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Measuring welfare loss caused by air pollution in Europe: A CGE analysis

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ABSTRACT

To evaluate the socio-economic impacts of air pollution, we develop an integrated approach based on computable general equilibrium (CGE). Applying our approach to 18 western European countries shows that even there, where air quality is relatively high compared with other parts of the world, health-related damages caused by air pollution may be substantial. We estimate that as of 2005, Europe experienced an annual loss in consumption of about 220 billion Euro in year 2000 prices (about 3% of total consumption) with a range based on 95% high and low epidemiological response functions of 107–335 billion Euro and a total welfare loss of about 370 billion Euro (range of 209–550) including both consumption and broader welfare losses (around 2% of welfare level) due to the accumulated effects of three decades of air pollution in Europe. In addition, we estimate that a set of air quality improvement policy scenarios as proposed in the 2005 CAFE program would bring 18 European countries as a whole a welfare gain of 37–49 billion Euro (year 2000 prices) in year 2020 alone.

1. Introduction

Outcomes related to human health account for the majority of the socio-economic costs induced by air pollution (EPA, 1997; Holland et al., 1999). This paper evaluates the impacts of air pollution on human health in Europe and on the European economy using an integrated model of pollution–health dynamics. Compared with standard methods, our approach addresses more comprehensively the cumulative health and economic burden of exposure to air pollution and the benefits of reducing pollution.

Conventional methods employed in other studies to quantify the health impacts of air pollutants are static, and provide estimates of damages at a single point in time (e.g., Aunan et al., 2004; Burtraw et al., 2003; Davis et al., 1997; EPA, 1999; Ostro and Chestnut, 1998; Vennemo et al., 2006; West et al., 2006; Williams, 2002, 2003; World Bank and SEPA, 2007). Point estimates may substantially underestimate health impacts of air pollution, because air pollution can affect health outcomes that only appear years later, and the effects of pollution can be cumulative. An example of this is premature death caused by chronic exposure to particulates.

A few studies have attempted to measure the health impacts of air pollution in the European region. Early studies defined exposure–response functions on the basis of existing epidemiological studies, and computed the number of diseases and premature deaths caused by air pollution at a single time (Krupnick et al., 1996; Olsthoorn et al., 1999; Künzli et al., 2000). They then valued these health endpoints by using survey data such as average costs that people are willing to pay in order to avoid specific health-related outcomes. More recent studies use a computable general equilibrium (CGE) modeling approach in order to assess economic impacts over time (Holland et al., 2005; Mayeres and van Regemorter, 2008). In their approach, labor and leisure loss caused by air pollution can affect market equilibrium in the future. In their CGE models, however, premature deaths due to chronic exposure are dealt with in the same manner as those due to acute exposure, which inaccurately captures the flow of lost labor over time.

We go beyond these previous studies by analyzing the economic impacts on health that result from cumulative and acute exposure as it occurs over time. We apply to Europe a method that was developed and applied to the United States and China (Matus, 2005; Matus et al., 2008). We consider 15 separate health endpoints[1] in combination with observed and modeled air

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[1] The 15 health endpoints include respiratory hospital admission, cerebrovascular hospital admission, cardiovascular hospital admission, respiratory symptom days, acute mortality, chronic bronchitis, chronic cough (only for children), cough and wheeze, restricted activity day, minor restricted activity day, work loss day, congestive heart failure, asthma attacks, bronchodilator usage, and chronic mortality.

pollution data from 1970 to 2005 to estimate the lost time and additional expenditures on health care. We then apply a CGE model of the economy to estimate the total economic impact, valuing both work and non-work (i.e., leisure) time as well as the economic cost of reallocating economic resources to the health care sector. An important implication of this approach demonstrated by previous applications is that economic damages accumulate—lost income in earlier years means lower GDP and savings, and therefore less investment and growth over time. In this study, we do not consider the costs of mitigating air pollution.

The paper is organized as follows. In Section 2 we describe the CGE model and modifications made to analyze health effects. Section 3 outlines the economic and epidemiological inputs and Section 4 the air quality data used in our study. In Section 5 we discuss the results of our simulations and a sensitivity analysis with respect to exposure–response relationships, and provide our benchmark analysis to Clean Air for Europe (CAFE)-proposed emission scenarios. Finally, Section 6 summarizes our results and discusses some of the caveats of our approach.

2. Theoretical framework and method: EPPA-HE

For our analysis, we use the MIT Emissions Prediction Policy Analysis (EPPA) model, modified as reported in Matus et al. (2008) to address health effects and with updates and applications to Europe described below. EPPA is a multi-region, multi-sector, recursive dynamic CGE model of the world economy (Paltsev et al., 2005), which uses economic data from the GTAP dataset (Dimaranan and McDougall, 2002).

Using a CGE model to estimate pollution costs has two major advantages. One is that a CGE model can describe economic dynamics (savings and investment) and resource reallocation implications of lost labor, leisure, and additional demands on the health services sector. The second is that a CGE model allows analysis of multiple scenarios. Our approach is to first develop a historical benchmark simulation that replicates actual economic performance where the health impacts associated with observed levels of pollution are included. We then analyze what would have happened if air pollution were at background levels, in order to estimate what economic performance would have been without pollution stemming from human activity. The difference between economic performance from this counterfactual scenario and our replication of actual performance gives us an estimate of the economic burden of air pollution. The estimate of burden changes over time as pollution levels change and as past exposure continues to affect economic performance. These dynamic effects of past exposure stem from lost lives due to chronic exposure and the impacts of lower economic activity on savings and investment, which then carry through to lower economic activity in future years. Our primary measure of economic performance is a change in welfare, which includes consumption and leisure and is measured as equivalent variation. Consumption is measured as total macroeconomic consumption. Leisure time is valued at the marginal wage rate. An average wage profile over the lifetime of an individual is applied to each age cohort to estimate the impact of air-pollution related deaths. Our counterfactual scenarios include simulation of the potential benefits of certain pollution goals.

As mentioned above, the EPPA-Health Effects (EPPA-HE) model is described in Matus et al. (2008). Briefly, it accommodates pollution-generated health costs in a feedback loop, which in turn affects the economy and the emissions of pollutants in later periods. The extended social accounting matrix (SAM), on which EPPA-HE is based, includes a household production sector that uses medical services and household labor to provide pollution health service (Fig. 1). An increase in pollution health related household labor reduces the pool of labor and leisure available for other economic activities. The elasticity of substitution between work time and non-work time (σ), which defines labor–leisure choices in the model, is parameterized at 0.2, given the own-price supply elasticity of labor (ε) of 0.25 (Babiker et al., 2002) and the estimated initial share of non-working time (α) of 0.55 (Matus et al., 2008). The relationship between ε and σ is:

$$\sigma = \frac{1-\alpha}{\alpha} \cdot \varepsilon \quad (1)$$

The allocation of total time between labor and leisure is thus endogenous and depends on the substitute elasticity and changes in the wage rate.

The EPPA-HE model captures the magnitude of pollution health impacts on the basis of the size of additional medical services and their factor inputs, produced by air pollution and the amount of labor and leisure lost due to acute and chronic exposure to pollutants. We report consumption changes that reflect market output and production effects, and welfare differences that include the change in leisure.[2] As described below, our valuation approach includes stated preference estimates of pain, suffering, and inconvenience associated with illness which we treat as a loss of leisure time, in addition to losses of work time and the cost of medical care. As our analysis is limited to the European aggregation in the EPPA model, which aggregates 18 European countries[3] into one region (EUR), we do not consider the EU-27, but only a subset of the EU countries (plus Norway, Iceland, and Switzerland) as a single region.[4]

EPPA-HE computes 15 different health outcomes on the basis of historical pollution levels, exposure–response (E–R) relationships, and demographic information. The health outcomes are then converted into health service requirements (i.e., cost of medical care) and lost labor and leisure where the leisure loss includes valuation of pain, suffering, and inconvenience caused by illness. These changed levels of health service demands and labor availability are simulated through EPPA-HE to calculate the broader effect on the economy, including a measure of welfare change.

Following the air pollution health effects literature, we treat deaths due to chronic and acute pollution exposure differently. For acute exposure, we follow the literature and assume that deaths in such cases occur to individuals whose health condition was already poor, with pollution exposure leading to half a year of life lost on average. For chronic exposure, we assume that death is related to cardio-pulmonary or lung disease, and so we use age-specific death rates from these diseases to estimate a distribution for the age of death. To do this, we include a demographic module in the model that tracks 5-year age cohorts, their exposure level throughout their lifetime and the death rate for each from cardio-pulmonary and lung diseases for each cohort. We assume that an increase in the death rate due to chronic exposure proportionally

[2] We focus on aggregate welfare and consumption changes. One might consider per capita effects but that raises an issue of whether to include those who died prematurely. We would use as a denominator population without pollution to include those individuals who had died from pollution exposure. If we evaluated per capita welfare with different population levels, we end up with the possibility that pollution could be a benefit to the remaining population, which then does not account for the welfare loss of those who died prematurely. If births were endogenous, the deeper philosophical issue would arise of whether the potential welfare of those who never existed should be considered.

[3] The region EUR in EPPA version 4 includes Austria, Belgium, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Italy, Luxembourg, the Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and the United Kingdom.

[4] EU-15 countries, represented in EUR region, account for 95% of the EU-27 GDP and 78% of the EU-27 population.

		INTERMEDIATE USE						*HOUSEHOLD SERVICES*		FINAL USE				OUTPUT
		1	2	…	*j*	…	*n*	*Mitigation of Pollution Health Effects*	*Labor-Leisure Choice*	Consumption	Investment	Government Purchase	Net-export	
DOMESTIC PRODUCTION	1													
	2													
	:													
	i													
	:													
	Medical Services for Health Pollution							*Medical Services*		*Health Services*				
	n													
IMPORTS	1													
	2													
	:													
	i													
	:													
	n													
LEISURE									*Leisure*	*Leisure*				
VALUE-ADDED	Labor							*Labor*	*Labor*					
	Capital													
	Indirect Taxes													
	Resources													
INPUT														

Fig. 1. Extended social accounting matrix for EPPA-HE. *Note*: Components of the added to the traditional SAM to capture health effects are displayed in italics.

increases the cardio-pulmonary and lung death rate in each cohort. Because deaths from these diseases are much less prevalent among younger people who have had less time for these diseases to develop, this weights the deaths to be among the older population thereby reducing the average number of years of lost life. We assume that (i) death from chronic exposure occurs only in age groups of 30 and older and (ii) the life expectancy of those who died due to chronic exposure is 75. We follow Bickel and Friedrich (2005) in excluding the possibility of death due to chronic exposure for those under 30. Deaths prior to age 30 are believed to be negligible because illnesses from chronic exposure take many years to develop. In addition, we follow Matus et al. (2008) in choosing an expected age of 75. The life expectancy at birth for the EU-15 in 2007 was 80.6 years, but we are simulating deaths for a population born between approximately 1900 and 2005, and thus a lower average is appropriate. It is also possible that those who succumb to chronic exposure are more likely to suffer impaired health conditions and thus may have lower life expectancy than the average population. If that is the case, our chronic mortality valuation may be somewhat overestimated.

3. Economic/demographic inputs and epidemiological parameters

3.1. Economic and demographic data

EPPA-HE requires historical information on market transactions, resource/income distribution, and demographic growth as key inputs. It solves for 5-year time intervals starting in 1970. We scale the GDP from the original GTAP data to 1970 levels and benchmark labor productivity growth to replicate actual GDP growth in Europe for the period 1970–2005 based on World Bank statistics (World Bank, 2009).

We construct the model's basic demographic inputs such as age cohort-specific population/mortality and urbanization rates at the EUR level (1970–2005) from time series estimates of national population, published by the United Nations Statistical Division (UN, 1999, 2008). Overall and cohort-specific cardio-pulmonary mortality rates are computed from the World Health Organization (WHO) database (WHO, 2009). Information on cardio-pulmonary mortality is used to modify the original *E–R* function for chronic mortality (0.25% mortality rate increase per unit PM_{10} concentration measured in $\mu g/m^3$) into age-conditioned forms. Section 3.2 provides further details on this conversion process.

3.2. Health endpoints and exposure–response functions

Epidemiological literature has extensively documented the link between major air pollutants and a variety of health outcomes (e.g., Anderson et al., 2004; Aunan and Pan, 2004; Dockery et al., 1993; Hiltermann et al., 1998; Hurley et al., 2005; Künzli et al., 2000; Ostro and Rothschild, 1989; Pope et al., 1995, 2004; Pope et al., 2002; Samet et al., 2000; Venners et al., 2003; Zhang et al., 2002). The ExternE project (Holland et al., 1999), initiated by the European Commission, synthesizes existing epidemiological studies, and provides a comprehensive list of *E–R* functions. We use these *E–R* functions from the ExternE study and their updated numbers for ozone and particulate matter (PM) reported in Bickel and Friedrich (2005) (Table 1). There are other possible relationships between air pollution and human health. For example, studies have shown that exposure to air pollution reduces semen quality (Robins et al., 1999; Seleven et al., 2000; Rubes et al., 2005), which could then affect fertility. We have not included other *E–R* relations than those listed in the table because the literature is relatively sparse and incomplete.

Many recent studies focus on $PM_{2.5}$ rather than PM_{10}, and some recent evidence suggests that health effects are related to the specific constituents in particulate matter. Unfortunately, widely measured data are only available for PM_{10}. Bickel and Friedrich (2005) scale epidemiological studies conducted using $PM_{2.5}$ to a PM_{10} *E–R* relationship based on an assumed fraction of $PM_{2.5}$ in PM_{10},[5] and incorporate more recent evidence relating health effects to $PM_{2.5}$. Given that health effects may be related to specific constituents of PM_{10} that vary by region or city and that the ratio of $PM_{2.5}$ in PM_{10} is likely to vary, ideally epidemiological relationships would be region- or city-specific or relate health outcomes to specific constituents of PM. Bickel and Friedrich (2005) draw *E–R* functions from a meta-analysis of the literature, and so they are not region-specific. The lower and upper bound estimates reflect error bars on epidemiological studies and

[5] Bickel and Friedrich (2005) used a factor of 0.6 to convert PM_{10} to $PM_{2.5}$.

Table 1
Exposure–response functions.

Receptor	Impact category	Pollutant	ExternE (1999)[a]			ExternE (2005)[b]			Notes
			E-R fct	CI (95%)		E-R fct	CI (95%)		
				Low	High		Low	High	
Entire population	Respiratory hospital admissions	PM_{10}	2.07E−06	3.58E−07	3.78E−06	7.03E−06	3.83E−06	1.03E−05	
		O_3	3.54E−06	6.12E−07	6.47E−06	Use ExternE (1999) numbers, except for elderly population			
	Cerebrovascular hospital admissions	PM_{10}	5.04E−06	3.88E−07	9.69E−06	5.04E−06	3.88E−07	9.69E−06	
	Cardiovascular hospital admissions	PM_{10}	n/a			4.34E−06	2.17E−06	6.51E−06	
	Respiratory symptoms days	O_3	3.30E−02	5.71E−03	6.03E−02	Use ExternE (1999) numbers			
	Asthma attacks	O_3	4.29E−03	3.30E−04	8.25E−03	Use ExternE (1999) numbers			
	Acute mortality	O_3	0.06%	0.00%	0.12%	0.03%	0.01%	0.04%	
		PM_{10}	0.04%	0.00%	0.08%	0.06%	0.04%	0.08%	
	Chronic mortality[c]	PM_{10}	0.25%	0.02%	0.48%	Use ExternE (1999) numbers			
Children	Chronic bronchitis	PM_{10}	1.61E−03	1.24E−04	3.10E−03	Use ExternE (1999) numbers			
	Chronic cough	PM_{10}	2.07E−03	1.59E−04	3.98E−03	Use ExternE (1999) numbers			
	Respiratory symptoms days	PM_{10}	n/a			1.86E−01	9.20E−02	2.77E−01	
	Bronchodilator usage	PM_{10}	7.80E−02	6.00E−03	1.50E−01	1.80E−02	−6.90E−02	1.06E−01	Defined on children aged 5–14 years meeting the PEACE study criteria (around 15% of children in Northern and Eastern Europe and 25% in Western Europe)
	Cough	PM_{10}	1.33E−01	2.30E−02	2.43E−01	n/a			
		O_3	n/a			9.30E−02	−1.90E−02	2.22E−01	ER functions on cough for ozone are defined on general population of ages 5–14
	Lower respiratory symptoms (wheeze)	PM_{10}	1.03E−01	1.78E−02	1.88E−01	1.86E−01	9.20E−02	2.77E−01	ExternE (2005) LRS values for PM include impacts on cough
		O_3	n/a			1.60E−02	−4.30E−02	8.10E−02	LRS ER functions for ozone, which do not take into account cough, are defined on general population of ages 5–14
Adults	Restricted activity day	PM_{10}	2.50E−02	1.92E−03	4.81E−02	5.41E−02	4.75E−02	6.08E−02	Restricted activity days include both minor restrcted days and work loss days
	Minor restricted activity day	O_3	9.76E−03	7.51E−04	1.88E−02	1.15E−02	4.40E−03	1.86E−02	Part of restricted activity days
		PM_{10}	4.90E−05	3.77E−06	9.42E−05	3.46E−02	2.81E−02	4.12E−02	
	Work loss day	PM_{10}	n/a			1.24E−02	1.06E−02	1.42E−02	Part of restricted activity days
	Respiratory symptoms days	PM_{10}	n/a			1.30E−01	1.50E−02	2.43E−01	Defined only on adults population with chronic respiritory symptoms (around 30% of adult population)
	Chronic bronchitis	PM_{10}	4.90E−05	8.48E−06	8.95E−05	2.65E−05	−1.90E−06	5.41E−05	
	Bronchodilator usage	PM_{10}	1.63E−01	1.25E−02	3.13E−01	9.12E−02	−9.12E−02	2.77E−01	Defined on population of 20+ with well-established asthma (around 4.5% of total adult population)
		O_3	n/a			7.30E−02	−2.55E−02	1.57E−01	
	Cough	PM_{10}	1.68E−01	2.91E−02	3.07E−01	n/a			
	Lower respiratory symptoms (wheeze)	PM_{10}	6.10E−02	1.06E−02	1.11E−01	1.30E−01	1.50E−02	2.43E−01	LRS ER functions for PM are defined on adult population with chronic respiratory symptoms (around 30% of total adult population); ExternE (2005) LRS values for PM include impacts on cough
Elderly 65+	Respiratory hospital admissions	O_3	n/a			1.25E−05	−5.00E−06	3.00E−05	
	Congestive heart failure	PM_{10}	1.85E−05	1.42E−06	3.56E−05	Use ExternE (1999) numbers			

Note: *E–R* functions for acute and chronic mortality have the unit of [%Δannual mortality rate/μg/m^3]. The rest *E–R* functions are measured in [cases/(yr-person-μg/m^3)].

[a] Computed from Holland et al. (1999).
[b] Computed from Bickel and Friedrich (2005).
[c] Adapted from Pope et al. (2002).

Table 2
Valuation of health-end points.
Source: Adapted from Bickel and Friedrich (2005, p. 156).

Outcome	*Unit*	*Cost (year 2000 Euro)*
Hospital admission	Per admission	2000
Emergency room visits for respiratory illness	Per visit	670
General practitioner visits		
Asthma	Per consultation	53
Lower respiratory symptoms	Per consultation	75
Respiratory symptoms in asthmatics		
Adults	Per event	130
Children	Per event	280
Respiratory medication use—adults and children	Per day	1
Restricted activity day	Per day	130
Cough day	Per day	38
Symptom day	Per day	38
Work loss day	Per day	82
Minor restricted activity day	per day	38
Chronic bronchitis	Per case	190,000

Note: Total costs—which include (i) resource costs such as cost of a hospital stay, (ii) opportunity costs due to lost work time, and (iii) disutility such as reduced enjoyment of leisure, discomfort, and inconvenience—were derived through a combination of market data (e.g., cost of hospital stay, wage rates) and stated preference surveys.

differences in *E–R* estimates for different regions. We show the sensitivity of the results to these error ranges in Section 5.3.

In addition to the *E–R* functions, we use the ExternE estimates of the value of health endpoints (Table 2). A benefit of the ExternE approach is that it combines market data on medical expenses and wages with survey data on stated preferences. For example, the 2000 Euro estimates for a hospital admission includes the costs of a hospital admission, loss of wages, and "disutility" associated with the illness, the latter estimated through a stated preference survey. Contingent valuation methods, especially the stated preference survey approach, are subject to a number of uncertainties. The Bickel and Friedrich (2005) estimates were subject to careful scrutiny, and were based on the questionnaire and sample design of Krupnick et al. (2002). Their computer-administered questionnaire included, for example, repeats of similar questions and audio and visual aides to improve consistency and accuracy, and they took care to produce a sample representative of the entire population.

As mentioned in Section 3.1, we used data on cardio-pulmonary death rates, in combination with the estimated *E–R* function for premature deaths due to chronic exposure, to establish increases in the death rate for affected age cohorts. The general issue is that the *E–R* function for chronic exposure estimates an increase in the average death rate for the population over age 30, and its magnitude may vary by age cohort. As fatal diseases from chronic exposure need substantial time to develop, we expect a larger increase in the death rate for older cohorts than for younger cohorts. In addition, as deaths from chronic pollution exposure likely occur through diseases of the heart and lung, we develop an age-conditioned form of the *E–R* function for chronic exposure from the following equation:

$$\mathrm{ER}_n(\mathrm{cm}) = \mathrm{ER}_T(\mathrm{cm}) \cdot \frac{M_n(\mathrm{cpl})/M_n(\mathrm{all})}{M_T(\mathrm{cpl})/M_T(\mathrm{all})} \quad (2)$$

where subscripts n and T are indices referring to age cohort and the total population at the age of 30 or higher, and ER(cm), M(cpl), and M(all) are the percent increase in mortality per $\mu g/m^3$ due to chronic exposure to pollution, the mortality rate from cardiopulmonary and lung diseases (cpl), and the mortality rate from all causes, respectively. Eq. (2) simply weights the death rates from pollution exposure for each age cohort based on the relative importance of cardio-pulmonary deaths for different age cohorts. For more discussion see Matus et al. (2008). Table 3 shows these weights for Europe, using data from WHO (2009). As shown, the weighting factor is greater than 1 for 60–69 and 70–79 age groups, near 1 for 45–59 and 80+ age groups, and less than 1 for the 30–44 cohort. This calculation allows us to identify the age distribution of likely deaths due to chronic exposure and to calculate years of lost lives to simulate the ongoing effects on the economy.

Table 3
Conversion factors for the chronic mortality E-R function (from unconditioned to age-conditioned), EUR.
Source: Computed from WHO database.

Age cohort	30–44	45–59	60–69	70–79	80+
Weight	0.43	0.96	1.16	1.39	0.95

4. Air quality data

In this section, we focus on impacts from exposure to ozone (O_3) and particulate matter (PM_{10}). Ozone and particulate matter are considered as the pollutants with the most potential to affect human health (EEA, 2009a). Confirming this conclusion, the US study of Matus et al. (2008) found that among the five criteria air pollutants defined by the United States Environmental Protection Agency (ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, and particulate matter), over 95% of the health costs were attributable to exposure to ozone and particulate matter.

Our estimates of ground-level ozone data are based on model results from the European Monitoring and Evaluation Program (EMEP) database, co-maintained by the United Nations Economic Commission for Europe (UNECE) and the Co-operative program for monitoring and evaluation of long range transmission of air pollutants in Europe (EMEP and UNECE, 2006). EMEP ozone data are available between 1980 and 2004. Because we are interested in the cumulative effects of air pollution, we assume that concentrations in 1970 and 1975 were the same as those in 1980. We also use 2004 data for 2005. Among various ground level ozone measurements, provided by the EMEP database, we use annual means of 8-h daily maximum, for which *E–R* functions are defined.

For input into EPPA-HE, we compute a representative air quality number for the European region for each year and each pollutant. As the goal of our research is to estimate the impact of air pollution on human health, we use population weights to construct average concentrations for Europe. For this purpose we use a $1^\circ \times 1^\circ$ world population share grid data for 1990 (SEDAC, 2009) as a weight for ozone and PM concentrations for all years' air quality data. Lacking data on migration among grids, we assume that populations in a grid are exposed to the time profile of pollution in that grid over their whole life. Original EMEP grids, each of which is sized at 50 km × 50 km, are converted into $1^\circ \times 1^\circ$ to match those of the population data by using ArcGIS software and the inverted distance weighted (IDW) spatial interpolation technique (see Figs. 2 and 3).

We do not use the same data sources for PM, however, because EMEP's PM concentration estimates substantially underestimate actual PM levels for two reasons (EMEP, 2001). One reason is that the EMEP model is designed to estimate PM concentration solely from secondary inorganic aerosol (SIA) concentrations and

primary emissions of particles, while ignoring other key components such as resuspended anthropogenic and natural mineral dust, sea salt, and biogenic aerosols, which also substantially contribute to PM concentration. Second, the EMEP model built on underestimated SIA concentration inputs. Thus, we use two alternative data sources for PM: the AirBase database, maintained by the European Environment Agency (2009b), and the World Development Indicators (WDI) database, published by the World Bank (2009). The AirBase database provides historical concentration levels both of PM and of total suspended particulate (TSP). When PM_{10} data are not available, we convert TSP data into PM_{10} concentrations by applying a factor of 0.55, following Dockery and Pope (1994). While for at least some major monitoring stations the data extends back to 1976, data for some stations for some years are missing and the station coverage prior to the late 1990s is very sparse. To fill missing data, we first compute the average ratio of PM data from a set of monitoring stations which have data for two consecutive years, and then apply this factor to monitoring stations, which have data for either of the 2 years. We eliminate monitoring stations which have missing data or cannot be filled this way for two consecutive years. As data for later years are more complete, we carry out this procedure from recent years to early years. After completing this procedure, we convert AirBase data layers for each year into $1^\circ \times 1^\circ$ raster maps in a similar way as for the EMEP ozone data. In this case, 1970 and 1975 PM levels are assumed to be constant at the 1976 level (see Fig. 4).

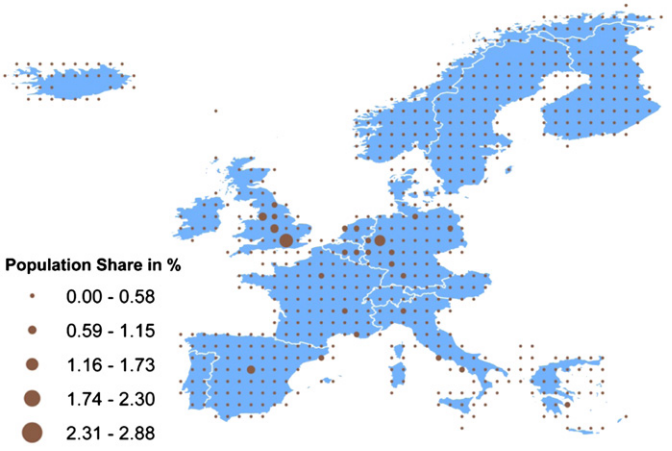

Fig. 2. Population share grid, EUR, 1990.

PM_{10} data from the WDI database are available between 1990 and 2005. As the database provides only nation-wide average concentration numbers, we calculate EUR-wide PM_{10} concentration numbers by using each country's population as weight. PM_{10} levels for 1985 and earlier are assumed to be constant at 1990 levels. See Fig. 5 for air quality numbers used here.

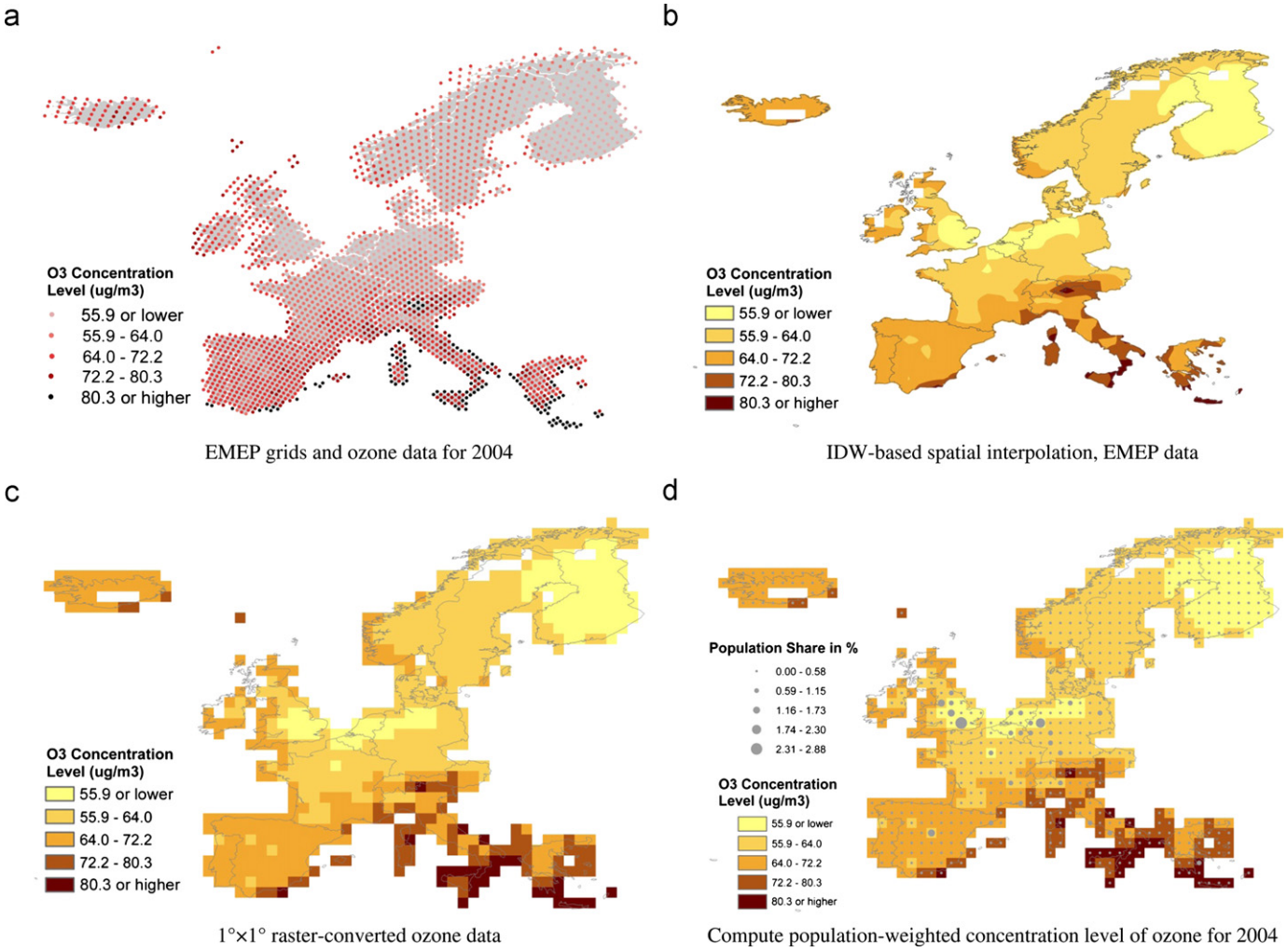

Fig. 3. Procedure of computing population-weighted concentration level of ozone, 2004.

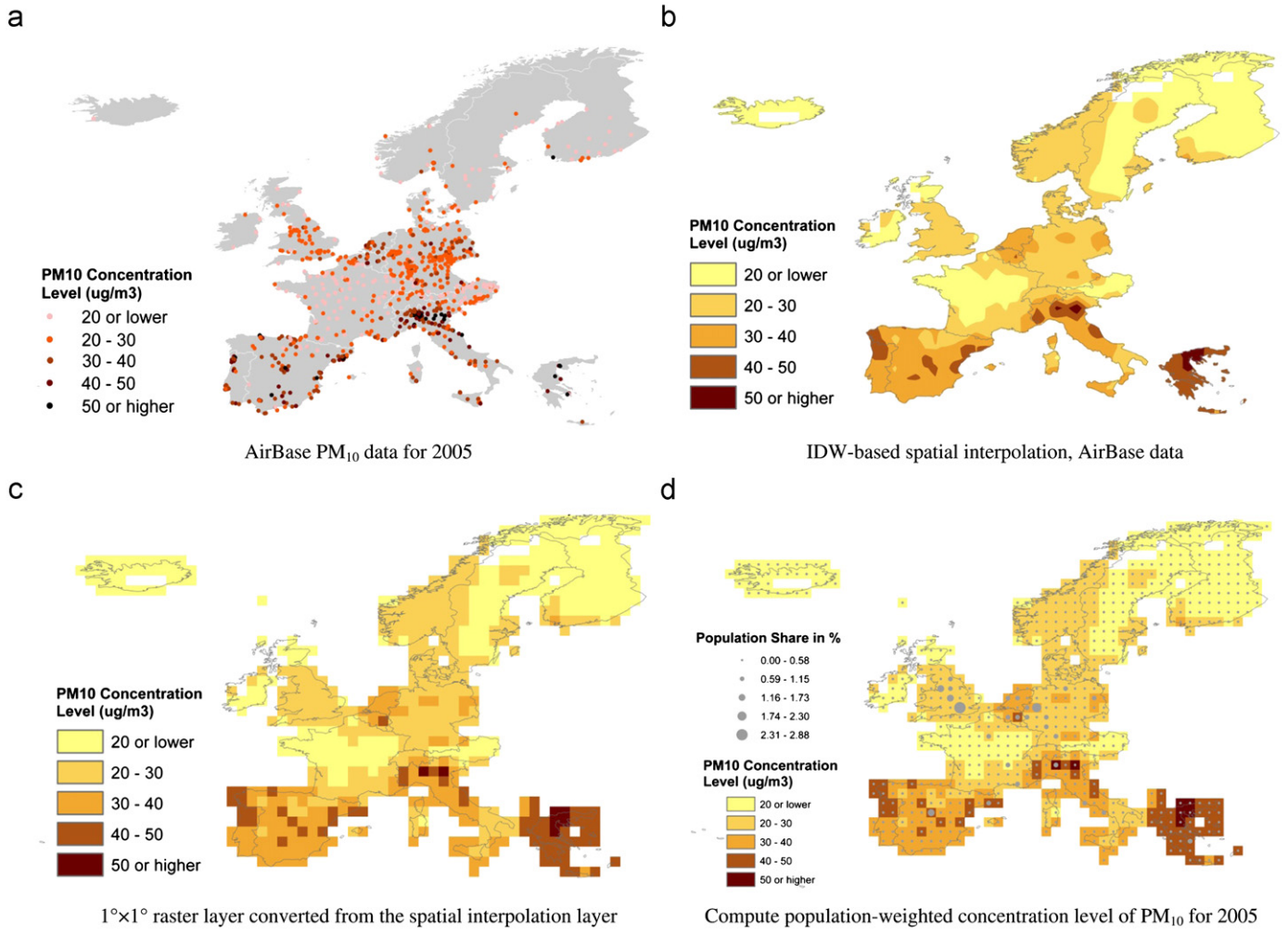

Fig. 4. Procedure of computing population-weighted concentration level of PM_{10}, 2005.

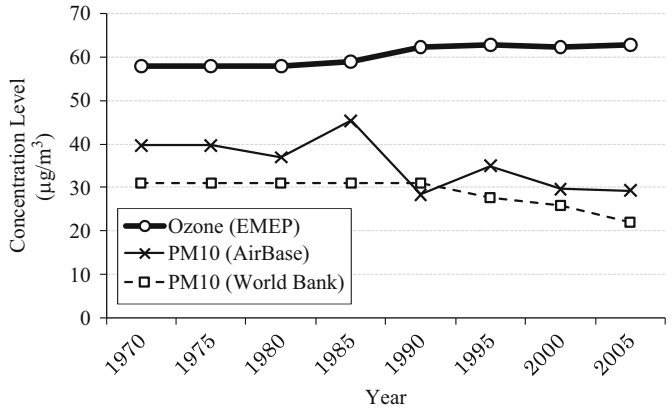

Fig. 5. Concentration levels of ozone and PM_{10}, EUR, 1970–2005. *Notes*: Here, the measurement standard for ozone is annual means of 8 h daily maximum, and that for PM_{10} is annual means of 24 h average.

To compare these two different historical PM concentration estimates, we set up two reference case scenarios. We use AirBase-estimated PM concentrations for Reference Case Scenario A, and WDI-based estimates for Reference Case Scenario B. All other inputs for the two reference scenarios except PM concentration are identical.

5. Results

We apply the model to two questions. The first question is what has been the economic burden of air pollution experienced from 1970 to 2005. The second question is what would be the benefits (i.e., avoided damages) from proposed air quality regulations. We compare our estimates for the latter to a 2005 CAFE study. To estimate the economic burden of pollution from 1970 to 2005, we need to simulate two scenarios. One scenario is the *Historical* scenario, in which air quality inputs are set at historical levels and GDP growth is benchmarked to observed levels for the 1970–2005 period. This reference scenario reflects the fact that these air pollution levels were observed, and observed economic results were already distorted by air pollution effects. To estimate the economic impact of these observed levels, a second *Green* scenario is simulated as a counterfactual simulation where concentrations of these pollutants are set at 20 $\mu g/m^3$ for ozone and 0.001 $\mu g/m^3$ for PM_{10}, which are levels that would be observed if there were no anthropogenic sources of pollutant emissions (Seinfeld and Pandis, 1998). Note that the *E–R* functions we use are linear and thus do not admit the existence of a threshold, below which damages do not occur, or other nonlinearities. If such thresholds exist at pollution levels beyond background levels, our method may overestimate the economic burden. The central results of our analysis are discussed in Section 5.1. We then decompose economic damages in 2005 among sources of damage from exposure in 2005 and sources of

Table 4
Consumption and welfare losses caused by air pollution (Reference Case A), EUR, 1975–2005.

Year	Consumption loss		Welfare loss	
	Billions of year 2000 Euro	% of Historical consumption level	Billions of year 2000 Euro	% of Historical welfare level
1975	169	4.7	293	3.3
1980	169	3.9	297	2.7
1985	175	3.7	260	2.2
1990	225	4.0	467	3.3
1995	219	3.6	374	2.4
2000	229	3.2	418	2.3
2005	217	2.8	354	1.8

Table 5
Consumption and welfare losses caused by air pollution (Reference Case B), EUR, 1975–2005.

Year	Consumption loss		Welfare loss	
	Billions of year 2000 Euro	% of historical consumption level	Billions of year 2000 Euro	% of historical welfare level
1975	169	4.7	292	3.2
1980	167	3.9	278	2.6
1985	180	3.8	300	2.5
1990	216	3.8	370	2.6
1995	210	3.4	358	2.3
2000	226	3.2	393	2.1
2005	217	2.8	373	1.9

economic damage from exposure prior to 2005 in Section 5.2. In Section 5.3, we consider sensitivity of these results to uncertainty in *E–R* estimates. The upper and lower limits of the estimates' 95% confidence interval may capture some of the uncertainty introduced by assuming linear *E–R* functions. In Section 5.4, we estimate benefits of new proposed pollution standards. These estimates are less dependent on linearity of the *E–R* functions because we are examining smaller changes in air quality.

5.1. Overview

We find that air pollution caused substantial socio-economic costs in the European region (Tables 4 and 5). First, we measure the pollution health cost in terms of consumption loss, which does not include leisure time value. In terms of consumption, we calculate that the European economy has lost annually 2.8% to 4.7% of historical consumption levels due to air pollution for the last three decades. With increasing concerns about air pollution and stricter air quality control, consumption-measured pollution–health cost shows a declining tendency, though with slight intra-period fluctuations. In absolute values, the region's consumption loss, which ranged between 169 billion Euro[6] and 229 billion Euro during the period 1975–2005, was estimated to reach its maximum of 229 billion Euro in 2000 (Reference Case A) or of 226 billion Euro in 2000 (Reference Case B). The simulation outcomes based on Reference Case B suggest that improving air quality in Europe led to lower consumption loss through the period of our analysis in terms not only of relative measure to historical consumption levels but also of absolute monetary units.

The loss of welfare, which we evaluate as a loss in the sum of consumption and leisure, shows a similarly declining tendency. This simulation outcome is not surprising, given the fact that the European region's air quality has been kept constant (in the case of ozone) or improved (PM), and the changes—whether positive or negative—in air quality are small relative to the region's economic growth. For the last three decades, the European region's annual welfare loss, caused by air pollution, ranged between 1.8% and 3.3% of the historical welfare level or between 260 billion Euro and 467 billion Euro, on the basis of Reference Case A. Welfare loss estimates based on Reference Case B were similarly between 278 billion Euro and 393 billion Euro and between 1.9% and 3.2% of the historical level.

[6] We measure Euro as year 2000 Euro unless specifically noted.

Table 6
Pollution-induced health outcomes by pollutant (Reference Case B), EUR, 2005 (Unit: thousands of cases).

	Ozone	PM_{10}
Respiratory hospital admission	242	70
Cerebrovascular hospital admission	n/a	50
Cardiovascular hospital admission	n/a	43
Respiratory symptom days	640,103	323,005
Acute mortality	56	58
Chronic bronchitis	n/a	176
Chronic cough (only for children)	n/a	4249
Cough and wheeze	35,163	532,759
Restricted activity day	177,083	448,065
Congestive heart failure	n/a	31
Asthma attacks	3329	n/a
Bronchodilator usage	53,021	41,515
Chronic mortality[a]		
Cases that occurred in 2005	n/a	124
Losses in 2005 from prior year cases[b]	n/a	1228

[a] Premature deaths due to chronic exposure to pollution.
[b] Those who would have been active in the given year without chronic exposure to pollution in earlier years.

5.2. Decomposition analysis

An important distinction of our approach is that it accumulates damages over time due to deaths that occurred in earlier years and due to economic losses that result in less savings and investment. To understand the importance of these cumulating effects we decomposed pollution-induced health costs for the Reference Case Scenario B to show how much of the estimated economic burden in 2005 is due to health effects that occurred in

Table 7
Decomposition of direct pollution health costs in 2005 (Reference Case B) (Unit: millions of year 2000 Euro).

Health outcome category	*Ozone*			*PM_{10}*		
	Medical expenses	*Wage loss*	*Leisure loss*	*Medical expenses*	*Wage loss*	*Leisure loss*
Non-fatal health outcomes	13,384	20	19,172	106,748	10,429	30,806
Acute mortality	n/a	436	1452	n/a	447	1490
Chronic mortality (year 2005 only)	n/a	n/a	n/a	n/a	2666	13,459
(Except year 2005)	n/a	n/a	n/a	n/a	20,219	110,197
Sub-total	13,384	456	20,624	106,748	33,761	155,952
Sub-total by pollutant		34,463 (10%)			296,461 (90%)	
Total direct costs			330,925 (100%)			

Table 8
Decomposition of welfare loss in 2005 (Reference Case B).

	Monetary value (billions of 2000 Euro)	*Share of total welfare loss (%)*
Total welfare loss	374	100
Direct loss due to chronic exposure	146	39
Cases that occurred in 2005	16	4
Losses in 2005 from prior year cases	130	35
Direct loss due to other health outcomes	184	49
Broader economic losses	44	12

2005 and how much is carry-over from health effects that occurred in earlier years. We calculate (i) direct costs related to premature deaths in 2005, (ii) direct costs in 2005 related to premature deaths in earlier years, (iii) direct costs from acute exposure in 2005, and (iv) broader economic losses. The latter are calculated as the residual between the total welfare loss and the sum of the three direct losses. Broader economic losses reflect the difference between direct costs and welfare measurement of damages in 2005 and the cumulative effects through savings and investment of earlier year losses. Here, the direct costs refer to the sum of medical expenses, wage loss caused by illness or premature deaths, and leisure loss caused by illness or premature deaths.

Table 6 shows the increase in the number of cases by health outcome induced by ozone and PM concentrations that exceed the background levels. Table 7 displays the direct pollution health costs of these cases for 2005 applying the estimated value of health outcomes from Table 2 and aggregating the results into four categories. We estimate that direct pollution health costs for 2005, were 331 billion Euro. Around 60% of ozone-related costs and 53% of PM_{10}-induced costs are from leisure loss. PM_{10} contributes nine times as much to direct pollution health costs as ozone: 90% of the 2005 total direct pollution health costs were caused by PM_{10}. Nearly half the PM-related direct costs are from premature deaths due to chronic exposure, and 89% of the costs related to chronic PM exposure are due to premature deaths that occurred in earlier years.

In Table 8, we show direct costs and broader economic losses. The latter was estimated as a residual after subtracting the direct costs from the total welfare loss we estimated by simulating our economic model. As shown, direct costs of health effects that occurred in 2005 account for just over half (53%) of the economic losses in 2005. About 35% of the economic losses are from prior year mortality from chronic exposure and about 12% of the 2005 total welfare loss is beyond the direct pollution effects that occurred in 2005. Thus, nearly half the total welfare loss we estimate for 2005 was the cumulative effect of pollution over the past 3 decades.

5.3. Sensitivity analysis

Given that *E–R* relationships can vary by time and place, even for the same pollutant and health outcome, a substantial degree of uncertainty may come from the *E–R* functions. In this section, we conduct two sets of sensitivity analysis on *E–R* functions to evaluate the robustness of the results presented above. The first analysis compares reference simulation outcomes with those using upper and lower bound values of *E–R* functions, acquired from the 95% confidence interval. For the second analysis, we run the model by replacing reference *E–R* functions by *E–R* functions from the 1998 ExternE study. We compared both sets of sensitivity analysis simulation results with those from Reference Case Scenario B, which employs WDI-based estimates for historical PM_{10} concentration levels.

When we used lower bound values of *E–R* functions, EPPA-HE not surprisingly produced lower estimates of air pollution-driven health costs than the reference case (Table 9). Compared with estimates in Table 5, both consumption and welfare loss fell by more than half. However, lower bound *E–R* values also produce non-trivial estimates for consumption and welfare loss from air pollution, which reach 1.4–2.1% of historical levels. In contrast, upper bound *E–R* values raised pollution-caused health damage estimates to 4.9–7.4% of consumption or 3.2–5.0% of welfare with a declining trend over time (Table 10). From this result, we can conclude that although uncertainty involved in *E–R* functions themselves widens the range of our pollution health cost estimates substantially, it does not undermine our general conclusions that substantial socio-economic burdens result from air pollution, and that relative pollution health costs have declined over time.

Table 11 summarizes simulation outcomes based on the 1998 ExternE study-proposed *E–R* functions instead of the updated values from the 2005 ExternE study. When 1998 *E–R* functions were used, pollution health cost estimates were reduced to 1.3–2.6% of consumption and 0.8–1.7% of welfare. This outcome, though lower in magnitude, does not contradict our general

Table 9
Sensitivity analysis 1-1: lower bound values (95% CI) of *E–R* functions.

Year	Consumption loss		Welfare loss	
	Billions of year 2000 Euro	*% of Historical consumption level*	*Billions of year 2000 Euro*	*% of Historical welfare level*
1975	76	2.1	143	1.6
1980	78	1.8	144	1.3
1985	85	1.8	158	1.3
1990	101	1.8	192	1.3
1995	101	1.7	192	1.2
2000	110	1.6	215	1.2
2005	107	1.4	209	1.1

Table 10
Sensitivity analysis 1-2: upper bound values (95% CI) of *E–R* functions.

Year	Consumption loss		Welfare loss	
	Billions of year 2000 Euro	*% of Historical consumption level*	*Billions of year 2000 Euro*	*% of Historical welfare level*
1975	269	7.4	452	5.0
1980	262	6.0	420	3.9
1985	281	6.0	451	3.8
1990	338	6.0	557	3.9
1995	328	5.3	533	3.4
2000	352	4.9	581	3.2
2005	335	4.3	550	2.8

Table 11
Sensitivity analysis 2: old *E–R* values from the 1998 ExternE study.

Year	Consumption loss		Welfare loss	
	Billions of year 2000 Euro	*% of Historical consumption level*	*Billions of year 2000 Euro*	*% of Historical welfare level*
1975	93	2.6	151	1.7
1980	93	2.2	146	1.3
1985	104	2.2	169	1.4
1990	126	2.3	213	1.5
1995	117	1.9	190	1.2
2000	117	1.7	186	1.0
2005	102	1.3	154	0.8

Table 12
Emission scenarios for the CAFE study, EU-25. (Unit: kt).
Source: Adopted and computed from Amann et al. (2005: 20–24) and Holland et al. (2005, p. 17).

	Year 2000	*Year 2020*			
		Baseline	*Scenario A*	*Scenario B*	*Scenario C*
SO_2	8735	2806	1814	1700	1594
NO_x	11,581	5886	4560	4136	3923
VOC	10,661	5907	5232	4867	4743
NH_3	3824	3683	n/a	n/a	n/a
Primary PM	37	27	23	22	22

conclusion that air pollution has generated substantial socio-economic costs to the European economy.

5.4. Comparison with the CAFE study

There are several studies that attempt to estimate health impacts of air pollution in Europe (e.g., Krupnick et al., 1996; Olsthoorn et al., 1999; Holland et al., 2005). It is difficult, however, to compare their estimates directly with ours due to different pollutants of interest, target years, target air quality, and geographical boundaries. Nonetheless, we concluded that the 2005 Clean Air for Europe (CAFE) study of Holland et al. took the most analogous approach with ours in estimating pollution health costs, and thus we present here a comparison to their results. For comparison, we modified EPPA-HE to simulate economic and health outcomes up to year 2020.

To conduct this analysis, we use the same emission scenarios used by Holland et al. (2005) and summarized in Table 12. Their 2020 Baseline scenario is consistent with that of the Regional Air pollution Information and Simulation (RAINS) model, which was also employed for other CAFE studies. EU-25's emission levels for policy alternative scenarios are set at around 11–43%-reduced levels from the Baseline emission levels. Among them, Policy Scenario C has the most ambitious emission reduction target, while Policy Scenario A has the least ambitious target.

As explained in previous sections, EPPA-HE needs concentration data of ozone and PM for the computation of health end point cases. Thus, emission-based scenarios shown in Table 12 should be converted into concentration-based ones. Holland et al. (2005) clarify that their PM and ozone concentration data are taken from

Table 13
Air quality inputs, EUR, 2020 (Unit: μg/m^3).

Ozone				*PM_{10}*			
Reference	*Policy A*	*Policy B*	*Policy C*	*Reference*	*Policy A*	*Policy B*	*Policy C*
52.5	48.7	47.5	46.7	9.0	7.4	7.0	6.8

Table 14
Net welfare gains from CAFE-proposed emission control, year 2020 only (Unit: billions of year 2000 Euro).

Holland et al. (2005)			*EPPA-HE*		
Policy A	*Policy B*	*Policy C*	*Policy A*	*Policy B*	*Policy C*
37	45	49	34	43	48

the RAINS model and the EMEP model, respectively. We obtained country-specific PM and ozone concentration data that were used for their CAFE reference and three policy scenarios (C. Heyes, pers. comm.). For PM_{10}, we computed population-weighted average for EPPA region EUR directly from the provided numbers. However, an additional step was necessary for the case of ozone, as the provided data was measured as the sum of excess of daily maximum 8 h means over the cut-off of 35 ppb (SOMO35). To approximate year 2020 ozone concentration numbers without thresholds, we first computed the ratio between year 2000 and year 2020 SOMO35 numbers, and then applied the ratio to year 2000 ozone concentration numbers without thresholds.[7] Annual means of ozone concentration for a large region are highly correlated (r=0.99) with SOMO35 (Dentener et al., 2006). Table 13 displays PM and ozone concentration numbers for 2020 by scenario. In addition, EPPA-HE's future projection assumes annual GDP growth rates of 1.8% for 2006–2015 and of 2.0% for 2016–2020 (Paltsev et al., 2005).

We compare our results to Holland et al. (2005). They provide two sets of estimates for net welfare benefits of CAFE-proposed emission regulation scenarios. One is a low set of estimates based on the value of a life year (VOLY) of 52,000 Euro, and the other is a high set of estimates based on the VOLY of 120,000 Euro. As EPPA-HE uses ExternE-proposed health end point valuation tables, which are based on the VOLY of 50,000 Euro, we compare our estimates with their low estimates. As shown in Table 14, we estimate that CAFE-proposed emission regulation measures will bring a welfare gain of 34 billion to 48 billion Euro. Our estimates are very close to those of Holland et al. which are between 37 billion and 49 billion Euro. Perhaps, part of the estimates difference is from dissimilar geographical boundaries of interest for each study as well as from difference in methodology. While EPPA region EUR includes EU-15 member states and three non-EU high-income countries (Switzerland, Norway, and Iceland), the CAFE study embraces the whole EU-25 member countries. As of 2000, the population of the former region was no more than 86% of EU-25's total.

[7] This calculation procedure can be expressed as the following equation, where Ozone_t indicates annual means of 8 h daily maximum (without threshold) in time t:

$$\text{Ozone}_{t+1} = \frac{\text{SOMO35}_{t+1}}{\text{SOMO35}_t} \times \text{Ozone}_t$$

6. Caveats and conclusions

Our results show that air pollution has likely generated a substantial economic burden for the 18-nation European region that was the focus of our analysis. Although air quality in Europe has been controlled, our central estimate shows that the region still lost 3% of consumption (or 2% of welfare) due to air pollution in 2005, even when only human health-related aspects and two key air pollutants (ozone and PM_{10}) were considered. This suggests that policy measures formulated to improve air quality may benefit society. We have not estimated the cost of emissions control, and so any benefit must be compared to control costs.

Our decomposition analysis shows that roughly half the 2005 welfare loss caused by air pollution in Europe was from cumulative sources. This suggests that a static approach may substantially underestimate pollution damages. In this regard, we believe that our dynamic modeling of air pollution health damages provides a more accurate and complete accounting of the economic effects of air pollution exposure as they occur over time with important effects accumulating as time passes.

As noted, a complete analysis would balance costs and benefits. An appropriate cost–benefit analysis should look ahead to capture the full stream of benefits over time, and conduct a similar dynamic cost analysis. In particular, a more complete cost–benefit analysis would consider mitigation costs to conduct a full dynamic cost–benefit study. In that regard, it is unlikely that cost–benefit analysis would support achieving background pollution levels as that would likely require extreme and costly measures to reduce all pollutants including emissions outside the region that are transported into the region. Our assumed linear E–R functions also do not allow for thresholds. Our sensitivity analysis that takes into account error in the E–R estimates suggests error bars of $\pm$50%.

Realistic proposals for pollution control call for more limited reductions in pollution levels. We use the model to simulate potential health benefits from actual air quality regulation scenarios examined in the CAFE study by Holland et al. (2005). Our results are very close to those of the 2005 CAFE study. A Europe-wide reduction from the 2020 baseline scenario of 10–40% of key air pollutants such as SO_2, NO_x, VOC, NH_3, and PM is estimated to bring a net welfare gain of 34 billion to 48 billion Euro for the year 2020 alone.

The work reported here is a first step toward a more complete dynamic cost–benefit analysis. The analysis suggests important dynamic effects that stem from deaths due to chronic exposure and economic costs generated by lower savings and investment in early years. The approach utilizes conventional epidemiological and valuation methods but embeds them in an economy-wide dynamic analysis. Future work would include the cost of control measures in the model.

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