



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

43 The impact of air pollution on human health and the associated external costs in Europe and
44 the United States (U.S.) for the year 2010 is modelled by a multi-model ensemble of regional
45 models in the frame of the third phase of the Air Quality Modelling Evaluation International
46 Initiative (AQMEII3). This is the first study known to use a common health assessment
47 approach across the two continents. The modelled surface concentrations of O₃, CO, SO₂ and



48 PM_{2.5} from each model are used as input to the Economic Valuation of Air Pollution (EVA)
49 system to calculate the resulting health impacts and the associated external costs. Along with
50 a base case simulation, additional runs were performed introducing 20% emission reductions
51 both globally and regionally in Europe, North America and East Asia.

52 Health impacts estimated by different models can vary up to a factor of three in Europe
53 (twelve models) and the United States (three models). In Europe, the multi-model mean
54 number of premature deaths is calculated to be 414 000 while in the U.S., it is estimated to be
55 160 000, in agreement with previous global and regional studies. In order to estimate the
56 impact of biases coming from each model, two multi-model ensembles were produced, the
57 first attributing an equal weight to each member of the ensemble, and the second where the
58 subset of models that produce the smallest error compared to the surface observations at each
59 time step. The latter results in increase of health impacts by up to 30% in Europe, thus giving
60 significantly higher mortality estimates compared to available literature. This is mostly due to
61 a 27% increase in the domain mean PM_{2.5} levels, along with a slight increase in O₃ by ~1%.
62 Over the U.S., the mean PM_{2.5} and O₃ levels decrease by 11% and 2%, respectively, when the
63 optimal ensemble mean is used, leading to a decrease in the calculated health impacts by
64 ~11%. These differences encourage the use of optimal-reduced multi-model ensembles over
65 traditional all model-mean ensembles, in particular for policy applications.

66 Finally, the role of domestic versus foreign emission sources on the related health impacts is
67 investigated using the 20% emission reduction scenarios applied over the source regions as
68 defined in the frame of HTAP2. The differences are calculated based on the models that are
69 common in the basic multi-model ensemble and the perturbation scenarios, resulting in five
70 models in Europe and all three models in the U.S. A 20% reduction of global anthropogenic
71 emissions avoids 54 000 and 27 500 premature deaths in Europe and the U.S., respectively. A
72 20% reduction of North American emissions foreign emissions avoids ~1 000 premature
73 deaths in Europe and 25 000 premature deaths in the U.S. A 20% decrease of emissions
74 within the European source region avoids 47 000 premature deaths in Europe. Reducing the
75 East Asian emission by 20% avoids ~2000 premature deaths in the U.S. These results show
76 that the domestic emissions make the largest impacts on premature death, while foreign
77 sources make a minor contributing to adverse impacts of air pollution.

78 **1. Introduction**

79 According to the World Health Organization (WHO), air pollution is now the world's largest
80 single environmental health risk (WHO, 2014). Around 7 million people died prematurely in
81 2012 as a result of air pollution exposure from both outdoor and indoor emission sources
82 (WHO, 2014). WHO estimates 3.7 million premature deaths in 2012 from exposure to
83 outdoor air pollution from urban and rural sources worldwide. According to the Global
84 Burden of Disease (GBD) study, exposure to ambient particulate matter pollution remains
85 among the ten leading risk factors. Air pollution is a transboundary and scale dependent
86 challenge with global, regional, national and local sources, leading to large differences in the
87 geographical distribution of human exposure. Short-term exposure to ozone (O₃) is associated
88 with respiratory morbidity and mortality (e.g. Bell et al., 2004), while long-term exposure to



89 O₃ has been associated with premature respiratory mortality (Jerrett et al., 2009). Short-term
90 exposure to particulate matter (PM_{2.5}) has been associated with increases in daily mortality
91 rates from respiratory and cardiovascular causes (e.g. Pope and Dockery, 2006), while long-
92 term exposure to PM_{2.5} can have detrimental chronic health effects, including premature
93 mortality due to cardiopulmonary diseases and lung cancer (Burnett et al., 2014). The Global
94 Burden of Disease Study 2013 (GBD 2013) estimated 210 000 premature deaths per year
95 associated with ambient O₃ and 2.9 million associated with anthropogenic PM_{2.5} (Forouzanfar
96 et al., 2015).

97 Changes in emissions from one region can impact air quality over others, affecting also air
98 pollution-related health impacts due to intercontinental transport (Anenberg et al., 2014;
99 Zhang et al., 2017). In the framework of the Task Force on Hemispheric Transport of Air
100 Pollution (TF-HTAP), Anenberg et al. (2009) found that reduction of foreign ozone precursor
101 emissions can contribute to more than 50% of the deaths avoided by simultaneously reducing
102 both domestic and foreign precursor emissions. Similarly, they found that reducing emissions
103 in North America (NA) and Europe (EU) reduced the ozone-related premature deaths more
104 outside the source region than within (Anenberg et al., 2009). This result agrees with Duncan
105 et al. (2008), which showed for the first time that emission reductions in NA and EU have
106 greater impacts on ozone mortality outside the source region than within. Anenberg et al.
107 (2014) estimates that 93–97 % of PM_{2.5}-related avoided deaths from reducing emissions
108 occurs within the source region while 3–7 % occur outside the source region from
109 concentrations transported between continents. In spite of the shorter lifetime of PM_{2.5}
110 compared to O₃, it was found to cause more deaths from intercontinental transport (Anenberg
111 et al., 2009; 2014). In the frame of the second phase of the Task Force on Hemispheric
112 Transport of Air Pollution (TF-HTAP2; Galmarini et al., 2017), an ensemble of global
113 chemical transport model simulations calculated that 20% emission reductions from one
114 region generally lead to more avoided deaths within the source region than outside (Liang et
115 al., 2017). They calculated that source emission reductions avoided ozone- and PM_{2.5}-related
116 premature deaths within the source region by 84% and 89%, respectively, indicating that
117 most avoided mortality occurred within the source region in general.

118 Recently, Lelieveld et al. (2015) used a global chemistry model to calculate that outdoor air
119 pollution led to 3.3 million premature deaths globally in 2010, while in Europe and North
120 America, 381 000 and 68 000 premature deaths occurred, respectively. They have also
121 calculated that these numbers are likely to roughly double in the year 2050 assuming a
122 business-as-usual scenario. Silva et al. (2016), using the ACCMIP model ensemble,
123 calculated that the global mortality burden of ozone is estimated to markedly increase from
124 382 000 deaths in 2000 to between 1.09 and 2.36 million in 2100. They also calculated that
125 the global mortality burden of PM_{2.5} is estimated to decrease from 1.70 million deaths in
126 2000 to between 0.95 and 1.55 million deaths in 2100. Silva et al. (2013) estimated that in
127 2000, 470 000 premature respiratory deaths are associated globally and annually with
128 anthropogenic ozone, and 2.1 million deaths with anthropogenic PM_{2.5}-related
129 cardiopulmonary diseases (93%) and lung cancer (7%).



130 In Europe, recent results show that outdoor air pollution due to O₃, CO, SO₂ and PM_{2.5} causes
131 a total number of 570 000 premature deaths in the year 2011 (Brandt et al., 2013a; 2013b).
132 The external (or indirect) costs to society related to health impacts from air pollution are
133 tremendous. OECD (2014) estimates that outdoor air pollution is costing its member
134 countries USD 1.57 trillion in 2010. Among the OECD member countries, the economic
135 valuation of air pollution in the U.S. was calculated to be ~500 billion USD and ~660 USD in
136 Europe. In the whole of Europe, the total external costs have been estimated to approx. 800
137 billion Euros in year 2011 (Brandt et al., 2013a). These societal costs have great influence on
138 the general level of welfare and especially on the distribution of welfare both within the
139 countries as air pollution levels are vastly heterogeneous both at regional and local scales and
140 between the countries as air pollution and the related health impacts are subject to long-range
141 transport. Geels et al. (2015), using two regional chemistry and transport models, estimated a
142 premature mortality of 455 000 and 320 000 in Europe (EU28 countries) for the year 2000,
143 respectively, due to O₃, CO, SO₂ and PM_{2.5}. They also estimated that climate change alone
144 leads to a small increase (15%) in the total number of O₃-related acute premature deaths in
145 Europe towards the 2080s and relatively small changes (<5%) for PM_{2.5}-related mortality.
146 They found that the combined effect of climate change and emission reductions will reduce
147 the premature mortality due to air pollution, in agreement with the results from Schucht et al.
148 (2015).

149 The U.S. Environmental Protection Agency estimated that in 2010 there were ~160 000
150 premature deaths in the U.S. due to air pollution (U.S. EPA, 2011). Fann et al. (2012)
151 calculated 130,000 and 350,000 O₃- and PM_{2.5} - related premature deaths in the U.S.,
152 respectively, for the year 2005. Caizzo et al. (2013) estimated 200 000 cases of premature
153 death in the U.S. due to air pollution from combustion sources for the year 2005.

154 The health impacts of air pollution and associated external costs are estimated based on
155 observed and/or modelled air pollutant levels. Observations have spatial limitations
156 particularly when fine scale assessments are needed. The impacts of air pollution on health
157 can be estimated using models, where the level of complexity can vary depending on the
158 geographical scale (global, continental, country or city), concentration input (observations,
159 model calculations, emissions) and the pollutants of interest that can vary from only few
160 (PM_{2.5} or O₃) to a whole set of all regulated pollutants. The health impact models normally
161 used may differ in the geographical coverage, spatial resolutions of the air pollution model
162 applied, complexity of described processes, the exposure-response functions (ERFs),
163 population distributions and the baseline indices (see Anenberg et al., 2015 for a review).

164 Air pollution related health impacts and associated costs can be calculated using Chemical
165 Transport Model (CTM) or with standardized source-receptor relationships characterizing the
166 dependence of ambient concentrations on emissions. (e.g. EcoSense model: ExternE, 2005,
167 TM5-FASST: Van Dingenen et al., 2014). Source-receptor relationships have the advantage
168 of reducing the computing time significantly and have therefore seen extensive application in
169 systems like GAINS (Amann et al., 2011). On the other hand, full CTM simulations have the
170 advantage of better accounting for non-linear chemistry-transport processes in the
171 atmosphere.



172 CTMs are useful tools to calculate the concentrations of health-related pollutants taking into
173 account non-linearities in the chemistry and the complex interactions between meteorology
174 and chemistry. However, the CTMs include different chemical and aerosol schemes that
175 introduce differences in the representation of the atmosphere as well as differences in the
176 emissions and boundary conditions they use (Im et al., 2015a,b). These different approaches
177 are present also in the health impact estimates that use CTM results as basis for their
178 calculations. Multi-model (MM) ensembles can be useful to the extent that allow us to take
179 into consideration several model results at the same time, define the relative weight of the
180 various members in determining the mean behavior, and produce also an uncertainty
181 estimated based on the diversity of the results (Potemski and Galmarini, 2010; Riccio et al.,
182 2013; Solazzo et al., 2013).

183 The third phase of the Air Quality Modelling Evaluation International Initiative (AQMEI3)
184 project brought together fourteen European and North American modelling groups to
185 simulate the air pollution levels over the two continental areas for the year 2010 (Galmarini et
186 al., 2017). Within AQMEI3, the simulated surface concentrations of health related air
187 pollutants from each modelling group serves as input to the Economic Valuation of Air
188 Pollution (EVA) model (Brandt et al., 2013a; 2013b). This is the first study in our knowledge
189 that uses a common approach across the two continents regarding health impact assessment
190 of air pollution and in particular, their economic valuation (Andersen, 2017). The EVA model
191 is used to calculate the impacts of health-related pollutants on human health over the two
192 continents as well as the associated external costs. We adopt a multi-model ensemble (MM)
193 approach, in which the outputs of the modelling systems are statistically combined assuming
194 equal contribution from each model and used as input for the EVA model. In addition, the
195 human health impacts (and the associated costs) of reducing anthropogenic emissions,
196 globally and regionally have been calculated, allowing to quantify the trans-boundary
197 benefits of emission reduction strategies. Finally, starting from the conclusions of Solazzo
198 and Galmarini (2015), the health impacts have been calculated also using a so-called optimal
199 ensemble of models, determined by error minimization and redundancy analyses in the
200 framework of the AQMEI3 project. This provided us with an assessment that guaranteed for
201 the absence of recursive or redundant results due to the lack of model independence that we
202 can compare with the classically derived estimates based on model averaging.

203 **2. Material and Methods**

204 **2.1. AQMEI**

205 In the framework of the AQMEI3 project, fourteen groups participated to simulate the air
206 pollution levels in Europe and North America for the year 2010. In the present study, we use
207 results from the thirteen groups that provided all health-related species (Table 1: Solazzo et
208 al., 2017a). The base-case emission inventories that are used in AQMEI for Europe and
209 North America are extensively described in Pouliot et al. (2015). For Europe, the 2009
210 inventory of TNO-MACC anthropogenic emissions was used. In regions not covered by the
211 emission inventory, such as North Africa, five modelling systems have complemented the
212 standard inventory with the HTAPv2.2 datasets (Janssens-Maenhout et al., 2015). For the



213 North American domain, the 2008 National Emission Inventory was used as the basis for the
214 2010 emissions, providing the inputs and datasets for processing with the SMOKE emissions
215 processing system (Mason et al., 2012). For both continents the regional scale emission
216 inventories were embedded in the global scale inventory (Janssens-Maenhout et al., 2015)
217 used by the global-scale HTAP2 modelling community so that to guarantee coherence and
218 harmonization of the information used by the regional scale modelling community. The
219 annual totals for European and North American emissions in the HTAP inventory are the
220 same as the MACC and SMOKE emissions. However, there are differences in the temporal
221 distribution, chemical speciation and the vertical distribution used in the models. The IFS
222 model (Flemming et al., 2015) provided chemical boundary conditions. Galmarini et al.
223 (2017) provides more details on the setup of the AQMEI3 and HTAP2 projects.

224 In addition to the base case simulations in AQMEI3, a number of emission perturbation
225 scenarios have been simulated (Table 1). The perturbation scenarios feature a reduction of
226 20% in the global anthropogenic emissions (GLO) as well as the HTAP2-defined regions of
227 Europe (EUR), North America (NAM) and East Asia (EAS), as explained in detail in
228 Galmarini et al. (2017). To prepare these scenarios, both the regional models and the global
229 IFS-MOZART model that provides the boundary conditions to the participating regional
230 models have been operated with the reduced emissions. For example, for the NAM scenario,
231 the IFS model makes a 20% reduction on the anthropogenic emissions from the HTAP-
232 defined NAM region, which is then used by the regional models as boundary conditions, in
233 addition to no emission perturbation for the European domain but the same perturbation for
234 the North American domain. Therefore, the NAM scenario is used to see the impact of long-
235 range transport of emissions from North America to Europe, while it shows the impact of the
236 North American sources on the North American air pollutant levels.

237 In addition to individual health impact estimates from each model, a multi-model mean
238 dataset (MM_m , in which all the modelling systems are averaged assuming equally weighted
239 contributions) has been created for each grid cell and time step, hence creating a new model
240 set of results that have the same spatial and temporal resolution of the ensemble-contributing
241 members. In addition to this simple MM_m , an optimal MM ensemble (MM_{opt}) has been
242 generated. MM_{opt} is created following the criteria extensively discussed and tested in the
243 previous phases of the AQMEI activity (Riccio et al., 2012; Kioutsioukis et al., 2016;
244 Solazzo and Galmarini, 2016), where it was shown that there are several ways to combine the
245 ensemble members to obtain a superior model, mostly depending on the feature we wish to
246 promote (or penalize). For instance, generating an optimal ensemble that maximizes the
247 accuracy would require a minimization of the mean error or of the bias, while maximizing the
248 associativity (variability) would require maximize the correlation coefficient (standard
249 deviation). In this study, the sub-set of models whose mean minimize the mean squared error
250 (MSE) is selected as optimal (MM_{opt}). The MSE is chosen for continuity with previous
251 AQMEI-related works. The MSE is chosen in the light of its property of being composed by
252 bias, variance and covariance types of error, thus lumping together measures of accuracy
253 (bias), variability (variance) and associativity (covariance) (Solazzo and Galmarini, 2016).
254 The minimum MSE has been calculated at the monitoring stations, where observational data



255 are available and then extended to the entire continental areas. This approximation might
256 affect remote regions away from the measurements. However, considering that for the main
257 pollutants (O_3 and $PM_{2.5}$) the network of measurements is quite dense around densely
258 populated areas (where the inputs of the MM ensemble are used for assessing the impact of
259 air pollutants on the health of the population), errors due to inaccurate model selection in
260 remote regions might be regarded as negligible (Solazzo and Galmarini, 2015). It should be
261 noted that the selection of the optimal combinations of models is affected by the model's bias
262 that might stem from processes that are common to all members of the ensemble (e.g.
263 emissions). Therefore, such a common bias does not cancel out when combining the models,
264 possibly creating a biased ensemble. Current work is being devoted to identify the optimal
265 combinations of models from which the offsetting bias is removed (Solazzo et al., 2017b).

266 2.2. EVA

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

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

286 The EVA system can be used to assess the number of various health outcomes including
287 different morbidity outcomes as well as short-term (acute) and long-term (chronic) mortality,
288 related to exposure of O_3 , CO and SO_2 (short-term) and $PM_{2.5}$ (long-term). Furthermore,
289 impact on infant mortality in response to exposure of $PM_{2.5}$ is calculated. EVA calculates and
290 uses the annual mean concentrations of CO, SO_2 and $PM_{2.5}$, while for O_3 , it uses the
291 SOMO35 metric that is defined as the yearly sum of the daily maximum of 8-hour running
292 average over 35 ppb, to calculate the acute effects of O_3 . The morbidity outcomes include
293 chronic bronchitis, restricted activity days, congestive heart failure, lung cancer, respiratory
294 and cerebrovascular hospital admissions, asthmatic children (<15 years) and adults (>15
295 years), which includes bronchodilator use, cough, and lower respiratory symptoms. The total



296 number of premature death is calculated as the sum of the acute deaths due to O₃ and the
297 chronic YOLL (years of life lost) by PM_{2.5} divided by 10.6 as recommended by the CAFÉ
298 (Clean Air for Europe Programme) report (Watkiss et al., 2005). The ERFs used, along with
299 their references, in both continents as well as the economic valuations for each health
300 outcome in Europe and the U.S., respectively, are presented in Table 2. For a full description
301 of the ERFs, see Andersen et al. (2004). The economic valuations are provided by Brandt et
302 al. (2013a); see also EEA (2013).

303 Table 2 lists the specific valuation estimates applied in the modelling of the economic
304 valuation of mortality and morbidity effects. A principal value of EUR 1.5 million was
305 applied for preventing an acute death, following expert panel advice (EC 2001). For the
306 valuation of a life year, the results from a survey relating specifically to air pollution risk
307 reductions were applied (Alberini et al., 2006), implying a value of EUR 57.500 per year of
308 life lost (YOLL). With the more conservative metric of estimating lost life years, rather than
309 'full' statistical lives, there is no adjustment for age. Most of the excess mortality is due to
310 chronic exposure to air pollution over many years and the life year metric is based on the
311 number of lost life years in a statistical cohort. Following the guidelines of the Organisation
312 for Economic Co-operation and Development (OECD, 2006), the predicted acute deaths,
313 mainly from O₃, are valued here with the adjusted value for preventing a fatality (VSL,
314 Value of a Statistical Life). The life tables are obtained from European data and are applied to
315 the U.S. as the average life expectancy in the U.S. is similar to that in Europe, and close to
316 the OECD average. The willingness to pay for reductions in risk obviously differs across
317 income levels. However, in the case of air pollution costs, adjustment according to per capita
318 income differences among different states is not regarded as appropriate, because long-range
319 transport implies that emissions from one state will affect numerous other states and their
320 citizens. The valuations are thus adjusted with regional purchasing power parities (PPP) of
321 EU27 and USA. The unit values have been indexed to 2013 prices as indicated in Table 2.

322 3. Results

323 3.1. Model Evaluation

324 Observed and simulated hourly surface O₃, CO, SO₂ and daily PM_{2.5}, which are species used
325 in the EVA model to calculate the health impacts, over Europe and North America for the
326 entire 2010 were compared in order to evaluate each model's performance. For a more
327 thorough evaluation of models and species, see Solazzo et al. (2017a). The results of this
328 comparison are presented in Table 3 for EU and NA, along with the multi-model mean and
329 median values. The monthly time series plots of observed and simulated health-related
330 pollutants are also presented in Figs. 2 and 3. The results show that over Europe, the temporal
331 variability of all gaseous pollutants is well captured by all models with correlation
332 coefficients (*r*) higher than 0.70 in general. The normalized mean biases (*NMB*) in simulated
333 O₃ levels are generally below 10% with few exceptions up to -35%. CO levels are
334 underestimated by up to 45%, while the majority of the models underestimated SO₂ levels by
335 up to 68%, while some models overestimated SO₂ by up to 49%. PM_{2.5} levels are
336 underestimated by 19% to 63%.



337 Over North America, the hourly O₃ variation is well captured by all models (Table 3), with
338 DK1 having slightly lower *r* coefficient compared to the other models and largest *NMB* (Fig.
339 3a). The hourly variation of CO and SO₂ levels are simulated with relatively lower *r* values
340 (Figs. 3b, c), with SO₂ levels having the highest underestimations. The PM_{2.5} levels are
341 underestimated by ~15% except for the DE1 model, having a large underestimation of 63%
342 (Table 3). As DE1 and US3 use the same SMOKE emissions and CTM, the large difference
343 in PM_{2.5} concentrations can be partly due to the differences in horizontal and vertical
344 resolutions in the model setups, as can also be seen in the differences in the CO
345 concentrations. There are also differences in the aerosol modules and components that each
346 model simulates. For example, DE1 uses an older version of the secondary organic aerosol
347 (SOA) module, producing ~3 μgm⁻³ less SOA, which can explain ~20% of the bias over
348 North America.

349 3.2. Health outcomes and their economic valuation in Europe

350 The different health outcomes calculated by each model in Europe as well as their multi
351 model mean and median are presented in Table 4. The health impact estimates vary
352 significantly between different models. The different estimates obtained are found to vary up
353 to a factor of three. Among the different health outcomes, the multi model ensemble
354 simulated the number of congestive heart failure cases to be between 19 000 to 41 000 (mean
355 of all individual models 31 000 ± 6 500). The number of lung cancer cases due to air
356 pollution are calculated to be between 30 000 to 78 000 (mean of all individual models
357 55 000 ± 14 000). Finally, the total (acute + chronic) number of premature death due to air
358 pollution is calculated to be 230 000 to 570 000 (mean of all individual models 414 000 ±
359 100 000). We have also produced a multi-model mean data (*MM_m*) and fed it to the EVA
360 model and obtained the number of premature death cases in Europe as 410 000 (difference
361 from the mean of all individual model estimates is smaller than 1%). The number of
362 premature death cases in Europe calculated by the multi model ensemble due to exposure to
363 O₃ is 12 000 ± 6 500, while the cases due to exposure to PM_{2.5} is calculated to be 390 000 ±
364 100 000 [180 000 – 550 000]. The O₃-related mortality well agrees with Liang et al. (2017)
365 that used the multi-model mean of the HTAP2 global model ensemble, which calculated an
366 O₃-related mortality of 12 800 [600 - 28 100]. The multi-model mean PM_{2.5}-related mortality
367 in the present study is much higher than that from the HTAP2 study (195 500 [4 400 –
368 454 800]). This difference can be attributed to the number of mortality cases as calculated by
369 the individual models, where the HTAP2 ensemble calculates a much lower minimum while
370 the higher ends from the two ensembles well agree.

371 The differences between the health outcomes calculated by the HTAP2 and AQMEII
372 ensembles arise firstly from the differences in the concentrations fields due to the differences
373 in models, in particular spatial resolutions as well as the gas and aerosols treatments in
374 different models, but also the differences in calculating the health impacts from these
375 concentrations fields. EVA calculates the acute premature death due to O₃ by using the
376 SOMO35 metric. On the other hand, in HTAP2 O₃-related premature death is calculated by
377 using the 6-month seasonal average of daily 1-h maximum O₃ concentrations. Both groups
378 use the annual mean PM_{2.5} to calculate the PM_{2.5}-related premature death. In addition to O₃



379 and PM_{2.5}, EVA also takes into account the health impacts from CO and SO₂, which is
380 missing in the HTAP2 calculations.

381 Among all models, DE1 model calculated the lowest health impacts for most health
382 outcomes, which can be attributed to the largest underestimation of PM_{2.5} levels (*NMB* =
383 63%: Table 3) due to lower spatial resolution of the model that dilutes the pollution in the
384 urban areas, where most of the population lives. The number of premature deaths calculated
385 by this study is in agreement with previous studies for Europe using the EVA system (Brandt
386 et al., 2013a; Geels et al., 2015). Recently, EEA (2015) estimated that air pollution is
387 responsible for more than 430 000 premature deaths in Europe, which is in good agreement
388 with the present study.

389 Fig. 4a. presents the geographical distribution of the number of premature death in Europe in
390 2010. The figure shows that the numbers of cases are strongly correlated to the population
391 density, with the largest numbers seen in the Benelux and the Po Valley regions that are
392 characterized as the pollution hot spots in Europe as well as in megacities such as London,
393 Paris, Berlin and Athens.

394 The economic valuation of the air pollution-associated health impacts calculated by the
395 different models along with their mean and median are presented in Table 5. A total cost of
396 196 to 451 billion Euros (*MM* mean cost of 300 ± 70 billion Euros) was estimated over
397 Europe (EU28). Results show that 5% [1% - 11%] of the total costs is due to exposure to O₃,
398 while 89% [80% - 96%] is due to exposure to PM_{2.5}. Brandt et al. (2013a) calculated a total
399 external cost of 678 billion Euros for the year 2011 for Europe, larger than the estimates of
400 this study, which can be explained by the differences in the simulation year and the emissions
401 used in the models as well as the countries included in the two studies (the previous study
402 includes e.g. Russia).

403 3.3. Health outcomes and their economic valuation in the U.S.

404 The different health outcomes calculated by each model for the U.S. as well as their mean
405 and median are presented in Table 4. The variability among the models (~3) is similar to that
406 in Europe. The number of congestive heart failure cases in the U.S. is calculated to be
407 13 000, while the lung cancer cases due to air pollution are calculated to be 22 000. Finally,
408 the number of premature deaths due to air pollution is calculated to be 165 000 ± 75 000,
409 where 25 000 ± 6 000 cases are calculated due to exposure to O₃ and 140 000 ± 72 000 cases
410 due to exposure to PM_{2.5}. The *MM_m* dataset leads to a number of premature death of 149 000
411 that is 6% smaller than the mean of individual models. The O₃- and PM_{2.5} mortality cases as
412 calculated by the AQMEII and HTAP2 model ensembles reasonably agree. Liang et al.
413 (2017) calculated an O₃-related mortality of 14 70 [900 – 30 400] and a PM_{2.5}-related
414 mortality of 78 600 [4 500 – 162 600]. Among all models, DE1 model calculated the lowest
415 health impacts for most health outcomes, which can be attributed to the largest
416 underestimation of PM_{2.5} levels (*NMB* = -63%: Table 4). The findings of this study are in
417 agreement with the U.S. EPA findings of premature death cases in the U.S., as well as the
418 Fann et al. (2012) study. The premature death cases in North America are mostly



419 concentrated over the New York area, as well as in hot spots over Chicago, Detroit, Houston
420 Los Angeles and San Francisco (Fig. 4b). The figure shows that the number of cases is
421 following the pattern of the population density by construction.

422 The economic valuation of the air pollution-associated health impacts calculated by the
423 different models in the U.S. are shown in Table 5. As seen in the table, a total cost of ~145
424 billion Euros is calculated. Results show that ~22% of the total costs is due to exposure to O₃
425 while ~78% is due to exposure to PM_{2.5}. The major health impacts in terms of their external
426 costs are slightly different in North America compared to Europe.

427 3.4. Health impacts and their economic valuation through optimal reduced ensemble subset

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

432 Results show that for the particular input (gridded air pollutant concentrations from
433 individual model)-output (each health outcome) configuration, the PM_{2.5} drives the variability
434 of the health impact indices (HII) and that at least 81% of the variation of the health indices
435 are explained by sole variations in the pollutants (i.e. without interactions: Table S1). Table
436 S1 also shows that the most important contribution to the HIIs is from PM_{2.5}, followed by CO
437 and O₃ (with much smaller influence though). The effect on the HII of perturbing PM_{2.5} by a
438 fixed fraction of its standard deviation is roughly double compared to CO and O₃.

439 We have run the EVA system over an all-models mean (MM_m) dataset and an optimal
440 reduced ensemble dataset (MM_{opt}) calculated for each of the pollutants in the two domains in
441 order to see how and whether an optimal reduced ensemble changes the assessment of the
442 health impacts compared to an all- models ensemble mean. Table 6 shows some sensible
443 error reduction, although the temporal and spatial averages mask the effective improvement
444 in accuracy from MM_m to MM_{opt} . In Europe, the optimal reduced ensemble decreases the
445 RMSE by up to 24%, while in NA, the error reduction is much larger (4% to up to 147%). On
446 a seasonal basis, MM_{opt} reduces RMSE in PM_{2.5} over Europe by 23% in winter while smaller
447 decreases are achieved in other seasons (~10%). Regarding O₃, improvement is 16%-22%,
448 with the largest improvement in spring. In NA, the improvement in winter RMSE in PM_{2.5} is
449 smallest (~2%) while larger improvements are achieved in other seasons (~7% - ~9%). For
450 O₃, the largest RMSE reduction in NA is achieved for the summer period by 14%.

451 The analysis of the aggregated health indices data for Europe (Table S1) shows that EVA
452 indices rely principally on the PM_{2.5} levels and then the CO and O₃ values. Therefore, the
453 relative improvement of the indices with the optimal ensemble should be proportional to the
454 relative improvement in PM_{2.5}, CO and O₃. The proportionality rate for each pollutant is
455 given in Table S1, assuming all pollutants are varied (from MM_m to MM_{opt}) away from their
456 mean by the same fraction of their variance. As seen in the Table 7, from MM_m to MM_{opt} , the
457 health indices increase by up to 30% in Europe. This increase is due to a 27% increase in the
458 domain mean PM_{2.5} levels when the optimal reduced ensemble is used, as well a slight



459 increase in O₃ by ~1%. The number of premature deaths in Europe increase from 410 000 to
460 524 000 (28%), resulting in a much higher estimate compared to previous mortality studies.
461 On the contrary, in the U.S., the mean PM_{2.5} and O₃ levels decrease from 2.94 μg m⁻³ to 2.62
462 μg m⁻³ (~11%) and 18.7 ppb to 18.4 ppb (~2%), respectively. In response, the health indices
463 decrease by ~11% (Table 7). The number of premature death cases in NA decrease from
464 149 000 to 133 000.

465 3.5. Impact of anthropogenic emissions on the health impacts and their economic valuation

466 The impacts of emission perturbations on the different health outcomes over Europe and the
467 U.S. as calculated by the individual models are presented in Tables S2-S4. Table 8 shows the
468 impacts of the different emission perturbations on the premature death cases in Europe and
469 the U.S. as calculated by a subset of models that simulated the base case and all three
470 perturbation scenarios *MM_c*. Results show that in Europe, the 20% reduction in the global
471 anthropogenic emissions leads to ~17% domain-mean reduction in all the health outcomes,
472 with a geographical variability as seen in Fig. 4c. The figure shows that the larger changes in
473 mortality is calculated in the central and northern parts of Europe (15-20% decreases), while
474 the changes are smaller in the Mediterranean region (5-10%), highlighting the non-linearity
475 of the response to emission reductions. However, it should be noted that global models or
476 coarse-resolution regional models (as in this study) cannot capture the urban features and
477 pollution levels and thus, non-linearities should be addressed further using fine spatial
478 resolutions or urban models. The models vary slightly simulating the response to the 20%
479 reduction in global emissions, estimating decreases of ~11% to 20%. The number of
480 premature deaths decreased on average by ~50 000, ranging from -39 000 (DK1) to -103 000
481 (IT1). This number is in good agreement with the ~45 000 premature death calculated by the
482 HTAP2 global models (Liang et al., 2017). The *MM_c* ensemble calculated a 15% and 17%
483 decrease in the O₃- and PM_{2.5}-related premature death cases, respectively, in response to the
484 GLO scenario. This decrease in the global anthropogenic emissions leads to an estimated
485 decrease of 56 ± 18 billion Euros in associated costs in Europe (Table 8).

486 As seen in Table 8, a 20% reduction of anthropogenic emissions in the EUR region, as
487 defined in HTAP2, avoids 47 000 premature death, while a 20% reduction of the
488 anthropogenic emissions in the NAM region leads to a much smaller decrease of premature
489 deaths (~1 000). These improvements in the number of premature deaths are in agreement
490 with a recent HTAP2 global study that calculated reductions of ~34 000 and ~1 000 for the
491 EUR and NAM scenarios, respectively (Liang et al., 2017). Both the global and regional
492 models agree that the largest impacts of reducing emissions with respect to premature deaths
493 come from emission within the source region, while foreign sources contribute much less to
494 improvements in avoiding adverse impacts of air pollution. The decreases in health impacts
495 in EUR and NAM scenarios corresponds to decreases in the associated costs by -47 ± 16
496 billion Euros and -1.4 ± 0.4 billion Euros, respectively. This is consistent with results in
497 Brandt et al. (2012), where a contribution of ~1% to PM_{2.5} concentrations in Europe is
498 originating from the NAM region.



499 The 20% reduction in global anthropogenic emissions leads to 18% reduction in the health
500 outcomes (Table 8) in the U.S., with a geographical variability in the response. Fig. 4d shows
501 that the largest decreases in mortality is calculated for the western coast of the U.S. (~20%)
502 and slightly lower response in the central and eastern parts of the U.S. (15-20%). The number
503 of premature death cases, as calculated by the mean of all individual models decreases from
504 $\sim 160\,000 \pm 70\,000$ to $\sim 130\,000 \pm 60\,000$, avoiding 24 ± 10 billion Euros (Table 8) in
505 external costs, also in agreement with the ensemble of HTAP2 global models ($\sim 23\,000$) The
506 O₃-related premature death cases decreased by 42% while the PM_{2.5}-related cases decreased
507 by 18%. A 20% reduction of the North American emissions avoids $\sim 25\,000 \pm 12\,000$
508 premature deaths (-16%), suggesting that ~80% of avoided premature deaths are achieved by
509 reductions within the source region while 20% ($\sim 5\,000$ premature deaths) is from foreign
510 sources. This number is also in good agreement with Liang et al. (2017) that estimated a
511 reduction of premature deaths of $\sim 20\,000$ due to O₃ and PM_{2.5} in the United States due to an
512 emission reduction of 20% within the region itself, using the ensemble mean of the HTAP2
513 global models. The corresponding benefit is calculated to be 21 ± 9 billion Euros in the NAM
514 scenario. According to results from the EAS scenario, among these 5 000 avoided cases that
515 are attributed to the foreign emission sources, $1\,900 \pm 2\,000$ premature deaths can be avoided
516 by a 20% reduction of the East Asian emissions, avoiding 2.5 ± 3 billion Euros. Our number
517 of avoided premature deaths due to the EAS scenario is much higher than 580 avoided
518 premature deaths calculated by Liang et al. (2017).

519 Conclusions

520 The impact of air pollution on human health and their economic valuation for the society
521 across Europe and the United States is modelled by a multi-model ensemble of regional
522 models from the AQMEII3 project. All regional models used boundary conditions from the
523 C-IFS model, and emissions from either the MACC inventory in Europe or the EPA
524 inventory for the North America, or the global inventory from HTAP. Sensitivity analysis on
525 the dependence of models on different sets of boundary conditions has not been conducted so
526 far but large deviations from the current results in terms of health impacts are not expected.
527 The modelled surface concentrations by each individual model are used as input to the EVA
528 system to calculate the resulting health impacts and the associated external costs from O₃,
529 CO, SO₂ and PM_{2.5}. Along with a base case simulation for the year 2010, some groups
530 performed additional simulations, introducing 20% emission reductions both globally and
531 regionally in Europe, North America and East Asia.

532 The base case simulation of each model is evaluated with available surface observations in
533 Europe and North America. Results show large variability among models, especially for
534 PM_{2.5}, where models underestimate by ~20% - ~60%, introducing a large uncertainty in the
535 health impact estimates as PM_{2.5} is the main driver for health impacts. The differences in the
536 models are largely due to differences in the spatial and vertical resolutions, meteorological
537 inputs, natural emissions, as well as missing or underestimated SOA mass, which is critical
538 for the PM_{2.5} mass.



539 The variability of health impacts among the models can be up to a factor of three in Europe
540 (twelve models) and the U.S. (three models), among the different health impacts. The multi-
541 model mean total number of premature death is calculated to be 414 000 in Europe and
542 160 000 in the U.S., where PM_{2.5} contributes by more than 90%. These numbers agree well
543 with previous global and regional studies for premature deaths due to air pollution. In order to
544 reduce the uncertainty coming from each model, an optimal ensemble set is produced, that is,
545 the subset of models that produce the smallest error compared to the surface observations at
546 each time step. The optimum ensemble results in an increase of health impacts by up to 30%
547 in Europe and a decrease by ~11% in the United States. These differences clearly
548 demonstrate the importance of the use of optimal-reduced multi-model ensembles over
549 traditional all model-mean ensembles, both in terms of scientific results, but also in policy
550 applications.

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

559 **Outlook**

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

567 Regarding ambient concentrations of PM and the exposure-response functions (ERFs), there
568 is a rich set of studies providing information on total PM mass. However, only few studies
569 focus on individual particulate species, mainly black carbon and carbonaceous particles. In
570 addition to PM, studies on human populations have not been able to isolate potential effects
571 of NO₂, because of its complex link to PM and O₃. The WHO REVIHAAP review from 2013
572 concludes that health assessments based on PM_{2.5} ERFs will be most inclusive (WHO, 2013).
573 In addition, the ERFs are based on urban background measurements, introducing
574 uncertainties regarding non-urban areas or high pollution areas as e.g. street canyons. Current
575 state-of-the-art health impact estimates, in particular on regional to global scales, assume a
576 correlation with exposure to outdoor air pollution, while in reality, exposure is dynamic and
577 depends on the behavior of the individual. In addition, differences in age groups, gender,
578 ethnicity and behavior should be considered in the future studies. There are also uncertainties
579 originating from the representations of the aerosols in the atmospheric models used in the



580 calculation of pollutant concentrations as well as the emissions. Further developments in the
581 aerosol modules, such as the representation of organic aerosols and windblown and
582 suspended dust, are need in order to achieve mass closure of PM to get robust estimates of
583 health impacts.

584 Due to above reasons, there is a large knowledge gap regarding the health impacts of
585 particles. There are a number of ongoing projects trying to identify the health impacts from
586 individual particle components and produce individual ERFs for these components.
587 NordicWelfAir project (<http://projects.au.dk/nordicwelfair/>) aims to investigate the potential
588 causal impact of individual chemical air pollutants as well as mixtures of air pollutants on
589 health outcomes. In pursuing this aim, the project uses the unique Nordic population-based
590 registers allowing linkage between historical residential address, air pollutants over decades
591 and later health outcomes. By linking the exposure to health outcomes, new exposure-
592 response relationships can be determined of health effects for different population groups
593 (e.g. age, education, ethnicity, gender, lifestyle, and working life vs. retirement conditions)
594 related to air pollution for the individual chemical air pollutants. In addition, the high
595 resolution simulations conducted will enable us to have a better understanding of non-
596 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	Europe				North America				
					BASE	GLO	NAM	EUR	BASE	GLO	EAS	NAM	
DE1	COSMO-CLM/CMAQ	HTAP	24 km × 24 km	30 layers, 50 hPa	×	×	×	×	×	×	×	×	×
DK1	WRF/DEHM	HTAP	50 km × 50 km	29 layers, 100 hPa	×	×	×	×	×	×	×	×	×
ES1	WRF/CHEM	MACC	23 km × 23 km	33 layers, 50 hPa	×		×						
FI1	ECMWF/SILAM	MACC	0.25° × 0.25°	12 layers, 13 km	×	×	×	×					
FRES1	ECMWF/CHIMERE	HTAP	0.25° × 0.25°	9 layers, 50 hPa	×	×	×	×					
IT1	WRF/CAMx	MACC	23 km × 23 km	33 layers, 50 hPa	×	×		×					
IT2	WRF/CHEM	MACC	23 km × 23 km	14 layers, 8 km	×	×							
NL1	LOTOS/EUROS	MACC	0.50° × 0.25°	4 layers, 3.5 km	×								
TR1	WRF/CMAQ	MACC	30 km × 30 km	24 layers, 10hPa	×	×	×	×					
UK1	WRF/CMAQ	MACC	15 km × 15 km	23 layers, 100 hPa	×	×	×	×					
UK2	WRF/CMAQ	HTAP	30 km × 30 km	23 layers, 100 hPa	×	×							
UK3	WRF/CMAQ	MACC	18 km × 18 km	35 layers, 16 km	×	×	×	×					
US3	WRF/CAMx	SMOKE	12 km × 12 km	35 layers, 50 hPa					×	×	×	×	×



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

Health effects (compounds)	Exposure-response coefficient	Valuation, € ₀₁₃
	(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. Model evaluation over the European and North American domains (hourly for O₃, CO and SO₂ and daily means for PM_{2.5}). Units are % for NMB and NMGE, $\mu\text{g m}^{-3}$ for all species for Europe and ppb for the gaseous species and $\mu\text{g m}^{-3}$ for PM_{2.5} in North America.

Models	O ₃			CO			SO ₂			PM _{2.5}						
	r	NMB	NMGE	RMSE	r	NMB	NMGE	RMSE	r	NMB	NMGE	RMSE				
Europe																
DE1	0.73	9.87	4.59	13.50	0.80	-42.07	41.75	133.24	0.77	4.34	21.07	1.33	0.88	-63.08	128.10	11.95
DK1	0.88	6.71	2.59	9.99	0.74	-41.67	43.10	135.84	0.85	-47.24	56.49	1.54	0.86	-45.69	56.82	9.65
ES1	0.79	-15.16	6.59	14.21	0.59	-46.27	55.42	147.82	0.78	-68.13	182.02	2.15	0.23	-30.84	44.68	9.66
FI1	0.84	-35.87	24.00	23.58	0.85	-26.75	15.78	92.11	0.82	-20.49	17.26	1.05	0.58	-26.98	29.18	8.02
FRES1	0.78	-9.65	4.79	12.51	0.82	-39.19	34.10	123.37	0.74	-76.81	320.13	2.44	0.87	-36.16	32.25	7.88
IT1	0.90	4.20	2.45	9.60	0.82	-36.81	31.23	120.35	0.79	-29.78	28.19	1.26	0.78	-18.25	14.88	6.06
IT2	0.92	-14.26	4.46	11.76	0.77	-43.53	45.13	136.44	0.81	-54.78	87.14	1.77	0.11	-48.40	87.23	11.65
NL1	0.92	-5.06	2.01	8.30	0.69	-46.09	55.74	148.51	0.80	-51.92	83.39	1.79	0.76	-55.55	99.64	11.56
TR1	0.86	8.09	8.91	18.65	0.84	-20.11	9.40	74.24	0.43	2.28	24.06	1.40	0.60	-19.16	21.08	7.17
UK1	0.91	7.51	2.13	9.10	0.59	-41.56	44.88	138.72	0.73	-12.96	16.19	1.06	0.78	-40.32	44.67	8.97
UK2	0.83	-2.75	4.17	12.10	0.64	-42.63	45.23	138.00	0.72	20.46	17.82	1.31	0.77	-28.28	23.59	7.15
UK3	0.78	-1.01	4.04	12.01	0.80	-45.04	48.32	139.60	0.64	48.75	46.00	2.34	0.94	-43.82	42.44	8.48
MEAN	0.84	-3.95	5.89	12.94	0.75	-39.31	39.17	127.35	0.74	-23.86	74.98	1.62	0.68	-38.04	52.05	9.02
MEDIAN	0.85	-1.88	4.32	12.06	0.78	-41.87	43.99	136.14	0.77	-25.14	37.10	1.47	0.77	-38.24	43.56	8.73
North America																
DE1	0.85	5.55	11.65	4.69	0.41	-40.68	40.71	92.20	0.45	-40.20	41.05	1.35	0.74	-62.65	62.65	6.97
DK1	0.72	21.75	23.80	10.33	0.47	-7.41	18.02	47.32	0.63	-42.36	43.47	1.35	0.64	-14.08	22.21	2.86
US3	0.88	-1.53	11.18	4.51	0.44	-3.89	19.89	51.42	0.52	-12.83	23.98	0.84	0.76	17.23	23.75	3.25
MEAN	0.82	8.59	15.54	6.51	0.44	-17.33	26.21	63.65	0.53	-31.80	36.17	1.18	0.71	-19.83	36.21	4.36
MEDIAN	0.85	5.55	11.65	4.69	0.44	-7.41	19.89	51.42	0.52	-40.20	41.05	1.35	0.74	-14.08	23.75	3.25



Table 4. Health impacts as calculated by the individual models over Europe and the United States ($\times 10^3$, except for IM). See Table 2 for the definitions of health impacts.

Models	CB	RAD	RHA	CHA	CHF	LC	BUC	BUA	COUC	COUA	LRSC	LRSA	PD	IM
Europe														
DE1	191	194 776	13	24	19	29	5 694	37 284	19 674	38 380	7 592	13 844	232	213
DK1	290	296 611	17	37	26	44	8 671	56 776	29 960	58 446	11 562	21 082	336	325
ES1	415	424 229	23	53	34	64	12 402	81 205	42 851	83 593	16 536	30 153	456	465
FI1	411	420 220	25	53	35	63	12 285	80 437	42 445	82 803	16 380	29 868	457	460
FRES1	373	381 243	22	48	32	57	11 146	72 976	38 509	75 123	14 861	27 098	419	418
IT1	507	517 996	30	65	41	78	15 144	99 153	52 322	102 070	20 191	36 818	571	568
IT2	310	317 256	18	40	27	48	9 275	60 728	32 045	62 514	12 367	22 550	345	348
NL1	264	269 418	16	34	24	40	7 876	51 571	27 213	53 088	10 502	19 150	303	295
TRI	460	470 496	29	59	40	70	13 755	90 061	47 524	92 710	18 340	33 442	538	516
UK1	343	351 026	23	44	30	53	10 262	67 192	35 456	69 169	13 683	24 950	404	516
UK2	417	425 950	28	53	35	64	12 453	81 534	43 024	83 932	16 603	30 275	488	467
UK3	342	349 974	26	44	29	52	10 231	66 991	35 350	68 961	13 642	24 875	416	383
MEAN	360	368 266	23	46	31	55	10 766	70 492	37 198	72 566	14 355	26 175	414	414
MEDIAN	358	366 135	23	46	31	55	10 704	70 084	36 982	72 146	14 272	26 024	418	439
The United States														
DE1	61	62 305	5	8	7	9	1 946	11 926	6 722	12 277	2 594	4 428	80	61
DK1	161	164 681	10	21	15	25	5 148	31 522	17 787	32 449	6 864	11 705	191	161
US3	204	209 023	13	27	18	31	6 604	40 009	22 819	41 186	8 806	14 856	224	209
MEAN	142	145 337	10	19	13	22	4 566	27 819	15 776	28 637	6 088	10 330	165	143
MEDIAN	161	164 681	10	21	15	25	5 148	31 522	17 787	32 449	6 864	11 705	191	161



Table 5. 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 6. Annual average RMSE of the multi-model ensemble mean (MM_m) and of the optimal reduced ensemble mean (MM_{opt}) for the health impact-related species. Units are $\mu\text{g m}^{-3}$ for all species for Europe and ppb for the gaseous species and $\mu\text{g m}^{-3}$ for $\text{PM}_{2.5}$ in North America.

	O_3		CO		SO_2		$\text{PM}_{2.5}$	
	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 7. All models-mean (MM_m) vs. optimal ensemble (MM_{opt}) calculated for different health impacts over Europe and U.S ($\times 10^3$, except for IM).

	Europe		The United States	
	MM_m	MM_{opt}	MM_m	MM_{opt}
CB	360	468	142	125
RAD	368 000	478 100	145 300	127 900
RHA	23	28	8	7
CHA	46	60	19	16
CHF	31	38	9	8
LC	55	72	22	19
BDUC	10 800	14 000	4 600	4 000
BDUA	70 500	91 500	27 800	24 500
COUC	37 200	48 300	15 800	13 900
COUA	72 600	94 200	28 600	25 200
LRSC	14 400	18 600	6 100	5 400
LRSA	26 170	34 000	10 300	9 100
PD	410	524	149	133
IM*	403	524	143	126



Table 8. Impact of the emission reduction scenarios on avoided premature death (Δ PD) 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	
	Δ PD	Δ Total Cost (billion €)	Δ PD	Δ Total Cost (billion €)
GLO	$-54\,000 \pm 18\,000$	-56 ± 18	$-27\,500 \pm 14\,000$	-24 ± 10
NAM	-940 ± 1100	-1.4 ± 0.4	$-25\,000 \pm 12\,000$	-21 ± 9
EUR	$-47\,000 \pm 24\,000$	-47 ± 16	-	-
EAS	-	-	$-1\,900 \pm 2\,200$	-2.5 ± 3

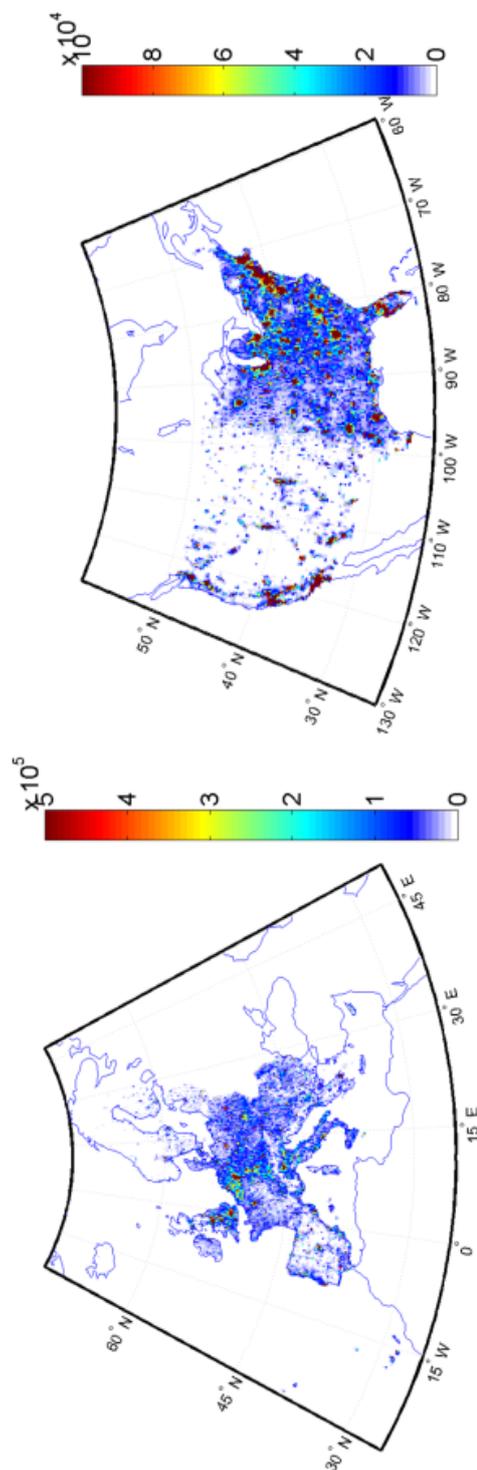


Fig.1. Population density over Europe and the United States.

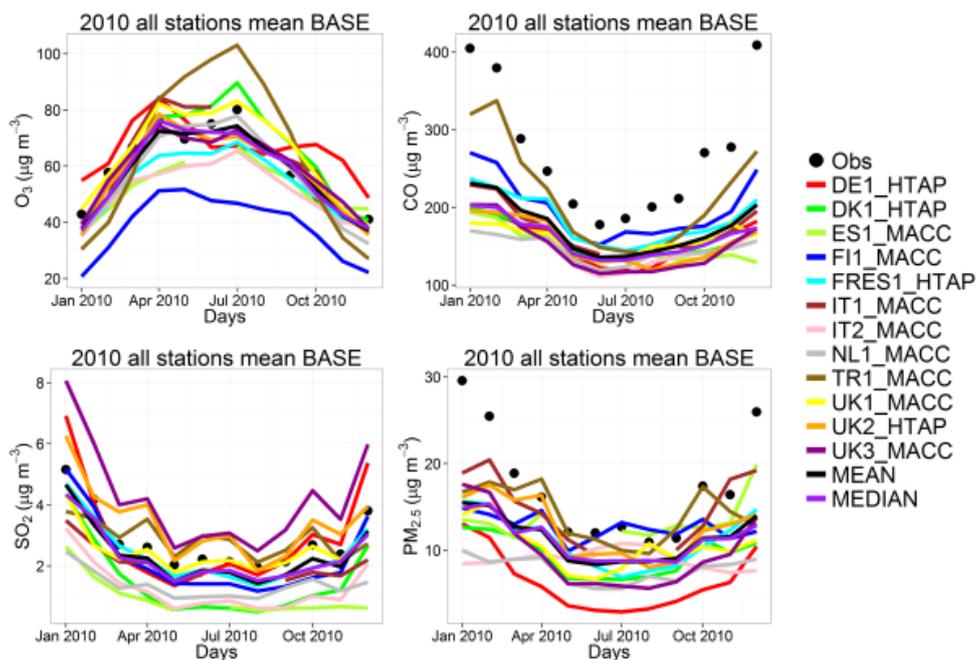


Fig. 2. Observed and simulated (base case) monthly a) O₃, b) CO, c) SO₂ and d) PM_{2.5} concentrations over Europe.

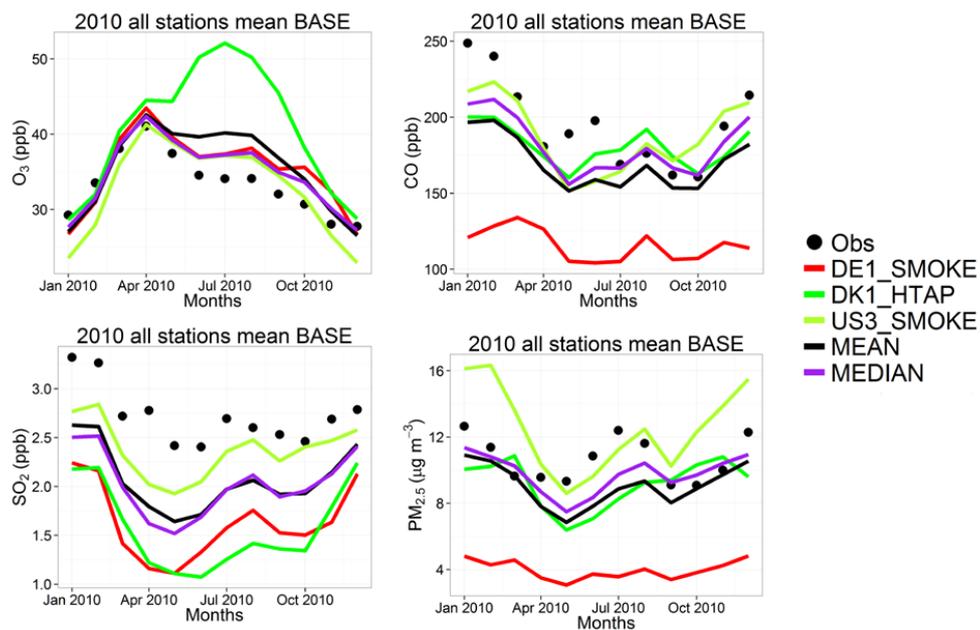


Fig. 3. Observed and simulated (base case) monthly a) O_3 , b) CO , c) SO_2 and d) $PM_{2.5}$ concentrations over the U.S.

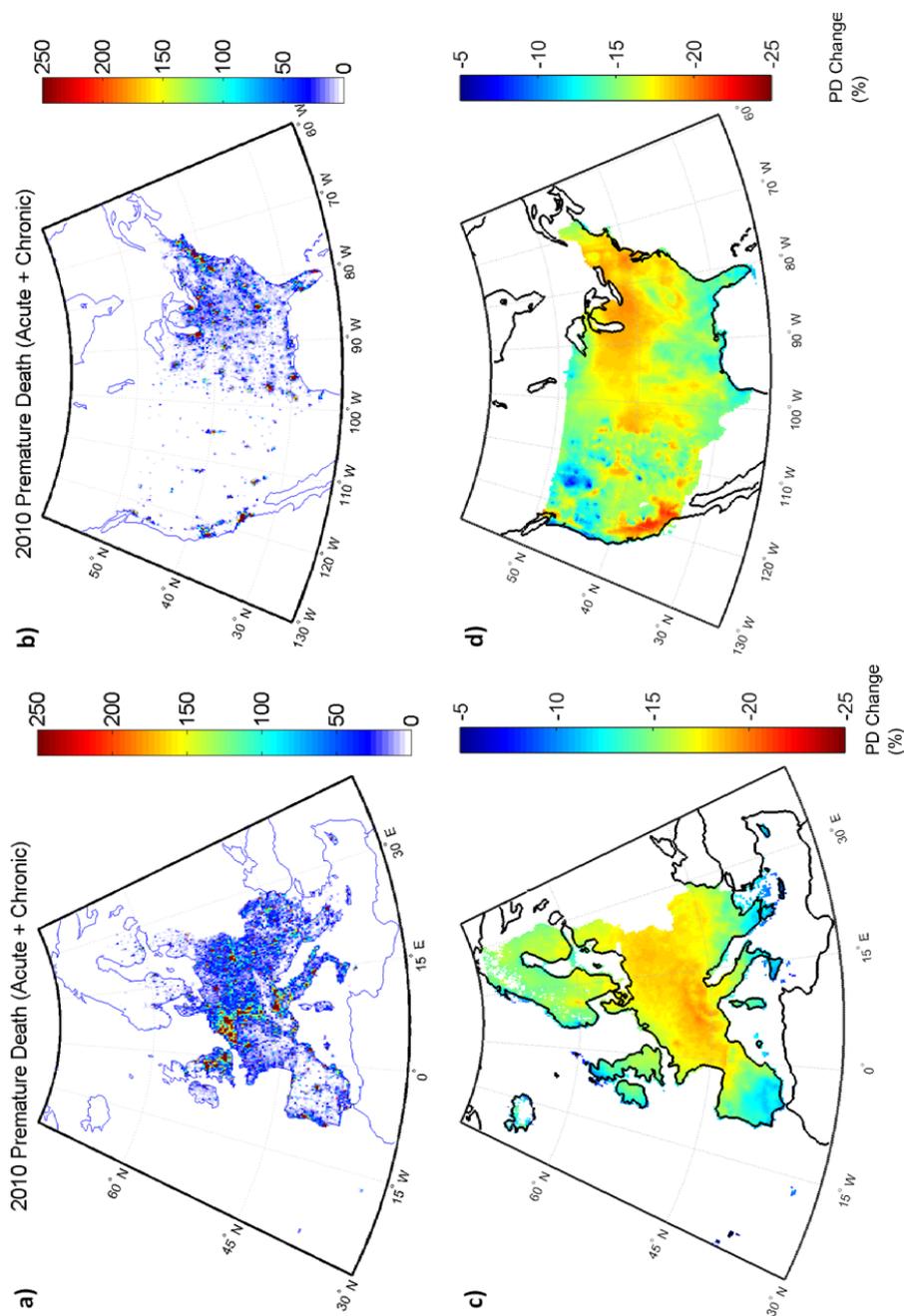


Fig. 4. Spatial distribution of the number of total premature death in Europe and the United States in 2010 as calculated by the multi-model mean ensemble.