and 68 000 premature deaths occurred, respectively. They have also calculated that these numbers are likely to roughly double in the year 2050 assuming a business-as-usual scenario. Silva et al. (2016), using the ACCMIP model ensemble, calculated that the global mortality burden of ozone is estimated to markedly increase from 382 000 deaths in 2000 to between 1.09 and 2.36 million in 2100. They also calculated that the global mortality burden of $PM_{2.5}$ is estimated to decrease from 1.70 million deaths in 2000 to between 0.95 and 1.55 million deaths in 2100. Silva et al. (2013) estimated that in 2000, 470 000 premature respiratory deaths are associated globally and annually with anthropogenic ozone, and 2.1 million deaths with anthropogenic $PM_{2.5}$-related cardiopulmonary diseases (93%) and lung cancer (7%). These studies employed global chemistry and transport models with coarse spatial resolution ($\geq 0.5° \times 0.5°$), therefore health benefits from reducing local emissions were not able to be adequately captured. Higher resolutions are necessary to calculate more robust estimates of health benefits from local vs. non-local sources (Fenech et al., 2017). In addition, these studies calculated number of premature deaths due to air pollution, however none of them addresses morbidity such as number of lung cancer or asthma cases, or restricted activity days. Finally, these studies did not include economic costs either. On the other hand, there are a number of regional studies that calculate health impacts on finer spatial resolutions, and address morbidity. However, they are mostly based on single air pollution models or do not evaluate the health benefits

from local vs. non-local emissions. Therefore, a comprehensive study employing multi model ensemble of high spatial resolution and focusing on both mortality and morbidity from local vs. non-local sources lacks in the literature.

In Europe, recent results show that outdoor air pollution due to O_3, CO, SO_2 and $PM_{2.5}$ causes a total number of 570 000 premature deaths in the year 2011 (Brandt et al., 2013a; 2013b). The external (or indirect) costs to society related to health impacts from air pollution are tremendous. OECD (2014) estimates that outdoor air pollution is costing its member countries USD 1.57 trillion in 2010. Among the OECD member countries, the economic valuation of air pollution in the U.S. was calculated to be ~500 billion USD and ~660 USD in Europe. In the whole of Europe, the total external costs have been estimated to approx. 800 billion Euros in year 2011 (Brandt et al., 2013a). These societal costs have great influence on the general level of welfare and especially on the distribution of welfare both within the countries as air pollution levels are vastly heterogeneous both at regional and local scales and between the countries as air pollution and the related health impacts are subject to long-range transport. Geels et al. (2015), using two regional chemistry and transport models, estimated a premature mortality of 455 000 and 320 000 in Europe (EU28 countries) for the year 2000, respectively, due to O_3, CO, SO_2 and $PM_{2.5}$. They also estimated that climate change alone leads to a small increase (15%) in the total number of O_3-related acute premature deaths in Europe towards the 2080s and relatively small changes (<5%) for $PM_{2.5}$-related mortality. They found that the combined effect of climate change and emission reductions will reduce the premature mortality due to air pollution, in agreement with the results from Schucht et al. (2015).

The U.S. Environmental Protection Agency estimated that in 2010 there were ~160 000 premature deaths in the U.S. due to air pollution (U.S. EPA, 2011). Fann et al. (2012) calculated 130,000 - 350,000 premature deaths associated with O_3 and $PM_{2.5}$from the anthropogenic sources in the U.S. for the year 2005. Caizzo et al. (2013) estimated 200 000 cases of premature death in the U.S. due to air pollution from combustion sources for the year 2005.

The health impacts of air pollution and their economic valuation are estimated based on observed and/or modelled air pollutant concentrations. Observations have spatial limitations particularly when assessments are needed for large regions. The impacts of air pollution on health can be estimated using models, where the level of complexity can vary depending on the geographical scale (global, continental, country or city), concentration input (observations, model calculations, emissions) and the pollutants of interest that can vary from only few ($PM_{2.5}$ or O_3) to a whole set of all regulated pollutants. The health impact models normally used may differ in the geographical coverage, spatial resolutions of the air pollution model applied, complexity of described processes, the exposure-response functions (ERFs), population distributions and the baseline indices (see Anenberg et al., 2015 for a review).

Air pollution related health impacts and associated costs can be calculated using Chemical Transport Model (CTM) or with standardized source-receptor relationships characterizing the dependence of ambient concentrations on emissions. (e.g. EcoSense model: ExternE, 2005,

TM5-FASST: Van Dingenen et al., 2014). Source-receptor relationships have the advantage of reducing the computing time significantly and have therefore been extensively used in systems like GAINS (Amann et al., 2011). On the other hand, full CTM simulations have the advantage of better accounting for non-linear chemistry-transport processes in the atmosphere.

CTMs are useful tools to calculate the concentrations of health-related pollutants taking into account non-linearities in the chemistry and the complex interactions between meteorology and chemistry. However, the CTMs include different chemical and aerosol schemes that introduce differences in the representation of the atmosphere as well as differences in the emissions and boundary conditions they use (Im et al., 2015a,b). These different approaches are present also in the health impact estimates that use CTM results as basis for their calculations. Multi-model (MM) ensembles can be useful to the extent that allow us to take into consideration several model results at the same time, define the relative weight of the various members in determining the mean behavior, and produce also an uncertainty estimated based on the diversity of the results (Potempski and Galmarini, 2010; Riccio et al., 2013; Solazzo et al., 2013).

The third phase of the Air Quality Modelling Evaluation International Initiative (AQMEII3) project brought together fourteen European and North American modelling groups to simulate the air pollution levels over the two continental areas for the year 2010 (Galmarini et al., 2017). Within AQMEII3, the simulated surface concentrations of health related air pollutants from each modelling group serves as input to the Economic Valuation of Air Pollution (EVA) model (Brandt et al., 2013a; 2013b). The EVA model is used to calculate the impacts of health-related pollutants on human health over the two continents as well as the associated external costs. EVA model has also been tested and validated for the first time outside Europe. We adopt a multi-model ensemble (MM) approach, in which the outputs of the modelling systems are statistically combined assuming equal contribution from each model and used as input for the EVA model. In addition, the human health impacts (and the associated costs) of reducing anthropogenic emissions, globally and regionally have been calculated, allowing to quantify the trans-boundary benefits of emission reduction strategies. Finally, following the conclusions of Solazzo and Galmarini (2015), the health impacts have been calculated using an optimal ensemble of models, determined by error minimization . This approach can assess the health impacts with reduced model bias, which we can then compare with the classically derived estimates based on model averaging.

2. Material and Methods

2.1. AQMEII

2.1.1. Participating Models

In the framework of the AQMEII3 project, fourteen groups participated to simulate the air pollution levels in Europe and North America for the year 2010. In the present study, we use results from the thirteen groups that provided all health-related species (Table 1). As seen in Table 1, six groups have operated the CMAQ model. The main differences among the CMAQ

runs reside in the number of vertical levels and horizontal spacing (Table 1) and in the estimation of biogenic emissions. UK1, DE1, and US3 calculated biogenic emissions using the BEIS (Biogenic Emission Inventory System version 3) model, while TR1, UK1, and UK2 calculated biogenic emissions through the MEGAN model (Guenther et al., 2012). Moreover, DE1 does not include the dust module, while the other CMAQ instances use the inline calculation (Appel et al., 2013) and TR1 uses the dust calculation previously calculated for AQMEII Phase 2. Finally, all runs were carried out using CMAQ version 5.0.2 except for TR1, which is based on the 4.7.1 version. The gas-phase mechanisms and the aerosol models are used by each group is also presented in Table 1.More details of the model system are provided in the supplementary material. The differences in the meteorological drivers and aerosol modules can lead to substantial differences in modelled concentrations (Im et al., 2015b).

2.1.2. Emission and Boundary Conditions

The base-case emission inventories that are used in AQMEII for Europe and North America are extensively described in Pouliot et al. (2015). For Europe, the 2009 inventory of TNO-MACC anthropogenic emissions was used. In regions not covered by the emission inventory, such as North Africa, five modelling systems have complemented the standard inventory with the HTAPv2.2 datasets (Janssens-Maenhout et al., 2015). For the North American domain, the 2008 National Emission Inventory was used as the basis for the 2010 emissions, providing the inputs and datasets for processing with the SMOKE emissions processing system (Mason et al., 2012). For both continents the regional scale emission inventories were embedded in the global scale inventory (Janssens-Maenhout et al., 2015) used by the global-scale HTAP2 modelling community so that to guarantee coherence and harmonization of the information used by the regional scale modelling community. The annual totals for European and North American emissions in the HTAP inventory are the same as the MACC and SMOKE emissions. However, there are differences in the temporal distribution, chemical speciation and the vertical distribution used in the models. The C-IFS model (Flemming et al., 2015 and 2017) provided chemical boundary conditions. The C-IFS model has been extensively evaluated in Flemming et al. (2015 and 2017), and in particular for North America (Hogrefe et al., 2017; Huang et al., 2017). Galmarini et al. (2017) provides more details on the setup of the AQMEII3 and HTAP2 projects.

2.1.3. Model Evaluation

The models' performance on simulating the surface concentrations of the health-related pollutants were evaluated using Pearson's Correlation (*r*), normalized mean bias (*NMB*), normalized mean gross error (*NMGE*) and root mean square error (*RMSE*) to compare the modelled and observed hourly pollutant concentrations over surface measurement stations in the simulation domains. The hourly modelled vs. observed pairs are averaged and compared on a monthly basis. The modelled hourly concentrations were first filtered based on observation availability before the averaging has been performed. The observational data used in this study are the same as the dataset used in second phase of AQMEII (Im et al., 2015a, b). Surface observations are provided in the Ensmeble system (http://ensemble2.jrc.ec.europa.eu/public/) that is hosted at the Joint Research Centre (JRC).

Observational data were originally derived from the surface air quality monitoring networks operating in EU and NA. In EU, surface data were provided by the European Monitoring and Evaluation Programme (EMEP, 2003; http://www.emep.int/) and the European Air Quality Database (AirBase; http://acm.eionet.europa.eu/databases/airbase/). In NA observational data were obtained from the NAtChem (Canadian National Atmospheric Chemistry) database and from the Analysis Facility operated by Environment Canada (http://www.ec.gc.ca/natchem/).

The model evaluation has been conducted for 491 European and 626 North American stations for O_3, 541 European stations and 37 North American stations for CO, 500 European station and 277 North American stations for SO_2, and 568 European stations and 156 North American stations for $PM_{2.5}$.

2.1.4. Emissions Perturbations

In addition to the base case simulations in AQMEII3, a number of emission perturbation scenarios have been simulated (Table 1). The perturbation scenarios feature a reduction of 20% in the global anthropogenic emissions (GLO) as well as the HTAP2-defined regions of Europe (EUR), North America (NAM) and East Asia (EAS), as explained in detail in Galmarini et al. (2017) and Im et al. (2017). To prepare these scenarios, both the regional models and the global C-IFS model that provides the boundary conditions to the participating regional models have been operated with the reduced emissions. The global perturbation scenario (GLO) reduces the global anthropogenic emissions by 20%, introducing a change in the boundary conditions as well as a 20% decrease in the anthropogenic emissions used by the regional models. The North American perturbation scenario (NAM) reduces the anthropogenic emissions in North America by 20%, introducing a change in the boundary conditions while anthropogenic emissions remain unchanged for Europe, showing the impact of long-range transport while for North America, while the scenarios introduces a 20% reduction of anthropogenic emissions in the HTAP-defined North American region. The European perturbation scenario (EUR) reduces the anthropogenic emissions in the HTAP-defined Europe domain by 20%, introducing a change in the anthropogenic emissions while boundary conditions remain unchanged in the regional models, showing the contribution from the domestic anthropogenic emissions only. Finally, the East Asian perturbation scenario (EAS) reduces the anthropogenic emissions in East Asia by 20%, introducing a change in the boundary conditions while anthropogenic emissions remain unchanged in the regional models, showing the impact of long-range transport from East Asia on the NA concentrations.

2.2. Health Impact Assessment

All modeling groups interpolate their model outputs on a common 0.25°×0.25° resolution AQMEII grid predefined for Europe (30°W - 60°E, 25°N - 70°N) and North America (130°W - 59.5°W, 23.5°N - 58.5°N). All the analyses performed in the present study use the pollutant concentrations on these final grids. Health impacts are first calculated for each individual model and then the ensemble mean, median and standard deviation are calculated

for each health impact. In order to be able to estimate an uncertainty in the health impacts calculations, none of the models were removed from the ensemble.

Along with the individual health impact estimates from each model, a multi-model mean dataset (MM_m, in which all the modelling systems are averaged assuming equally weighted contributions) has been created for each grid cell and time step, hence creating a new model set of results that have the same spatial and temporal resolution of the ensemble-contributing members. In addition to this simple MM_m, an optimal MM ensemble (MM_{opt}) has been generated. MM_{opt} is created following the criteria extensively discussed and tested in the previous phases of the AQMEII activity (Riccio et al., 2012; Kioutsioukis et al., 2016; Solazzo and Galmarini, 2016), where it was shown that there are several ways to combine the ensemble members to obtain a superior model, mostly depending on the feature we wish to promote (or penalize). For instance, generating an optimal ensemble that maximizes the accuracy would require a minimization of the mean error or of the bias, while maximizing the associativity (variability) would require maximize the correlation coefficient (standard deviation). In this study, the sub-set of models whose mean minimize the mean squared error (*MSE*) is selected as optimal (MM_{opt}). MM_m and MM_{opt} have therefore the same spatial resolution with the individual models. The *MSE* is chosen for continuity with previous AQMEII-related works. The *MSE* is chosen in the light of its property of being composed by bias, variance and covariance types of error, thus lumping together measures of accuracy (bias), variability (variance) and associativity (covariance) (Solazzo and Galmarini, 2016). The minimum *MSE* has been calculated at the monitoring stations, where observational data are available and then extended to the entire continental areas. This approximation might affect remote regions away from the measurements. However, considering that for the main pollutants (O_3 and $PM_{2.5}$) the network of measurements is quite dense around densely populated areas (where the inputs of the MM ensemble are used for assessing the impact of air pollutants on the health of the population), errors due to inaccurate model selection in remote regions might be regarded as negligible (Solazzo and Galmarini, 2015). It should be noted that the selection of the optimal combinations of models is affected by the model's bias that might stem from processes that are common to all members of the ensemble (e.g. emissions). Therefore, such a common bias does not cancel out when combining the models, possibly creating a biased ensemble. Current work is being devoted to identify the optimal combinations of models from which the offsetting bias is removed (Solazzo et al., 2017b).

2.2.1. EVA System

The EVA system (Brandt et al., 2013a, b) is based on the impact-pathway chain (e.g. Friedrich and Bickel, 2001), consisting of the emissions, transport and chemical transformation of air pollutants, population exposure, health impacts and the associated external costs. The EVA system requires hourly gridded concentration input from a regional-scale CTM as well as gridded population data, exposure-response functions (ERFs) for health impacts, and economic valuations of the impacts from air pollution. A detailed description of the integrated EVA model system along with the ERFs and the economic valuations used are given in Brandt et al. (2013a).

The gridded population density data over Europe and the U.S. used in this study are presented in Fig. 1. The population data over Europe are provided on a 1km spatial resolution from Eurostat for the year 2011 (http://www.efgs.info). The U.S. population data has been provided from the U.S. Census Bureau for the year 2010. The total populations used in this study are roughly 532 and 307 million in Europe and the U.S., respectively. As the health outcomes are age-dependent, the total population data has been broken down to a set of age intervals being babies (under 9 months), children (under 15), adult (above 15), above 30, and above 65. The fractions of population in these intervals for Europe is derived from the EUROSTAT 2000 database, where the number of persons of each age at each grid cell was aggregated into the above clusters (Brandt et al., 2011), while for the U.S. they are derived from the U.S. Census Bureau for the year 2010 at 5-year intervals.

The EVA system can be used to assess the number of various health outcomes including different morbidity outcomes as well as short-term (acute) and long-term (chronic) mortality, related to exposure of O_3, CO and SO_2 (short-term) and $PM_{2.5}$ (long-term). Furthermore, impact on infant mortality in response to exposure of $PM_{2.5}$ is calculated. The health impacts are calculated using an ERF of the following form:

$$R = \alpha \times \delta_c \times P$$

where R is the response (in cases, days, or episodes), c denotes the pollutant concentration, P denotes the affected share of the population, and α an empirically determined constant for the particular health outcome. EVA uses ERFs that are modelled as a linear function, which is a reasonable approximation as showed in several studies (e.g. Pope et al., 2000; the joint World Health Organization/UNECE Task Force on Health (EU, 2004; Watkiss et al., 2005)). Many epidemiological studies have analyzed the concentration-response relationship between ambient PM and mortality using various statistical models. In general, the shapes of the estimated curves did not differ significantly from linear. However, some studies showed non-linear relationships, being steeper at lower than at higher concentrations (e.g. Samoli et al., 2005). Therefore, linear relationships may lead to overestimated health impacts over highly polluted areas. The concentration metrics used in each ERF is shown in Table 2. The sensitivity of EVA to the different pollutant concentrations are further evaluated in the supplementary material and depicted in Fig. S1. EVA calculates and uses the annual mean concentrations of CO, SO_2 and $PM_{2.5}$, while for O_3, it uses the SOMO35 metric that is defined as the yearly sum of the daily maximum of 8-hour running average over 35 ppb, following WHO (2013) and EEA (2017).

The morbidity outcomes include chronic bronchitis, restricted activity days, congestive heart failure, lung cancer, respiratory and cerebrovascular hospital admissions, asthmatic children (<15 years) and adults (>15 years), which includes bronchodilator use, cough, and lower respiratory symptoms. The exposure-response functions are broadly in line with estimates derived with detailed analysis in EU funded research (Rabl, Spadaro and Holland, 2014; EEA, 2013) To figure out the total number of premature deaths from the years of life lost due to $PM_{2.5}$, they have been converted into lost lives according to a lifetable method (explained in detail in Andersen, 2017) but using the factor of 10.6, as reported by (Watkiss et al., 2005).

To these deaths are added the acute deaths due to O_3 and SO_2. The ERFs used, along with their references, in both continents as well as the economic valuations for each health outcome in Europe and the U.S., respectively, are presented in Table 2. Baseline incidence rates are not assumed to be dissimilar, which is a coarse approach for morbidity. The baseline rates are from Statistics Denmark (http://www.statistikbanken.dk/statbank5a/default.asp?w=1280) and lifetables are based on Denmark, which is close to the US and Eurozone average (Andersen, 2017). For a description of the morbidity ERFs, see Andersen et al. (2004 and 2008). The economic valuations are provided by Brandt et al. (2013a); see also EEA (2013).

ERF for all-cause chronic mortality due to $PM_{2.5}$ were based on the findings of Pope et al. (2002), which is the most extensive study available, following conclusions from the scientific review of the Clean Air For Europe (CAFÉ) programme (Hurley et al., 2005; Krupnick et al., 2005). The results from Pope et al. (2002) are further supported by Krewski et al. (2009), and more recently by the latest HRAPIE project report (WHO, 2013a). Therefore, as recommended by WHO (2013a), EVA uses the ERFs based on the meta-analysis of 13 cohort studies as described in Hoek et al. (2013). In EVA, the number of lost life years for a Danish population cohort with normal age distribution, when applying the ERF of Pope et al. (2002) for all-cause mortality (relative risk, RR= 1.062 (1.040-1.083) on 95% confidence interval), and the latency period indicated, sums to 1138 yr of life lost (YOLL) per 100 000 individuals for an annual $PM_{2.5}$ increase of 10 μg m^{-3} (Andersen, 2008)..EVA uses a counterfactual $PM_{2.5}$ concentration of 0 μgm^{-3} following the EEA methodology, meaning that the impacts have been estimated for the full range of modelled concentrations from 0 μgm^{-3} upwards. Applying a low counterfactual concentration can underestimate health impacts at low concentrations if the relationship is linear or close to linear (Anenberg et al., 2016). However, it is important to note that uncertainty in the health impact results may increase at low concentrations due to sparse epidemiological data. Assuming linearity at very low concentrations may distort the true health impacts of air pollution in relatively clean atmospheres (Anenberg et al., 2016).

It has been shown that O_3 concentrations above the level of 35 ppb involve an acute mortality increase, presumably for weaker and elderly individuals. EVA applies the ERFs selected in CAFE for post-natal death (age group 1–12 months) and acute death related to O_3 (Hurley et al., 2005). WHO (2013a) also recommends the use of the daily maximum of 8-hour mean O_3 concentrations for the calculation of the acute mortality due to O_3. There are also studies showing that SO_2 is associated with acute mortality, and EVA adopts the ERF identified in the APHENA study – Air Pollution and Health: A European Approach (Katsouyanni et al., 1997).

Chronic exposure to $PM_{2.5}$ is also associated with morbidity, such as lung cancer. EVA employs the specific ERF (RR = 1.08 per 10 μg m^{-3} $PM_{2.5}$ increase) for lung cancer indicated in Pope et al. (2002). Bronchitis has been shown to increase with chronic exposure to $PM_{2.5}$ and we apply an ERF (RR = 1.007) for new cases of bronchitis based on the AHSMOG study (involving non-smoking Seventh-Day Adventists; Abbey et al., 1999), which is the same epidemiological study as in CAFE (Abbey, 1995; Hurley et al., 2005). The ExternE crude

incidence rate was chosen as a background rate (ExternE, 1999), which is in agreement with a Norwegian study, rather than the pan-European estimates used in CAFE (Eagan et al., 2002). Restricted activity days (RADs) comprise two types of responses to exposure: so-called minor restricted activity days as well as work-loss days (Ostro, 1987). This distinction enables accounting for the different costs associated with days of reduced well-being and actual sick days. It is assumed that 40% of RADs are work-loss days based on Ostro (1987). The background rate and incidence are derived from ExternE (1999). Hospital admissions are deducted to avoid any double counting. Hospital admissions and health effects for asthmatics (here corresponding to the three responses bronchodilator use, cough and lower respiratory symptoms) are also based on ExternE (1999).

Table 2 lists the specific valuation estimates applied in the modelling of the economic valuation of mortality and morbidity effects. A principal value of EUR 1.5 million was applied for preventing an acute death, following expert panel advice (EC 2001). For the valuation of a life year, the results from a survey relating specifically to air pollution risk reductions were applied (Alberini et al., 2006), implying a value of EUR 57.500 per year of life lost (YOLL). With the more conservative metric of estimating lost life years, rather than 'full' statistical lives, there is no adjustment for age. This is due to the fact that government agencies in Europe, including the European Commission, apply a methodology for costing of air pollution that is based on accounting for lost life years, rather than for entire statistical lives as is customary in USA. While the average traffic victim, for instance, is mid-aged and likely to lose about 35-40 years of life expectancy, pollution victims are believed to suffer significantly smaller losses of years (EAHEAP, 1999:64; Friedrich and Bickel, 2001). To avoid overstating the benefits of air pollution control, these are treated as proportional to the number of life years lost. Most of the excess mortality is due to chronic exposure to air pollution over many years and the life year metric is based on the number of lost life years in a statistical cohort. Following the guidelines of the Organisation for Economic Co-operation and Development (OECD, 2006), the predicted acute deaths, mainly from O_3, are valuated here with the adjusted value for preventing a fatality (VSL, Value of a Statistical Life). The life tables are obtained from European data and are applied to the U.S. as the average life expectancy in the U.S. is similar to that in Europe, and close to the OECD average (OECD, 2016). The willingness to pay for reductions in risk obviously differs across income levels. However, in the case of air pollution costs, adjustment according to per capita income differences among different states is not regarded as appropriate, because long-range transport implies that emissions from one state will affect numerous other states and their citizens. The valuations are thus adjusted with regional purchasing power parities (PPP) of EU27 and USA.

Cost-benefit analysis in the U.S. related to air pollution proceeds from a standard approach, where abatement measures preventing premature mortality are considered according to the number of statistical fatalities avoided, which are appreciated according to the value of VSL (presently USD 7.4 million). In contrast, and following recommendations from the UK working group on Economic Appraisal of the Health Effects of Air Pollution (EAHEAP, 1999), focus in EU has been on the possible changes in average life expectancy resulting

from air pollution. In EU, the specific number of life years lost as a result of changes in air pollution exposures are estimated based on lifetable methodology, and monetized with Value-Of-Life-Year (VOLY) unit estimates (Holland et al. 1999; Leksell and Rabl 2001). The theoretical basis is a life-time consumption model according to which the preferences for risk reduction will reflect expected utility of consumption for remaining life years (Hammitt 2007; OECD 2006:204). The much lower VSL values customary in Europe (presently €2.2 million) add decisively to the differences, as VOLY is deducted from this value. By using a common valuation framework according the EU approach we allow for direct comparisons of the monetary results. It follows from OECD recommendations (2012) to correct with PPP when doing such benefit transfer. The unit values have been indexed to 2013 prices as indicated in Table 2.

3. Results

3.1. Model Evaluation

Observed and simulated hourly surface O_3, CO, SO_2 and daily $PM_{2.5}$, which are species used in the EVA model to calculate the health impacts, over Europe and North America for the entire 2010 were compared in order to evaluate each model's performance. The statistical parameters to evaluate the models and their equations are provided in the supplementary material. For a more thorough evaluation of models and species, see Solazzo et al. (2017a). The results of this comparison are presented in Table S1 for EU and NA, along with the multi-model mean and median values. The monthly time series plots of observed and simulated health-related pollutants are also presented in Figs. 2 and 3. The monthly means are calculated using the hourly pairs of observed and modelled concentrations at each station. The results show that over Europe, the temporal variability of all gaseous pollutants is well captured by all models with correlation coefficients (*r*) higher than 0.70 in general. The normalized mean biases (*NMB*) in simulated O_3 levels are generally below 10% with few exceptions up to -35%. CO levels are underestimated by up to 45%, while the majority of the models underestimated SO_2 levels by up to 68%, while some models overestimated SO_2 by up to 49%. $PM_{2.5}$ levels are underestimated by 19% to 63%. Over Europe, the median of the ensemble performs better than the mean in terms of model bias (*NMB*) for O_3 (by 52%), while for CO, SO_2 and $PM_{2.5}$, the mean performs slightly better than the median (Table S1).

We have further evaluated the models' performance on simulating the annual mean pollutant levels over individual measurements stations and plotted the geographical distribution of the bias. Fig. 4 presents the multi model mean geographical distribution of bias from daily max 8-hour (DM8H) average O_3, CO, SO_2 and $PM_{2.5}$ over Europe, while Fig. S2-S5 show annual mean bias for O_3, CO, SO_2 and $PM_{2.5}$ for each model, respectively. DM8H O_3 levels over Europe are generally underestimated by up to 50 μgm^{-3}, with few overestimations up to 50 μgm^{-3} over southern Europe (Fig. 4a) The geographical pattern of annual mean O_3 bias is similar among the models with slight differences ($\pm$ 10 μgm^{-3}) in the bias (Fig. S2). CO levels are underestimated over all stations by up to 600 μgm^{-3} except for few stations where

CO levels are overestimated by up to 100 μgm^{-3} (Fig. 4b). All models underestimated CO levels over the majority of the stations (Fig. S3). SO_2 levels are slightly overestimated over central and southern Europe (Fig. 4c). There are also underestimation over few stations with no specific geographical pattern. Similar to CO, all models underestimated SO2 levels over the majority of the stations (Fig. S4). Finally, $PM_{2.5}$ levels are underestimated by up to 10 μgm^{-3} over most of Europe (Fig. 4d), with larger underestimations over the eastern Europe up to 30 μgm^{-3}.

Over North America, the hourly O_3 variation is well captured by all models (Table S1), with DK1 having slightly lower *r* coefficient compared to the other models and largest *NMB* (Fig. 3a). The hourly variation of CO and SO_2 levels are simulated with relatively lower *r* values (Figs. 3b, c), with SO_2 levels having the highest underestimations. The $PM_{2.5}$ levels are underestimated by ~15% except for the DE1 model, having a large underestimation of 63% (Table S1). As DE1 and US3 use the same SMOKE emissions and CTM, the large difference in $PM_{2.5}$ concentrations can be partly due to the differences in horizontal and vertical resolutions in the model setups, as can also be seen in the differences in the CO concentrations. There are also differences in the aerosol modules and components that each model simulates. For example, DE1 uses an older version of the secondary organic aerosol (SOA) module, producing ~3 μgm^{-3} less SOA, which can explain ~20% of the bias over North America. Over the North American domain, the median outscores the mean for O_3 (by 35%), CO (by 52%) and $PM_{2.5}$ (by 29%) while for SO_2, the median produces 26% higher *NMB* compared to the mean. DK1 model simulates a much higher bias for O_3 and SO_2 compared to other models in the North American domain, while DE1 has the largest bias for CO and $PM_{2.5}$.

DM8H O_3 levels are generally underestimated by the MM mean over the U.S. by up to 20 ppb, while over the eastern and central U.S. there are also overestimations by up to 10 ppb (Fig. 5a). As seen in Fig. S6, all three models have very similar performance over the U.S., with DK1 simulating a slightly lower underestimation and a higher overestimation compared to DE1 and US3. DE1 and DK1 have very similar spatial pattern in terms of CO bias, in particular over the eastern coast of the U.S. (Fig. S7). CO levels are underestimated by ~100 ppb over majority of the stations, especially over the eastern U.S., while there are much larger underestimation over the western U.S. by up to 1000 ppb (Fig. 5b). SO_2 levels are underestimated by up to 5 ppb over the majority of the stations in the U.S., with few overestimations of up to 5 ppb (Fig. 5c). DE1 and DK1 have very similar spatial distribution of bias, while US3 has slightly more overestimations (Fig. S8) Finally, PM2.5 levels are underestimated over majority of the stations by up to 6 μgm^{-3}, with few overestimations by 2-4 μgm^{-3} (Fig. 5d). DE1 has the largest underestimations compared to DK1 and US3 (Fig. S9).

Table S1 shows that the ensemble median performs slightly better than the ensemble mean for all pollutants over both continents regarding the bias and error, while the difference on *r* is rather small. Over the European stations, the median has improved results over the mean by up to 14% for *r* and up to 9% for the *RMSE*. The improvements in *r* over the U.S. are

much smaller compared to Europe (up to ~4%), while the *RMSE* is improved by up to 27%, except for SO_2 where the median has 14% higher *RMSE* than the mean.

3.2. Health outcomes and their economic valuation in Europe

The different health outcomes calculated by each model in Europe as well as their multi model mean and median are presented in Table S2. Table 3 presents the mean of the individual model estimates as MM_{mi}. Standard deviations calculated from the individual model estimates are presented along with the MM_{mi} in the text. The health impact estimates vary significantly between different models. The different estimates obtained are found to vary up to a factor of three. Among the different health outcomes, the individual models simulated the number of congestive heart failure (CHF) cases to be between 19 000 to 41 000 (mean of all individual models, MM_{mi}, 31 000 ± 6 500). The number of lung cancer cases due to air pollution are calculated to be between 30 000 to 78 000 (mean of all individual models, MM_{mi}, 55 000 ± 14 000). Finally, the total (acute + chronic) number of premature death due to air pollution is calculated to be 230 000 to 570 000 (mean of all individual models, MM_{mi}, 414 000 ± 100 000). The health impacts calculated as the median of individual models differ slightly (~±1%) from those calculated as the mean of individual models (Table S2) due to the slight differences in the model bias (*NMB*) and error (*NMGE* and *RMSE*) between the mean and the median performance statistics of the models.

In addition to averaging the health estimates from individual models (MM_{mi}), we have also produced a multi-model mean concentration data (MM_m) by taking the average of concentrations of each species calculated by all models at each grid cell and hour, and fed it to the EVA model. We have calculated the number of premature death cases in Europe as 410 000 (Table 3) using MM_m. Difference between the health impacts calculated using MM_m data from the mean of all individual model (MM_{mi}) estimates is smaller than 1%. The number of premature death cases in Europe as calculated as the average of all models in the multi model ensemble, MM_{mi}, due to exposure to O_3 is 12 000 ± 6 500, while the cases due to exposure to $PM_{2.5}$ is calculated to be 390 000 ± 100 000 [180 000 – 550 000]. The O_3-related mortality well agrees with Liang et al. (2017) that used the multi-model mean of the HTAP2 global model ensemble, which calculated an O_3-realted mortality of 12 800 [600 - 28 100]. The multi-model mean (MM_{mi}) $PM_{2.5}$-related mortality in the present study is much higher than that from the HTAP2 study (195 500 [4 400 – 454 800]). The results also agree with the most recent EEA findings (EEA, 2015), which calculated a total premature death of 419 000 die to O_3 and $PM_{2.5}$ in the EU-28 countries. There is also agreement with Geels et al. (2015) that calculated 388 000 premature death cases in Europe for the year 2000. This difference can be attributed to the number of mortality cases as calculated by the individual models, where the HTAP2 ensemble calculates a much lower minimum while the higher ends from the two ensembles well agree.

The differences between the health outcomes calculated by the HTAP2 and AQMEII ensembles arise firstly from the differences in the concentrations fields due to the differences in models, in particular spatial resolutions as well as the gas and aerosols treatments in different models, but also the differences in calculating the health impacts from these

concentrations fields. EVA calculates the acute premature death due to O_3 by using the SOMO35 metric. On the other hand, in HTAP2 O_3-related premature death is calculated by using the 6-month seasonal average of daily 1-h maximum O_3 concentrations. Both groups use the annual mean $PM_{2.5}$ to calculate the $PM_{2.5}$-related premature death. In addition to O_3 and $PM_{2.5}$, EVA also takes into account the health impacts from CO and SO_2, which is missing in the HTAP2 calculations.

Among all models, DE1 model calculated the lowest health impacts for most health outcomes, which can be attributed to the largest underestimation of $PM_{2.5}$ levels (*NMB*=-63%: Table S2) due to lower spatial resolution of the model that dilutes the pollution in the urban areas, where most of the population lives. The number of premature deaths calculated by this study is in agreement with previous studies for Europe using the EVA system (Brandt et al., 2013a; Geels et al., 2015). Recently, EEA (2015) estimated that air pollution is responsible for more than 430 000 premature deaths in Europe, which is in good agreement with the present study.

Fig. 6a. presents the geographical distribution of the number of premature death in Europe in 2010. The figure shows that the numbers of cases are strongly correlated to the population density (Fig. 1a), with the largest numbers seen in the Benelux and the Po Valley regions that are characterized as the pollution hot spots in Europe as well as in megacities such as London, Paris, Berlin and Athens.

The economic valuation of the air pollution-associated health impacts calculated by the different models along with their mean and median are presented in Table 4. A total cost of 196 to 451 billion Euros (MM mean cost of 300 ± 70 billion Euros) was estimated over Europe (EU28). Results show that 5% [1% - 11%] of the total costs is due to exposure to O_3, while 89% [80% - 96%] is due to exposure to $PM_{2.5}$. Brandt et al. (2013a) calculated a total external cost of 678 billion Euros for the year 2011 for Europe, larger than the estimates of this study, which can be explained by the differences in the simulation year and the emissions used in the models as well as the countries included in the two studies (the previous study includes e.g. Russia).

3.3. Health outcomes and their economic valuation in the U.S.

The different health outcomes calculated by each model for the U.S. as well as their mean and median are presented in Table S2. The variability among the models (~3) is similar to that in Europe. The number of congestive heart failure cases in the U.S. as calculated as the average of all models in the ensemble (MM_{mi}) is calculated to be 13 000 [7 000 – 18 000], while the lung cancer cases due to air pollution are calculated to be 22 000 [9 000 – 31 000]. Finally, the number of premature deaths due to air pollution is calculated to be 165 000 ± 75 000, where 25 000 ± 6 000 cases are calculated due to exposure to O_3 and 140 000 ± 72 000 cases due to exposure to $PM_{2.5}$. The MM_m dataset leads to a number of premature death of 149 000 that is 6% smaller than the average estimate from individual models (MM_{mi}). Due to the large reduction of *NMB* by the median compared to the mean of individual models (Table S1), the multi-model health impacts calculated as the median of health impacts from

individual models are ~13% higher than the health impacts calculated from the MM_{mi}. The O_3- and $PM_{2.5}$ mortality cases as calculated by the AQMEII and HTAP2 model ensembles reasonably agree. Liang et al. (2017) calculated an O_3-related mortality of 14 700 [900 – 30 400] and a $PM_{2.5}$-related mortality of 78 600 [4 500 – 162 600]. These results are in very good agreement with the U.S. EPA (2011) estimates of number of premature death cases of 160 000 in year 2010 and with Caizzo et al. (2013), who calculated 200 000 premature death cases from combustion sources in the U.S. Among all models, DE1 model calculated the lowest health impacts for most health outcomes, which can be attributed to the largest underestimation of $PM_{2.5}$ levels (*NMB*=-63%: Table S2).

The premature death cases in North America are mostly concentrated over the New York area, as well as in hot spots over Chicago, Detroit, Houston Los Angeles and San Francisco (Fig. 6b). The figure shows that the number of cases is following the pattern of the population density. The economic valuation of the air pollution-associated health impacts calculated by the different models in the U.S. are shown in Table 4. As seen in the table, a total cost of ~145 billion Euros is calculated. Results show that ~22% of the total costs is due to exposure to O_3 while ~78% is due to exposure to $PM_{2.5}$. The major health impacts in terms of their external costs are slightly different in North America compared to Europe.

3.4. Health impacts and their economic valuation through optimal reduced ensemble subset

The effect of pollution concentrations (EVA input) on health impacts (EVA output) is investigated in order to estimate the contribution of each air pollutant in the EVA system to health impacts over different concentration levels. The technical details are provided in the supplement.

Results show that for the particular input (gridded air pollutant concentrations from individual model)-output (each health outcome) configuration, the $PM_{2.5}$ drives the variability of the different health impact and that at least 81% of the variation of the health impacts are explained by sole variations in the pollutants (i.e. without interactions: Table S3). Table S1 also shows that the most important contribution to the health impacts is from $PM_{2.5}$, followed by CO and O_3 (with much smaller influence though). The impact of perturbing $PM_{2.5}$ by a fixed fraction of its standard deviation on the health impact is roughly double compared to CO and O_3.

We have run the EVA system over an all-models mean (MM_m) dataset and an optimal reduced ensemble dataset (MM_{opt}) calculated for each of the pollutants in the two domains in order to see how and whether an optimal reduced ensemble changes the assessment of the health impacts compared to an all- models ensemble mean. Table 5 shows some sensible error reduction, although the temporal and spatial averages mask the effective improvement in accuracy from MM_m to MM_{opt}. In Europe, the optimal reduced ensemble decreases the RMSE by up to 24%, while in NA, the error reduction is much larger (4% to up to 147%). On a seasonal basis, MM_{opt} reduces *RMSE* in $PM_{2.5}$ over Europe by 23% in winter while smaller decreases are achieved in other seasons (~10%). Regarding O_3, improvement is 16%-22%, with the largest improvement in spring. In NA, the improvement in winter *RMSE* in $PM_{2.5}$ is

smallest (~2%) while larger improvements are achieved in other seasons (~7% - ~9%). For O_3, the largest *RMSE* reduction in NA is achieved for the summer period by 14%.

The analysis of the aggregated health indices data for Europe (Table S1) shows that EVA indices rely principally on the $PM_{2.5}$ levels and then the CO and O_3 values. Therefore, the relative improvement of the indices with the optimal ensemble should be proportional to the relative improvement in $PM_{2.5}$, CO and O_3. The proportionality rate for each pollutant is given in Table S3, assuming all pollutants are varied (from MM_m to MM_{opt}) away from their mean by the same fraction of their variance. As seen in the Table 3, from MM_m to MM_{opt}, the health indices increase by up to 30% in Europe. This increase is due to a 27% increase in the domain mean $PM_{2.5}$ levels when the optimal reduced ensemble is used, as well a slight increase in O_3 by ~1%. The number of premature deaths in Europe increase from 410 000 to 524 000 (28%), resulting in a much higher estimate compared to previous mortality studies. On the contrary, in the U.S., the mean $PM_{2.5}$ and O_3 levels decrease from 2.94 $\mu g\ m^{-3}$ to 2.62 μgm^{-3} (~11%) and 18.7 ppb to 18.4 ppb (~2%), respectively. In response, the health indices decrease by ~11% (Table 3). The number of premature death cases in NA decrease from 149 000 to 133 000.

3.5. Impact of anthropogenic emissions on the health impacts and their economic valuation

The impacts of emission perturbations on the different health outcomes over Europe and the U.S. as calculated by the individual models are presented in Tables S4-S6. Table 6 shows the impacts of the different emission perturbations on the premature death cases in Europe and the U.S as calculated by a subset of models that simulated the base case and all three perturbation scenarios (MM_c). Results show that in Europe, the 20% reduction in the global anthropogenic emissions leads to ~17% domain-mean reduction in all the health outcomes, with a geographical variability as seen in Fig. 6c. The figure shows that the larger changes in mortality is calculated in the central and northern parts of Europe (15-20% decreases), while the changes are smaller in the Mediterranean region (5-10%), highlighting the non-linearity of the response to emission reductions. However, it should be noted that global models or coarse-resolution regional models (as in this study) cannot capture the urban features and pollution levels and thus, non-linearities should be addressed further using fine spatial resolutions or urban models. The models vary slightly simulating the response to the 20% reduction in global emissions, estimating decreases of ~11% to 20%. The number of premature deaths decreased on average by ~50 000, ranging from -39 000 (DK1) to -103 000 (IT1). This number is in good agreement with the ~45 000 premature death calculated by the HTAP2 global models (Liang et al., 2017). The MM_c ensemble calculated a 15% and 17% decrease in the O_3- and $PM_{2.5}$-related premature death cases, respectively, in response to the GLO scenario. This decrease in the global anthropogenic emissions leads to an estimated decrease of 56 ± 18 billion Euros in associated costs in Europe (Table 6).

As seen in Table 8, a 20% reduction of anthropogenic emissions in the EUR region, as defined in HTAP2, avoids 47 000 premature death, while a 20% reduction of the anthropogenic emissions in the NAM region leads to a much smaller decrease of premature deaths in Europe (~1 000). These improvements in the number of premature deaths are in

agreement with a recent HTAP2 global study that calculated reductions of ~34 000 and ~1 000 for the EUR and NAM scenarios, respectively (Liang et al., 2017) and with Anenberg et al. (2009 and 2014), which totals to a sum of avoided premature deaths being ~39 000 and 1 800 as calculated by the MM mean. Both the global and regional models agree that the largest impacts of reducing emissions with respect to premature deaths come from emission within the source region, while foreign sources contribute much less to improvements in avoiding adverse impacts of air pollution. The decreases in health impacts in EUR and NAM scenarios corresponds to decreases in the associated costs by -47 ± 16 billion Euros and -1.4 ± 0.4 billion Euros, respectively. This is consistent with results in Brandt et al. (2012), where a contribution of ~1% to $PM_{2.5}$ concentrations in Europe is originating from the NAM region.

The 20% reduction in global anthropogenic emissions leads to 18% reduction in the health outcomes (Table 8) in the U.S., with a geographical variability in the response. Fig. 6d shows that the largest decreases in mortality is calculated for the western coast of the U.S. (~20%) and slightly lower response in the central and eastern parts of the U.S. (15-20%). The number of premature death cases, as calculated by the mean of all individual models decreases from ~160 000 ± 70 000 to ~130 000 ± 60 000, avoiding 24 ± 10 billion Euros (Table 6) in external costs, also in agreement with the ensemble of HTAP2 global models (~23 000) The O_3-related premature death cases decreased by 42% while the $PM_{2.5}$-relared cases decreased by 18%.

A 20% reduction of the North American emissions avoids ~25 000 ± 12 000 premature deaths (-16%), suggesting that ~80% of avoided premature deaths are achieved by reductions within the source region while 20% (~5 000 premature deaths) is from foreign sources. This number is also in good agreement with Liang et al. (2017) that estimated a reduction of premature deaths of ~20 000 due to O_3 and $PM_{2.5}$ in the United States due to an emission reduction of 20% within the region itself, using the ensemble mean of the HTAP2 global models. These results are much larger than the number of avoided premature death of ~11 000 as calculated by the sum of Anenberg et al. (2009 and 2104).The corresponding benefit is calculated to be 21 ± 9 billion Euros in the NAM scenario. According to results from the EAS scenario, among these 5 000 avoided cases that are attributed to the foreign emission sources, 1 900 ± 2 000 premature deaths can be avoided by a 20% reduction of the East Asian emissions, avoiding 2.5 ± 3 billion Euros. Our number of avoided premature deaths due to the EAS scenario is much higher than 580 avoided premature deaths calculated by Liang et al. (2017) and 380 avoided cases as calculated by Anenberg et al. (2009 and 2014).

Conclusions

The impact of air pollution on human health and their economic valuation for the society across Europe and the United States is modelled by a multi-model ensemble of regional models from the AQMEII3 project. All regional models used boundary conditions from the C-IFS model, and emissions from either the MACC inventory in Europe or the EPA inventory for the North America, or the global inventory from HTAP. Sensitivity analysis on the dependence of models on different sets of boundary conditions has not been conducted so

far but large deviations from the current results in terms of health impacts are not expected. The modelled surface concentrations by each individual model are used as input to the EVA system to calculate the resulting health impacts and the associated external costs from O_3, CO, SO_2 and $PM_{2.5}$. Along with a base case simulation for the year 2010, some groups performed additional simulations, introducing 20% emission reductions both globally and regionally in Europe, North America and East Asia.

The base case simulation of each model is evaluated with available surface observations in Europe and North America. Results show large variability among models, especially for $PM_{2.5}$, where models underestimate by ~20% - ~60%, introducing a large uncertainty in the health impact estimates as $PM_{2.5}$ is the main driver for health impacts. The differences in the models are largely due to differences in the spatial and vertical resolutions, meteorological inputs, inclusion of natural emissions, dust in particular, as well as missing or underestimated SOA mass, which is critical for the $PM_{2.5}$ mass. As shown in the supplementary material, the CTMs diverge a lot on the representation of particles and their size distribution, SOA formation, as well as the inclusion of natural sources. As the anthropogenic emissions are harmonized in the models, they represent a minor uncertainty in terms of model-to-model variation. However, differences in the treatment of the temporal, vertical and chemical distributions of the particulate and volatile organic species have an influence in the model calculations and therefore lead to model-to-model variations.

The variability of health impacts among the models can be up to a factor of three in Europe (twelve models) and the U.S. (three models), among the different health impacts. The multi-model mean total number of premature death is calculated to be 414 000 in Europe and 160 000 in the U.S., where $PM_{2.5}$ contributes by more than 90%. These numbers agree well with previous global and regional studies for premature deaths due to air pollution. In order to reduce the uncertainty coming from each model, an optimal ensemble set is produced, that is, the subset of models that produce the smallest error compared to the surface observations at each time step. The optimum ensemble results in an increase of health impacts by up to 30% in Europe and a decrease by ~11% in the United States. These differences clearly demonstrate the importance of the use of optimal-reduced multi-model ensembles over traditional all model-mean ensembles, both in terms of scientific results, but also in policy applications.

Finally, the role of domestic versus foreign emission sources on the related health impacts is investigated using the emission perturbation scenarios. A global reduction of anthropogenic emissions by 20% decreases the health impacts by 17%, while the reduction of foreign emissions decreases the health impacts by less than 1%. The decrease of emissions within the source region decreases the health impacts by 16%. These results show that the largest impacts of reducing emissions with respect to the premature death come from emissions within the source region, while foreign sources contributing to much less improvements in avoiding adverse impacts of air pollution.

Outlook

Currently health assessments of airborne particles are carried out under the assumption that all fine fraction particles affect health to a similar degree independent of origin, age and chemical composition of the particles. A 2013 report from WHO concludes that the cardiovascular effects of ambient $PM_{2.5}$ are greatly influenced, if not dominated, by their transition metal contents (WHO, 2013b). It is known that trace metals and traffic markers are highly associated with daily mortality (Lippmann, 2014). Even low concentrations of trace metals can be influential on health related responses.

Regarding ambient concentrations of PM and the exposure-response functions (ERFs), there is a rich set of studies providing information on total PM mass. However, only few studies focus on individual particulate species, mainly black carbon and carbonaceous particles. In addition to PM, studies on human populations have not been able to isolate potential effects of NO_2, because of its complex link to PM and O_3. The WHO REVIHAAP review from 2013 concludes that health assessments based on $PM_{2.5}$ ERFs will be most inclusive (WHO, 2013b). In addition, the ERFs are based on urban background measurements, introducing uncertainties regarding non-urban areas or high pollution areas as e.g. street canyons. Current state-of-the-art health impact estimates, in particular on regional to global scales, assume a correlation with exposure to outdoor air pollution, while in reality, exposure is dynamic and depends on the behavior of the individual. In addition, differences in age groups, gender, ethnicity and behavior should be considered in the future studies. There are also uncertainties originating from the representations of the aerosols in the atmospheric models used in the calculation of pollutant concentrations as well as the emissions. Further developments in the aerosol modules, such as the representation of organic aerosols and windblown and suspended dust, are need in order to achieve mass closure of PM to get robust estimates of health impacts. In addition, new findings show that O_3 has also chronic health impacts in addition to its acute impacts (WHO, 2013a; Turner et al., 2016).

Due to above reasons, there is a large knowledge gap regarding the health impacts of particles. There are a number of ongoing projects trying to identify the health impacts from individual particle components and produce individual ERFs for these components. NordicWelfAir project (http://projects.au.dk/nordicwelfair/) aims to investigate the potential causal impact of individual chemical air pollutants as well as mixtures of air pollutants on health outcomes. In pursuing this aim, the project uses the unique Nordic population-based registers allowing linkage between historical residential address, air pollutants over decades and later health outcomes. By linking the exposure to health outcomes, new exposure-response relationships can be determined of health effects for different population groups (e.g. age, education, ethnicity, gender, lifestyle, and working life vs. retirement conditions) related to air pollution for the individual chemical air pollutants. In addition, the high resolution simulations conducted will enable us to have a better understanding of non-linearities between the emissions, health impacts, and their economic valuation.

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Table 1.Key features (meteorological/chemistry and transport models, emissions, horizontal and vertical grids) of the regional models participating to the AQMEII3 health impact study and the perturbation scenarios they performed.

Group Code	Model	Emissions	Horizontal Resolution	Vertical Resolution	Gas Phase	Aerosol Model	Europe				North America			
							BASE	GLO	NAM	EUR	BASE	GLO	EAS	NAM
DE1	COSMO-CLM/CMAQ	HTAP	24 km × 24 km	30 layers, 50 hPa	CB5-TUCL	3 modes	×	×	×	×	×	×	×	×
DK1	WRF/DEHM	HTAP	50 km × 50 km	29 layers, 100 hPa	Brandt et al. (2012)	2 modes	×	×	×	×	×	×	×	×
ES1	WRF/CHEM	MACC	23 km × 23 km	33 layers, 50 hPa	RADM2	3 modes, MADE/SORGAM	×		×					
FI1	ECMWF/SILAM	MACC	0.25° × 0.25°	12 layers, 13 km	CB4	1-5 bins, VBS	×	×	×	×				
FRES1	ECMWF/CHIMERE	HTAP	0.25° × 0.25°	9 layers, 50 hPa	MELCHIOR2	8 bins	×	×	×	×				
IT1	WRF/CHEM	MACC	23 km × 23 km	33 layers, 50 hPa	RACM-ESRL	3 modes, MADE/VBS	×	×		×				
IT2	WRF/CAMx	MACC	23 km × 23 km	14 layers, 8 km	CB5	3 modes	×	×						
NL1	LOTOS/EUROS	MACC	0.50° × 0.25°	4 layers, 3.5 km	CB4	2 modes, VBS	×							
TR1	WRF/CMAQ	MACC	30 km × 30 km	24 layers, 10hPa	CB5	3 modes	×	×	×					
UK1	WRF/CMAQ	MACC	15 km × 15 km	23 layers, 100 hPa	CB5-TUCL	3 modes	×	×	×	×				
UK2	WRF/CMAQ	HTAP	30 km × 30 km	23 layers, 100 hPa	CB5-TUCL	3 modes	×	×						
UK3	WRF/CMAQ	MACC	18 km × 18 km	35 layers, 16 km	CB5	3 modes	×	×	×					
US3	WRF/CMAQ	SMOKE	12 km × 12 km	35 layers, 50 hPa	CB5-TUCL	3 modes					×	×	×	×

Table 2. Exposure-response functions, the concentrations metrics, and economic valuations used in the EVA model.

Health effects (compounds)	**Exposure-response coefficient**	**Valuation, €$_{2013}$**
	(α)	**(EU27 & NA)**
Morbidity		
Chronic Bronchitis[1], CB (PM)	8.2E-5 cases/µgm^{-3} (adults)	38,578 per case
Restricted activity days[2], RAD (PM)	=8.4E-4 days/ µgm^{-3} (adults)	98 per day
	-3.46E-5 days/ µgm^{-3} (adults)	
	-2.47E-4 days/ µgm^{-3} (adults>65)	
	-8.42E-5 days/ µgm^{-3} (adults)	
Congestive heart failure[3], CHF (PM)	3.09E-5 cases/ µgm^{-3}	10,998 per case
Congestive heart failure[3], CHF (CO)	5.64E-7 cases/ µgm^{-3}	
Lung cancer[4], LC (PM)	1.26E-5 cases/ µgm^{-3}	16,022 per case
Hospital admissions		
Respiratory[5], RHA (PM)	3.46E-6 cases/ µgm^{-3}	5,315 per case
Respiratory[5], RHA (SO_2)	2.04E-6 cases/ µgm^{-3}	
Cerebrovascular[6], CHA (PM)	8.42E-6 cases/ µgm^{-3}	6,734 per case
Asthma children (7.6 % < 16 years)		
Bronchodilator use[7], BUC (PM)	1.29E-1 cases/ µgm^{-3}	16 per case
Cough[8] – COUC (PM)	4.46E-1 days/ µgm^{-3}	30 per day
Lower respiratory symptoms[7], LRSA (PM)	1.72E-1 days/ µgm^{-3}	9 per day
Asthma adults (5.9 % > 15 years)		
Bronchodilator use[9], BUA (PM)	2.72E-1 cases/ µgm^{-3}	16 per case
Cough[9], COUA (PM)	2.8E-1 days/ µgm^{-3}	30 per day
Lower respiratory symptoms[9], LRSA (PM)	1.01E-1 days/ µgm^{-3}	9 per day
Mortality		
Acute mortality[10,11] (SO_2)	7.85E-6 cases/ µgm^{-3}	1,532,099 per case
Acute mortality[10,11] (O_3)	3.27E-6*SOMO35 cases/ µgm^{-3}	
Chronic mortality[4,12,], YOLL (PM)	1.138E-3 YOLL/ µgm^{-3} (>30 years)	57,510 per YOLL
Infant mortality[13], IM (PM)	6.68E-6 cases/ µgm^{-3} (> 9 months)	2,298,148 per case

[1] Abbey et al. (1995), [2] Ostro (1987), [3] Schwartz and Morris (1995), [4] Pope et al. (2002), [5] Dab et al. (1996), [6] Wordley et al. (1997), [7] Roemer et al. (1993), [8] Pope and Dockerey (1992), [9] Dusseldorp et al. (1995), [10] Anderson (1996), [11] Touloumi (1996), [12] Pope et al. (1995), [13] Woodruff et al. (1997).

Table 3. Health impacts calculated by the mean of individual model estimates (denoted as MM_{mi}) and the standard deviation, multi-model mean ensemble without error reduction (MM_m) and the optimal ensemble (MM_{Opt}) in Europe and the U.S. See Table 2 for the definitions of health impacts. PD stands for premature death. All health impacts are in units of number of cases × 1000, except for Infant Mortality (IM), which reports directly the number of cases.

	EU			NA		
	MM_{mi}	MM_m	MM_{Opt}	MM_{mi}	MM_m	MM_{Opt}
CB	360±89	360	468	142±74	142	125
RAD	368 266±90 670	368245	478073	145 337±75 250	145337	127921
RHA	23±5	23	28	10±4	8	7
CHA	46±11	46	60	19±10	19	16
CHF	31±6	31	38	13±6	9	8
LC	55±14	55	72	22±11	22	19
BDUC	10 766±2 650	10766	13976	4 566±2 383	4566	4019
BDUA	70 492±17 400	70489	91511	27 819±14 400	27819	24485
COUC	37 198±9 160	37196	48289	15 776±8 230	15776	13886
COUA	72 566±17 900	72562	94203	28 637±14 830	28637	25206
LRSC	14 355±3 530	14354	18635	6 088±3 180	6088	5359
LRSA	26 175±6 400	26174	33980	10 330±5 350	10330	9092
AYOLL	26±13	23	20	25±7	9	9
YOLL	4 111±1 010	4111	5337	1 481±762	1481	1304
PD	414±98	410	524	165±76	149	133
IM*	403±99	403	524	143±75	143.3667	126.1

Table 4. External costs (in million Euros) related to the health impacts of air pollution as calculated by the individual models over Europe and the United States.

Models	CO	SO_2	O_3	$PM_{2.5}$	TOTAL
Europe					
DE1	70	19 000	22 000	155 000	196 000
DK1	80	13 000	24 000	237 000	274 000
ES1	70	8 000	6 000	339 000	353 000
FI1	90	18 000	5 000	335 000	358 000
FRES1	90	15 000	13 000	305 000	333 000
IT1	80	17 000	21 000	413 000	451 000
IT2	70	11 000	6 000	253 000	270 000
NL1	70	12 000	18 000	215 000	245 000
TR1	110	30 000	35 000	376 000	441 000
UK1	80	28 000	25 000	280 000	333 000
UK2	80	34 000	27 000	340 000	401 000
UK3	80	47 000	25 000	279 000	351 000
MEAN	81	21 000	19 000	294 000	334 000
MEDIAN	80	17 500	21 500	292 500	342 000
The United States					
DE1	30	9 000	21 000	46 000	76 000
DK1	55	11 000	39 000	123 000	172 000
US3	60	14 000	22 000	155 000	191 000
MEAN	50	11 500	27 000	108 000	146 000
MEDIAN	55	11 000	22 000	123 000	172 000

Table 5. Annual average RMSE of the multi-model ensemble mean (MM_m) and of the optimal reduced ensemble mean (MM_{opt}) for the heath impact-related species. Units are ppb for the gaseous species and μg m^{-3} for $PM_{2.5}$.

	O_3		CO		SO_2		$PM_{2.5}$	
	MM_m	MM_{opt}	MM_m	MM_{opt}	MM_m	MM_{opt}	MM_m	MM_{opt}
Europe								
Winter	10.3	8.6	502.4	490.3	6.3	5.6	22.5	20.7
Spring	12.4	9.6	247.1	239.5	4.6	3.1	9.9	7.8
Summer	13.4	10.7	197.4	188.0	3.9	2.3	8.2	5.7
Autumn	10.7	8.8	314.5	305.5	4.6	3.1	11.0	8.7
Annual	11.7	9.4	315.3	305.8	4.8	3.5	12.9	10.7
North America								
Winter	10.9	10.4	356.7	328.1	5.7	5.5	8.3	8.1
Spring	12.0	11.4	288.7	270.2	5.4	5.1	7.2	6.6
Summer	15.1	13.0	258.3	238.7	5.4	5.0	9.7	8.8
Autumn	12.8	11.6	330.6	307.6	5.8	5.3	7.8	7.2
Annual	12.7	11.6	308.6	286.1	5.6	5.2	8.2	7.7

Table 6. Impact of the emission reduction scenarios on avoided premature death (ΔPD) and corresponding change in external cost as calculated by the multi-model mean over Europe and the United States.

Source	Receptor			
	Europe		The United States	
	ΔPD	ΔTotal Cost (billion €)	ΔPD	ΔTotal Cost (billion €)
GLO	-54 000 ± 18 000	-56 ± 18	-27 500 ± 14 000	-24 ± 10
NAM	-940 ± 1100	-1.4 ± 0.4	-25 000 ± 12 000	-21 ± 9
EUR	-47 000 ± 24 000	-47 ± 16	-	-
EAS	-	-	-1 900 ± 2 200	-2.5 ± 3

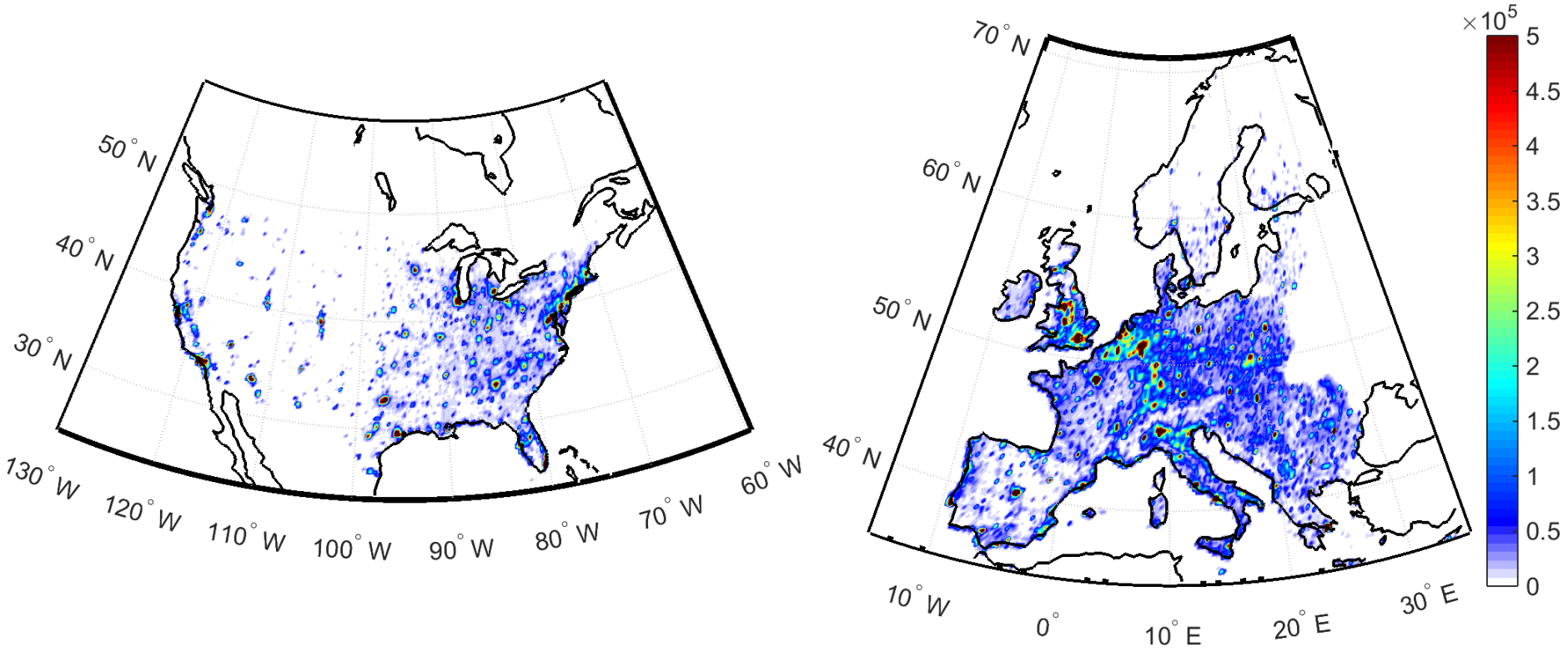

Fig.1. Population density (population per 0.25°×0.25° grid box) over a) the United States and b) Europe.

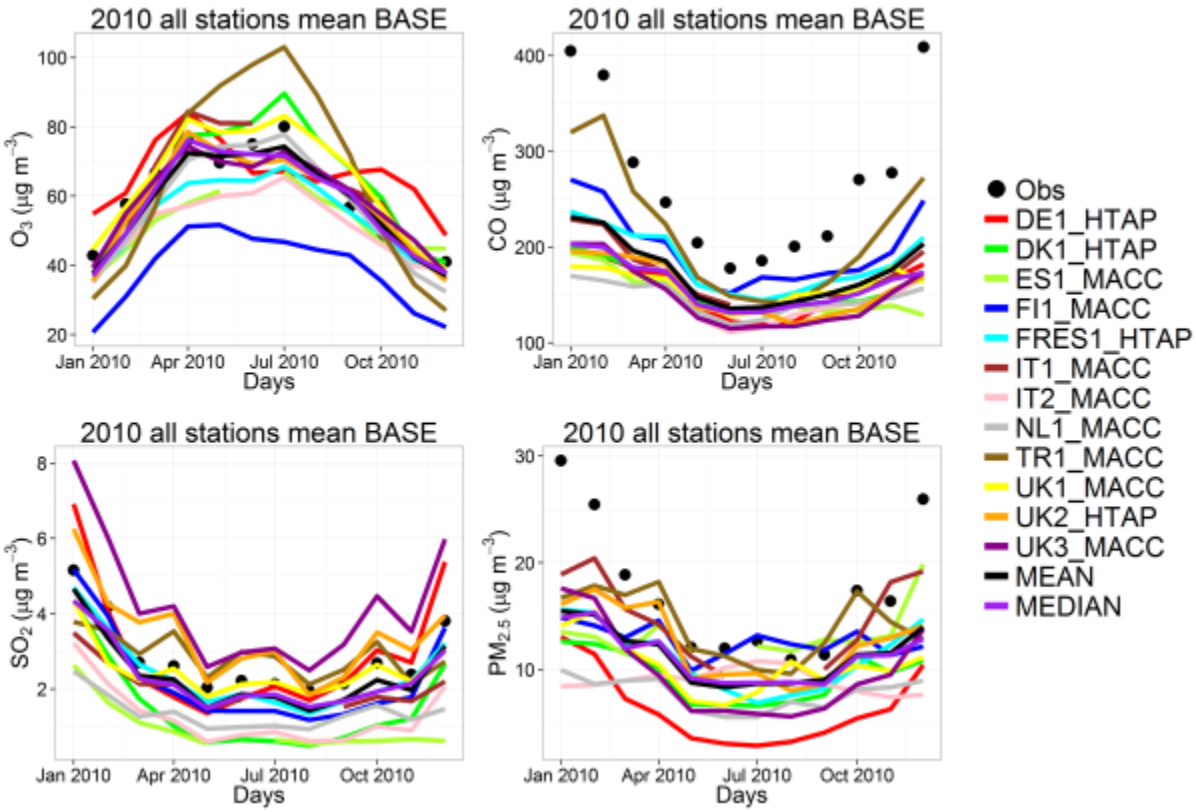

Fig. 2. Observed and simulated (base case) monthly a) O_3, b) CO, c) SO_2 and d) $PM_{2.5}$ concentrations over Europe.

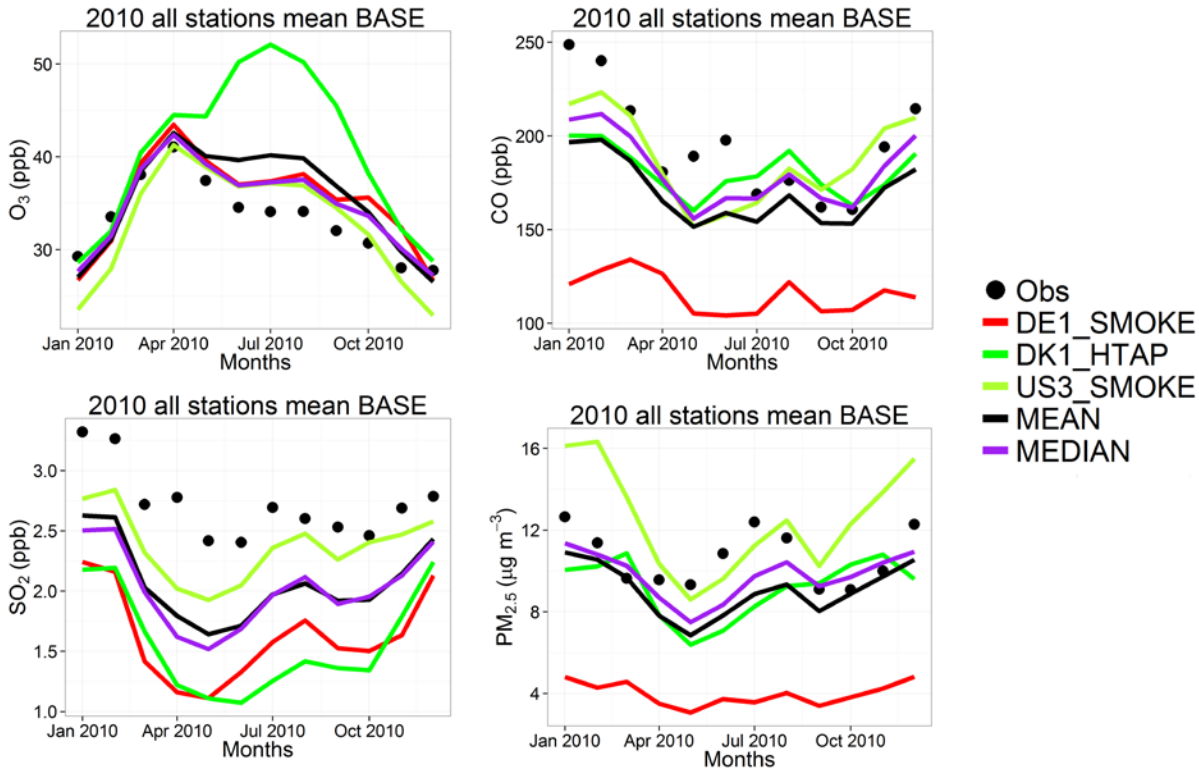

Fig. 3. Observed and simulated (base case) monthly a) O_3, b) CO, c) SO_2 and d) $PM_{2.5}$ concentrations over the U.S.

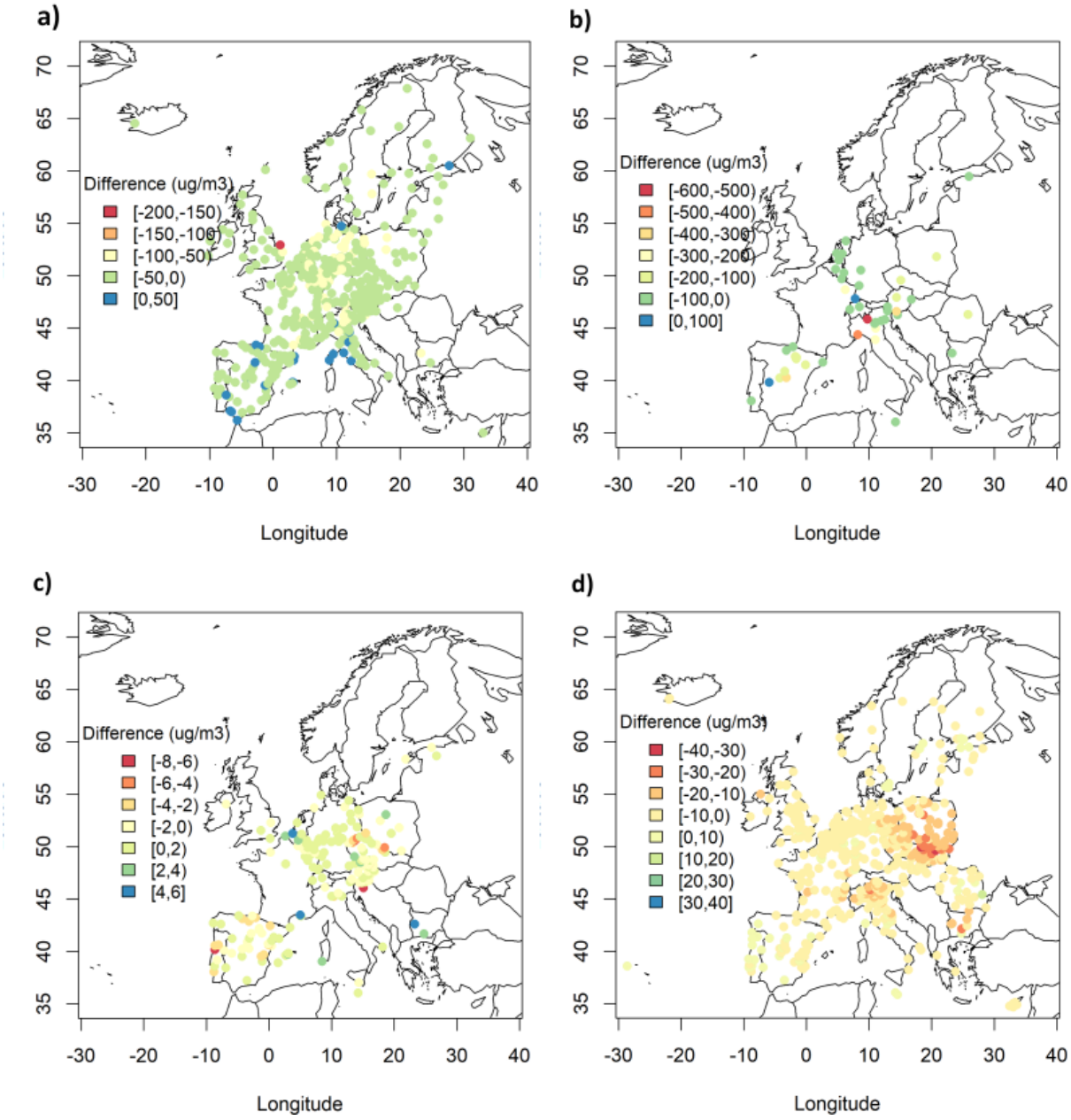

Fig. 4. Spatial distribution of annual MM mean bias (μgm^{-3}) for a) DM8H O_3, b) CO, c) SO_2 and d) $PM_{2.5}$ over Europe.

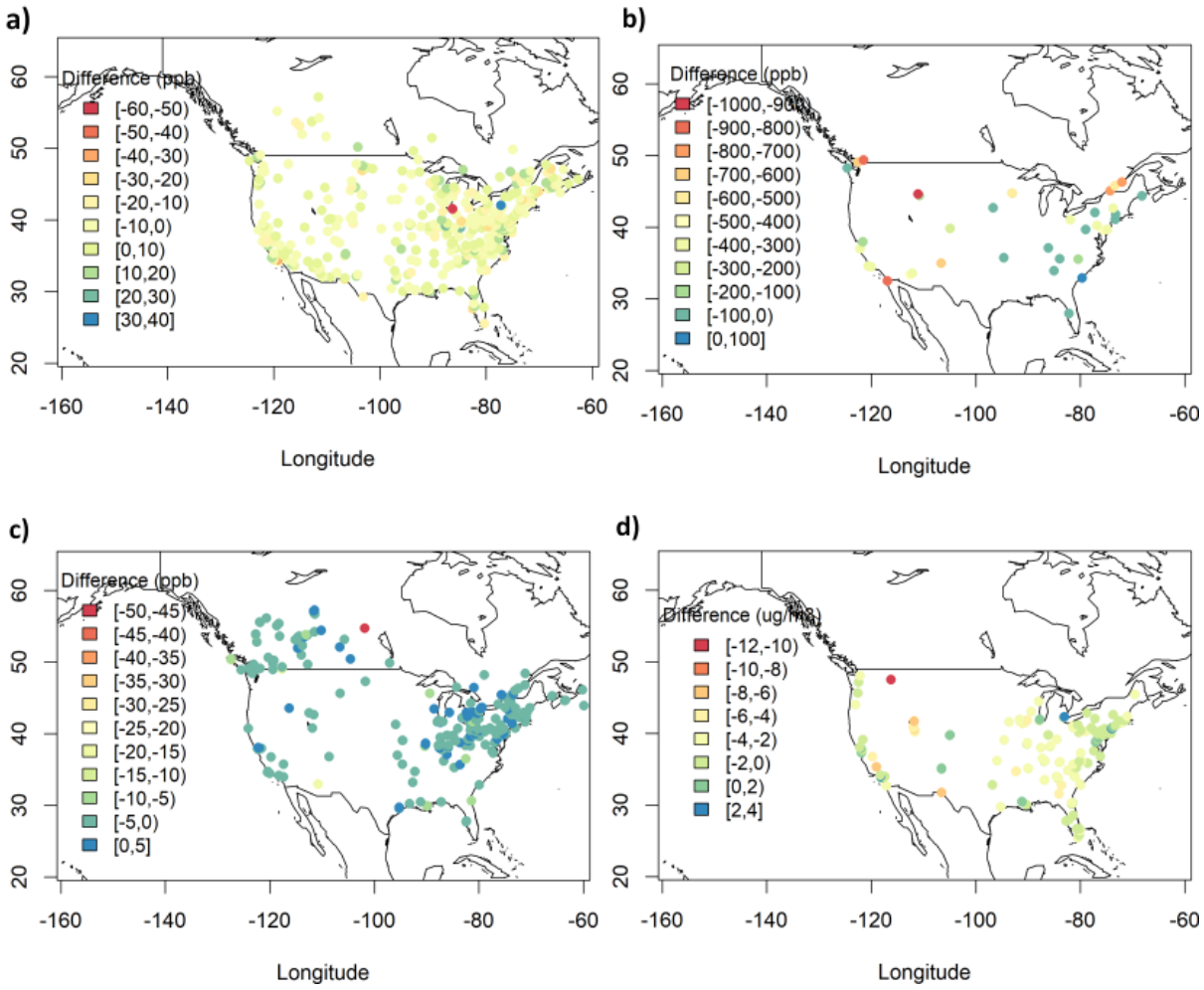

Fig. 5. Spatial distribution of annual MM mean bias (ppb for gases and μgm^{-3} for $PM_{2.5}$) for a) DM8H O_3, b) CO, c) SO_2 and d) $PM_{2.5}$ over North America.

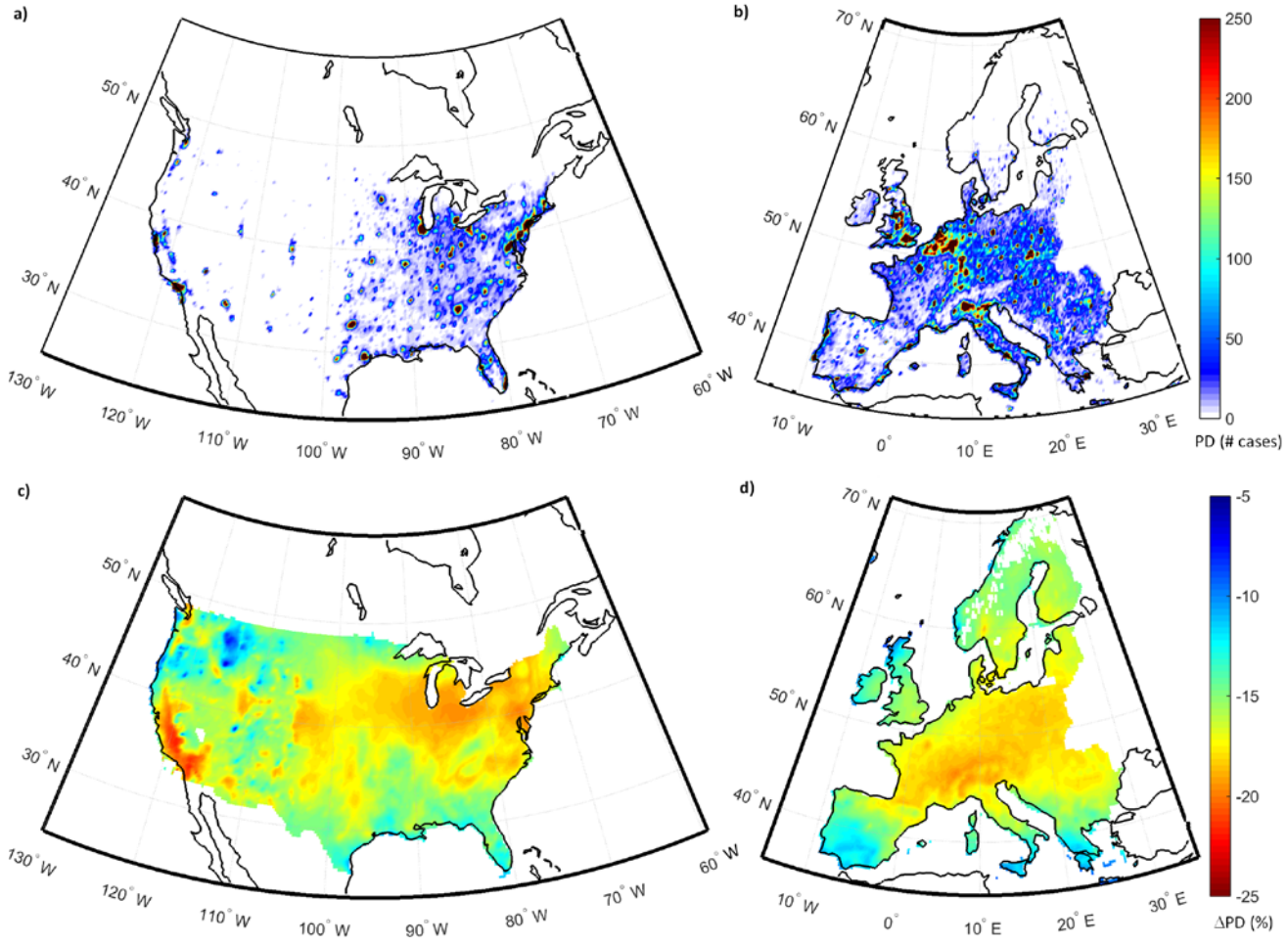

Fig. 6. Spatial distribution of the number of total premature death (PD: units in number of cases per 0.25°×0.25° grid box) in a) the United States and b) Europe and the relative change (%) in the number of premature death (PD) in response to the GLO scenario in c) the United States and d) Europe in 2010 as calculated by the multi-model mean ensemble.