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A methodology to estimate changes in statistical life expectancy due to the control of particulate matter air pollution

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Abstract

Studies in the United States have shown that those living in less polluted cities live longer than those living in more polluted cities. After adjustments for other factors, an association remained between ambient concentrations of fine particles and shorter life expectancy. This paper presents a methodology to apply the findings of these epidemiological studies to scenarios to control fine particulate matter in Europe and to estimate the resulting losses in statistical life expectancy that can be attributed to particulate matter pollution. Calculations are carried out for all of Europe with a 50*50 km resolution, distinguishing higher PM2.5 levels in urban areas. The methodology uses population statistics and projections from the United Nations, and applies changes in mortality risk identified by the epidemiological studies to the life tables for the individual countries. The preliminary implementation suggests that, for constant 1990 pollution levels, statistical life expectancy is reduced by approximately 500 days (95 percent confidence interval ranging from 168 – 888 days). By 2010, the control measures presently decided for emissions of primary particles and the precursors of secondary aerosols are expected to reduce these losses to about 280 days (94 - 497), while the theoretical maximum technically feasible emission reductions could bring reduced life expectancy below 200 (65 – 344) days. While the quantifications in this study must be considered as preliminary, the methodology will allow the introduction of health impacts from fine particulate matter into a multi-pollutant/multi-effect framework so that control measures can be explored taking full account of their ancillary benefits for acidification, eutrophication and ground-level ozone.

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1 Introduction

Over the past decade epidemiologic studies in Europe and worldwide have measured increases in mortality and morbidity associated with air pollution. Studies in the United States have shown that those living in less polluted cities live longer than those living in more polluted cities (Dockery *et al.*, 1993; Pope *et al.*, 1995). After adjustments for other factors, an association remained between ambient concentrations of fine particles and shorter life expectancy. These findings were confirmed by a reanalysis of the original studies published by the Health Effects Institute (Krewski *et al.*, 2000) and by a recently published large-scale assessment of mortality based on data collected by the American Cancer Society (Pope *et al.*, 2002).

With accumulating evidence about health effects of air pollution, interest is growing to use data from these studies to inform environmental policies. The World Health Organization (WHO) has produced a guideline document ("*Evaluation and use of epidemiologic evidence for environmental health risk assessment"),* providing a general methodology for the use of epidemiological studies for health impact assessment (WHO, 2000). In 2001, WHO convened a working group to examine several of the aspects introduced in this report as they apply specifically to air pollution health impact assessment (WHO, 2001).

Following these guidelines from WHO this report develops a methodology for estimating losses in life expectancy due to air pollution and presents an initial implementation assessing the implications of present and future policies in Europe to control exposure to particulate matter. At this point in time, the paper focuses on the methodological framework in order to demonstrate how information relevant for health impact assessment can be put together in a consistent and meaningful way. It integrates population data, findings from epidemiological studies, information about the formation and dispersion of fine particles in the atmosphere, estimates of present and future levels of emissions of fine particles and their precursors. Awaiting further refinements in the scientific disciplines, the quantitative implementation should be considered as preliminary and needs to be revised as soon as more substantiated scientific information becomes available.

This assessment of health impacts of air pollution provides an extension of the Regional Air Pollution Information and Simulation (RAINS) model developed at the International Institute for Applied Systems Analysis (IIASA), Laxenburg, Austria. To the extent possible, the methodology relies on calculations and data already implemented in the RAINS model for the assessment of emission control strategies focusing on acidification, eutrophication and ground-level ozone (Schöpp *et al.*, 1999) and on the extension to particulate matter, which is presently under development (Amann *et al.*, 2001).

This report introduces the conceptual framework for the quantification of health impacts of air pollution and provides a detailed mathematical description of the methodology (Section 2). Section 3 presents the results of a first pilot implementation based on presently available data. Uncertainties are explored in Section 4, while Section 5 discusses the results and their main uncertainties, outlines priorities for further research and draws conclusions.

2 The conceptual framework

This study uses the following basic steps to estimate health impacts of air pollution control scenarios:

1. Obtain, for all European countries, information (a) on current mortality rates from UN population statistics and (b) on future baseline mortality rates that are implied by the UN world population projections.
2. Estimate exposure of the European population to particulate matter pollution (a) for 1990, (b) for 2010 assuming implementation of presently decided emission controls, and (c) for the lowest PM levels that could hypothetically be achieved by full application of present-day technical emission controls. This requires (i) spatially explicit information about population densities, and (ii) spatially explicit information of PM levels resulting from the three emission scenarios.
3. Using associations between particulate matter pollution and mortality found by epidemiological studies, determine the modification of mortality rates due to PM pollution.
4. Calculate changes in life expectancy (compared to the baseline UN scenario) resulting from the modified exposures to PM pollution of the three emission scenarios.
5. Examine how sensitive these estimates are to changes in the underlying assumptions.

With this approach, the study combines information about

- results from epidemiological studies that quantify mortality impacts of exposure to air pollution,
- demographic structures in the various European countries and their expected development over time,
- geographically explicit estimates of exposure to air pollution, based on gridded population data and concentration fields of fine particulate matter, distinguishing urban and rural areas,
- the formation and dispersion of aerosols (fine particles) in the atmosphere from
- primary emissions of fine particles as well as the precursor emissions (sulphur dioxide, nitrogen oxides, ammonia, volatile organic compounds) leading to secondary aerosols,
- the situation estimated for 1990, the predicted conditions in the year 2010 if presently decided emission control strategies were fully implemented and the maximum technically feasible emission controls that could be achieved in the year 2010, taking into account the presently envisaged economic development in the various European countries.

2.1 Population data

2.1.1 Population statistics and projections

For all European countries considered in the analysis, demographic information on total population, cohort size and expected deaths was extracted from the recent (medium fertility) world population projections of the United Nations (UN, 2000). These projections provide data in five-years intervals up to 2050. Population data for 2000 and 2010 are provided in Table 2.

2.1.2 Life tables

The probability that an individual will die at a certain age depends both on him/her not dying before that age, and on a probability (or risk) that in adults increases with age. Age-related differences can be described in form of a "life table" such as Table 1. For each country, the age-specific baseline, non-accidental mortalities contained in such life tables are calculated from population statistics as the quotient of deaths to population for five-year time-periods for 2000-2050. These calculations were carried out on the national level, using statistics and projections of cohort sizes and death numbers provided by the UN Population Division (2000).

For estimating losses in life expectancy, all cohorts at least aged 30 years in 2010 are followed over their whole lifetime, i.e., from 2010 to 2075. The mortality rates projected by the UN scenario for 2050 were assumed prevail constant between 2050 and 2075. Younger cohorts are not followed, since they were not addressed in the supporting epidemiological studies. Further analysis is necessary to explore the impacts of including younger cohorts, especially since recent studies indicate a correlation between air pollution and infant mortality (see also Englert, 1999).

2.1.3 Gridded population data

For this study, estimates of losses in life expectancy are carried out for all of Europe with a spatial resolution of 50*50 km, corresponding to the grid system defined by the European Monitoring and Evaluation (EMEP) Programme (www.emep.int). While the dispersion model used for this analysis calculates ambient concentrations of PM at this resolution, the present spatial distribution of population in Europe had to be compiled from a variety of sources.

For the EU countries, basic population data on NUTS3 level are taken from the EUROSTAT statistics for the year 1997 (EUROSTAT, 2000). Administrative borders were updated with information from the Austrian Statistical Office (Statistik Österreich, 1999). These population data were spatially allocated to the administrative boundaries provided by the Environmental Systems Research Institute (ESRI, 2000a). For a number of countries (Austria, Belgium, Denmark, Finland, Ireland, Germany, Luxemburg, Netherlands, Portugal, Sweden and the UK) recent information on the NUTS3 boundaries was from NUTS databases (Statistik Österreich, 1999).

For Poland, information on population distribution was acquired from national sources (http://www.stat.gov.pl/english/index.htm), and data for Russia were taken from the UNEP-GRID database http://www.grid.unep.ch). Information for other countries was extracted from the ESRI demographic database (ESRI, 2000b).

While this information originates from different points in time between 1996 and 1999, for 2010 the assumption was made that, within each country, the spatial distribution of population will remain unchanged.

In this way, for each 50*50 km grid cell total population was derived. The age group distribution as well as the life tables for the population in a grid cell were deduced from the UN national data set.

Table 1: Example life table for Albania, giving the mortality rates for different cohorts for the time span 2010-2075

Country	Age	Y2010	Y2015	Y2020	Y2025	Y2030	Y2035	Y2040	Y2045	Y2050	Y2055	Y2060	Y2065	Y2070	Y2075
Albania	30	0.00358	0.00316	0.00274	0.00249	0.00225	0.00220	0.00203	0.00186	0.00180	0.00180	0.00180	0.00180	0.00180	0.00180
Albania	35	0.00444	0.00398	0.00358	0.00309	0.00283	0.00281	0.00278	0.00259	0.00238	0.00238	0.00238	0.00238	0.00238	0.00238
Albania	40	0.00627	0.00567	0.00520	0.00472	0.00413	0.00411	0.00413	0.00411	0.00382	0.00382	0.00382	0.00382	0.00382	0.00382
Albania	45	0.00895	0.00915	0.00843	0.00781	0.00719	0.00665	0.00668	0.00671	0.00666	0.00666	0.00666	0.00666	0.00666	0.00666
Albania	50	0.01436	0.01440	0.01490	0.01381	0.01293	0.01228	0.01146	0.01147	0.01146	0.01146	0.01146	0.01146	0.01146	0.01146
Albania	55	0.02593	0.02461	0.02480	0.02576	0.02397	0.02265	0.02174	0.02019	0.02011	0.02011	0.02011	0.02011	0.02011	0.02011
Albania	60	0.05729	0.04593	0.04357	0.04385	0.04552	0.04215	0.04037	0.03861	0.03574	0.03574	0.03574	0.03574	0.03574	0.03574
Albania	65	0.09746	0.10341	0.08260	0.07821	0.07857	0.08060	0.07575	0.07230	0.06893	0.06893	0.06893	0.06893	0.06893	0.06893
Albania	70	0.18224	0.17394	0.18437	0.14692	0.13874	0.13698	0.14323	0.13430	0.12796	0.12796	0.12796	0.12796	0.12796	0.12796
Albania	75	0.32178	0.31382	0.29759	0.31517	0.25116	0.23287	0.23480	0.24475	0.22923	0.22923	0.22923	0.22923	0.22923	0.22923
Albania	80	0.58120	0.52568	0.50917	0.47864	0.50486	0.39713	0.37838	0.38054	0.39524	0.39524	0.39524	0.39524	0.39524	0.39524
Albania	85	0.84345	0.86702	0.79404	0.76041	0.70321	0.72985	0.60221	0.57657	0.58031	0.58031	0.58031	0.58031	0.58031	0.58031
Albania	90	1.00000	1.00000	1.00000	1.00000	1.00000	0.98687	1.00000	0.88015	0.84620	0.84620	0.84620	0.84620	0.84620	0.84620
Albania	95	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000	1.00000

2.1.4 Distinguishing urban and rural population

An attempt was made to distinguish population living in cities and in rural areas. As a first step, population density was calculated for all administrative districts. All territorial units with a population density of more than 2.5 persons per hectare were classified as cities. For the remaining administrative regions, city population was taken from www.citypopulation.de and the corresponding cities were allocated using their geographical location given by ESRI (ESRI, 2000a). As mentioned above, no changes in urbanization were assumed for the future (i.e., up to 2075).

The resulting spatial distribution of population (including the locations of the city considered in the analysis) is displayed in Figure 1, and the allocation to urban and rural population is listed in Table 2.

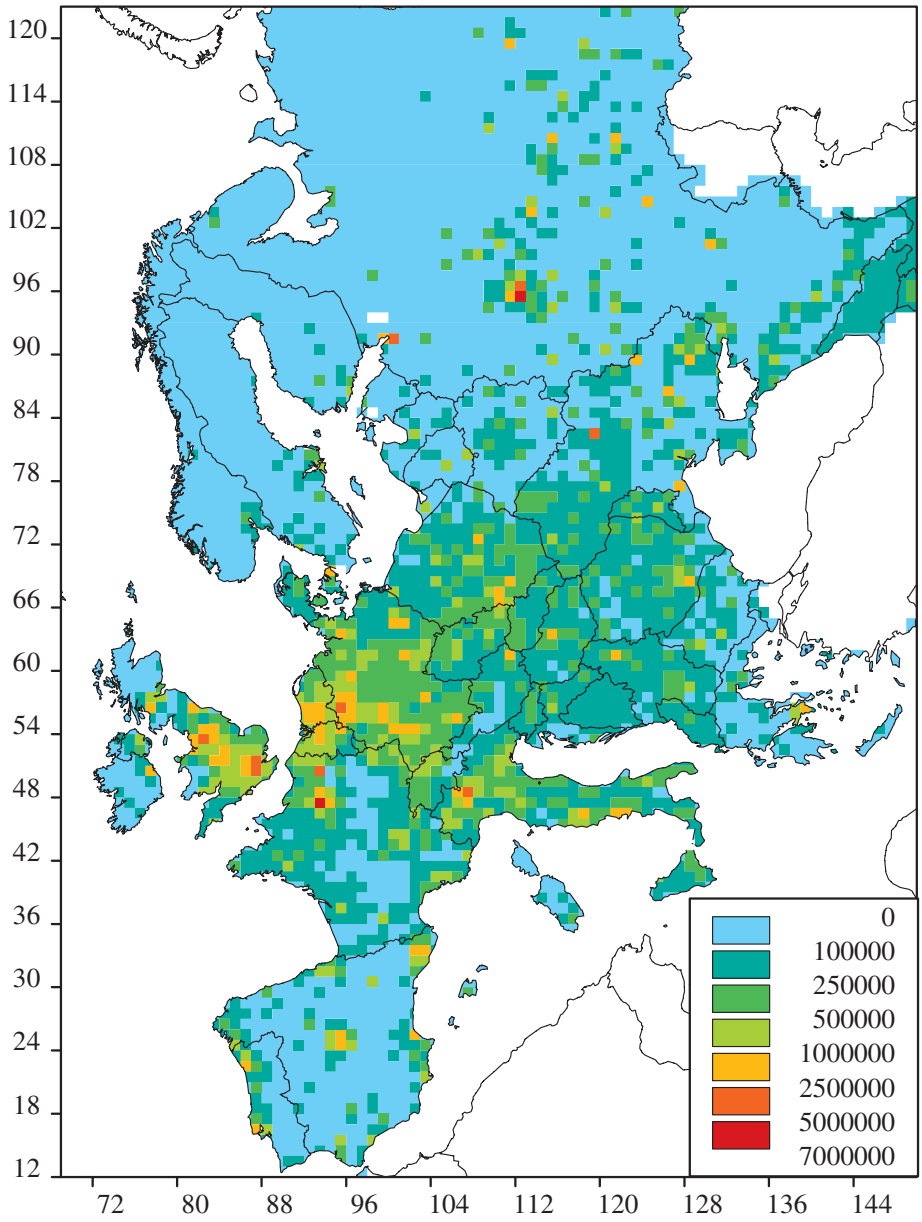

Figure 1: European population in 2000 (persons per 50*50 km grid cell)

Table 2: Rural and urban population in Europe in 2000 and 2010 (1000 persons)

	Total population in 2000			2010	
	Rural	Urban	Total	Total	older than 30 years
Austria	4,341	3,731	8,072	7,953	5,475
Belgium	1,976	8,205	10,181	10,296	6,903
Denmark	3,742	1,543	5,285	5,374	3,548
Finland	2,442	2,699	5,140	5,187	3,426
France	31,774	30,162	61,936	61,203	38,904
Germany	36,100	45,875	81,976	81,353	56,638
Greece	4,956	5,543	10,499	10,579	7,345
Ireland	2,145	1,515	3,660	4,201	2,390
Italy	20,392	37,121	57,512	56,390	40,379
Luxembourg	421	0	421	490	304
Netherlands	2,502	13,109	15,611	16,313	10,757
Portugal	4,308	5,379	9,687	10,082	6,678
Spain	15,024	22,456	37,480	39,569	27,696
Sweden	2,168	6,678	8,846	8,703	5,864
United Kingdom	10,378	48,835	59,213	60,262	38,956
Total	142,668	232,851	375,519	377,955	255,263
Albania	2,422	826	3,248	3,311	1,650
Belarus	7,088	3,148	10,236	9,819	6,162
Bosnia and Herzegovina	4,299	79	4,377	4,269	2,744
Bulgaria	4,730	3,655	8,385	7,185	4,869
Croatia	2,882	1,902	4,784	4,650	2,968
Czech Republic	6,734	3,582	10,315	10,138	6,870
Estonia	785	677	1,462	1,253	812
Hungary	5,355	4,820	10,175	9,489	6,334
Latvia	954	1,467	2,421	2,288	1,504
Lithuania	1,482	2,214	3,696	3,594	2,301
Norway	2,556	1,837	4,393	4,614	2,948
Poland	24,395	14,265	38,660	38,253	23,919
Republic of Moldova	2,969	1,348	4,318	4,190	2,366
Romania	12,735	9,946	22,681	21,819	13,889
Russian Federation	46,567	70,998	117,565	136,976	87,026
Slovakia	3,059	2,329	5,388	5,430	3,384
Slovenia	1,255	732	1,987	1,955	1,342
Switzerland	3,117	3,964	7,081	7,073	4,906
TFYR Macedonia	924	1,060	1,983	2,072	1,233
Ukraine	28,247	21,999	50,245	45,239	29,222
Yugoslavia	5,831	4,107	9,938	10,404	6,416
Total Non-EU	168,454	154,954	323,407	331,417	212,866
Total Europe	311,122	387,805	698,926	709,372	468,129

2.2 Endpoint: Loss in life expectancy

Exposure to outdoor air pollution is associated with a broad spectrum of acute and chronic health effects ranging from irritant effects to death (American Thoratic Society (ATS), 1996a,b). While all these outcomes are potentially relevant for health impact assessment, this study restricts itself to the quantification of changes in mortality resulting from alternative air pollution control scenarios.

Associations between air pollution exposure and mortality have been assessed through two types of epidemiological studies:

- Time series studies of daily mortality measure the proportional increase in the daily death rate attributable to recent exposure to air pollution.
- Cohort studies follow large populations for years and relate their mortality to their exposure to air pollution over extended periods.

Both designs provide estimates of relative risk of mortality that can be associated with exposure to air pollution. It is important to point out that the relative risks derived from time series and cohort studies have different meanings, but refer to similar effects of air pollution: in both cases, pollution-related mortality reflects a combination of acute and chronic effects (Englert, 1999).

The WHO working group on health impact assessment (WHO, 2001) concluded that both designs could contribute useful, albeit different, information. Through their design, time series studies yield estimates of "premature" deaths due to recent exposure, in all likelihood among those who are frail due to either chronic disease, or to some transient condition. Because such studies cannot quantify chronic effects of long-term exposure, some deaths attributable to air pollution will be missed and the extent to which air pollution advances the time of death cannot be quantified (Kuenzli, 2001; McMichael, 1998). For this reason, the use of risk estimates from time series studies of daily mortality will in most cases underestimate the impact of pollution exposure on both the attributable numbers of deaths and average lifespan in a given population.

Cohort studies can provide the most complete estimates. Such studies include not only those whose deaths were advanced by recent exposure to air pollution, but also those who died from chronic disease cause by long-term exposure.

2.2.1 Review of cohort studies

Due to the complexity of conducting cohort studies, only few analyses are available that examine the relation between long-term exposure to air pollution and mortality. These studies quantify relative risks (RR) of mortality that can be attributed to changes in exposure to air pollution. Table 4 summarizes these studies.

An early attempt was made in 1991 by Abbey *et al.*, to look for relationships between air pollution and mortality using health data of Californian Seventh-Days Adventists communities. At that time, statistical analysis was hampered by the non-availability of measurements of fine particulate matter (PM2.5), so that only relations with total suspended particles (TSP) could be examined. No consistent associations between TSP and mortality were found. The study was updated in 1999, following 6,338 subjects from 1977 to 1992 and extending it to PM10 (Abbey *et al.*, 1999). After corrections for age, past smoking, education, occupation and body mass index, a positive association between all-cause mortality and the number of days with PM10 above 100 $\mu g/m^3$ was found for males, but not for females. No associations were found with mean PM10, and with cardiopulmonary and respiratory mortality.

In 1993, Dockery *et al.*, analyzed the mortality of 8000 adults living in six cities in the USA. This "Six Cities Study" followed cohorts of adults aged 25-74 over 14-16 years. The study estimated relative risk (RR) of 1.14 for a 10 μg/m^3 increase in PM10, which corresponds to an 11% change in mortality for each 10 μg/m^3 change in PM2.5. The 95 percent confidence interval of RR was determined at 1.04-1.24.

The largest study using data of the American Cancer Society (ACS) examined the linkage between air pollution and mortality for more than 500,000 people aged older than 30 years in the USA over a time period of eight years (Pope *et al.*, 1995). For fine particulate matter (PM2.5), a relative risk of 1.07 for all-cause mortality (equivalent to a 6.8 percent change in mortality per 10 μg PM2.5/m^3) was found. The 95 percent confidence interval of RR was estimated at 1.04 to 1.11.

In the year 2000, the Health Effects Institute (Krewski *et al.*, 2000) conducted a reanalysis of the original Six City (Dockery *et al.*, 1993) and ACS (Pope *et al.*, 1995) cohort studies. This reanalysis assured the quality of the original data, replicated the original results, and tested those results against alternative risk models and analytic approaches without substantively altering the original findings of an association between indicators of particulate matter air pollution and mortality. In particular, it reconfirmed the relative risks found in the original studies for associations with PM2.5. Smaller associations with mortality were shown for PM15 and PM15-2.5 (coarse particles).

A recent study (Pope *et al.*, 2002) extended the time span of the ACS study to 16 years and tested possible associations of mortality with a wide range of explanatory variables (age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure and diet). It was found that fine particulate (PM2.5) and sulphur oxide pollutions were associated with all-cause, lung cancer and cardiopulmonary mortality (Table 3). Using the Cox proportional hazard model, the study conducted separate analyses for PM observations of the period (1979-1983) of the first ACS study, for the follow-up period (1999-2000) and for both periods combined.

Table 3: Adjusted mortality relative risks (RR) associated with a 10 μg/m^3 change in PM2.5 (Source: Pope *et al.*, 2002).

	Adjusted RR (95% confidence interval)		
Cause of mortality	1979-1983	1999-2000	Average
All-cause	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)
All other cause	1.01 (0.97-1.05)	1.01 (0.97-1.06)	1.01 (0.95-1.06)

Consistent associations were found between ambient levels of PM2.5 and all-cause mortality, cardiopulmonary mortality and lung cancer. For the first period, the relative risks were found to be slightly smaller than those determined in the original study, while the RR resulting from the extension up to the year 2000 match the original estimates. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality.

Table 4: Available cohort studies

Study	Study object	Relative risk (RR) for all-cause mortality
Abbey *et al.*, 1991 (Seventh-Day Adventists study)	TSP 6303 non-smoking Seventh-Day Adventists in California from 1977-1986 Al-cause mortality	No correlation between TSP and all-cause mortality found
Abbey *et al.*, 1999 Update of Seventh-Day Adventists study	PM10 6338 non-smoking Seventh-Day Adventists in California from 1977-1992 Al-cause mortality	RR=1.12 (1.01-1.24) for 10 μg/m^3 PM10
Dockery *et al.*, 1993 (Six Cities Study)	PM 2.5 8000 adults in 6 cities in USA followed up for 14-16 years from 1974-1991, Age: 25-74 at enrolment (max. 90 at end) All-cause mortality	RR=1.13 (1.04-1.24)
Pope *et al.*, 1995 (American Cancer Society, ACS Study)	PM 2.5 Cohort of >552,138 living in 151 cities in US for 7 years from 1982-1989 Age: 30+ at enrolment Average annual all-cause mortality	RR=1.07 (1.04-1.11)
Krewski *et al.*, 2000 (HEI Re-analysis)	PM2.5 Re-analysis of Pope *et al*. (1995) and Dockery *et al*. (1993)	Re-analysis of Dockery *et al.* : RR=1.14 Pope *et al.* (1995): RR=1.07
Pope *et al.*, 2002	PM2.5 Analysis of ACS data for 116 cities in the US for 16 years Age: 30+ at enrolment All-cause mortality, cardiopulmonary mortality, lung cancer	For 1979-1983: RR=1.04 (1.01-1.08) For 1999-2000: RR=1.06 (1.02-1.14) For 1979-2000: RR=1.06 (1.02-1.11)

2.2.2 Personal exposure versus cohort exposure

It is often suggested that personal exposure of individuals may not be well represented by ambient concentrations of pollutants in urban background air, which are usually monitored on a routine basis. As shown by a number of studies, the relation between personal exposure and background concentration depends on the pollutant under consideration, particularly on its dispersion characteristics and whether significant indoor pollution sources exist (e.g., gas cooking for NO_2). While for individuals such relationships were found to be weak, for larger

groups of people ambient background concentrations of PM2.5 represents well the characteristic exposure (Boudeta, 2001).

For purposes of health impact assessment WHO (2001) has pointed out that, while it is common to refer to the results of epidemiological studies of air pollution as providing estimates of the exposure-response relation, most epidemiological studies actually measure the relation between ambient concentration and response. Thus a health impact assessment, to be consistent with the original evidentiary studies, must relate to ambient concentrations rather than to actual personal exposure.

2.2.3 The Cox Proportional Hazards Model

For estimating the concentration-response function, the epidemiological studies described above used the Cox proportional hazards model (Cox, 1972). The proportional hazards model postulates that changing the stress variable (here the change in PM concentrations) is equivalent to multiplying the hazard rate (here the mortality rate) by a proportionality factor, which is here the relative risk function. The fatalities due to PM impacts are usually assumed to be Poisson-distributed, thus the concentration-response function is of log-linear type. The Cox proportional hazard model expresses the number of fatalities in a time period as a function of the baseline fatalities and PM concentrations:

$$y = y_0 * e^{\beta * PM} \quad (1)$$

with y number fatalities

y_0 baseline fatalities

PM PM concentrations

β functional parameter

With the baseline mortality rate μ_0 defined as the quotient of baseline fatalities y_0 and population size P, the adjusted mortality rate $\overline{\mu}$ is calculated as

$$\overline{\mu} = \frac{y}{P} = \frac{y_0}{P} * e^{\beta * PM} = \mu_0 * e^{\beta * PM} \quad . \quad (2)$$

The factor multiplying the baseline hazard rate is also termed "relative risk" RR, which is determined as

$$RR(PM) = e^{\beta * PM} \quad . \quad (3)$$

In the epidemiological studies discussed above, beta is found to be low and the RR function to behave quasi-linearly in the exposure range studied (Pope *et al.*, 2002, p. 1136). Thus, RR can be approximated linearly around 0 by a first-order Taylor series in order to speed up calculations and to facilitate sensitivity und uncertainty analyses:

$$RR(PM) = (\beta . PM) + 1 \quad . \quad (4)$$

2.2.4 Calculating life expectancy from mortality rates

Using the Cox proportional hazards model, a methodology was developed to calculate impacts of various scenarios of precursor emissions of fine particles on the life expectancy of the European population.

The methodology starts from the cohort- and country-specific mortality taken from the life tables and calculates for each cohort the survival function over time. The survival function is modified by exposure to PM pollution, and can then be converted into reduced life

expectancy for an individual person. The calculation uses life-tables and applies an approximation method described in Vaupel and Yashin (1985) for the calculation of the change in life expectancy.

For an age cohort *c* of age *c* at starting time *s* (here 2010) in a grid cell, the change in life-expectancy can be calculated as follows:

The basis for the calculation of life expectancy is the so-called survival function *l(t)* that indicates the percentage of a cohort alive after time *t* has elapsed since starting time *s*. *l(t)* is an exponential function of the sum of the mortality rates $\mu_{a,b}$, which are derived from the life table with *a* as age and *b* as calendar time. As the relative risk function taken from Pope *et al.* (2002) applies only to cohorts that are at least 30 years old, younger cohorts were excluded from this analysis. Accordingly, for an age cohort aged *c* at start *s*, $l_c(t)$ is:

$$l_c(t) = e^{-\sum_{z=c}^{t} \mu_{z,z-c+s}} \tag{5}$$

where c=30, 35,...,95.

Thereby, $l_{30}(t)$ signifies the cohort of age 30 at starting time 2010, μ(30,2010) is the mortality rate for this age cohort in 2010 and μ(35,2015) the mortality rate in 2015 for the same cohort, which will be by then five years older.

The remaining life expectancy e_c for a cohort aged *c* is the integral from *c* to w_1 over $l_c(t)$:

$$e_c = \int_{c}^{w_1} l_c(t)\, dt \tag{6}$$

where w_1 is the maximum age considered (in this study 95 years, this age group also contains persons older than 95).

Exposure to different PM concentrations changes the mortality rate and consequently life expectancy:

$$\bar{e}_c = \int_{c}^{w_1} \bar{l}_c(t)\, dt = \int_{c}^{w_1} e^{-\sum_{z=c}^{t} \mu_{z,z-c+s}}\, dt = \int_{c}^{w_1} e^{-\sum_{z=c}^{t} RR(PM)\mu_{z,z-c+s}}\, dt \tag{7}$$

where $\bar{l}_c$ is the survival function with the modified mortality rates and RR a function of (the change in) PM concentrations following Equation (4):

$$RR(PM) = (\beta PM) + 1$$

The absolute change in life expectancy per person is

$$
\begin{aligned}
\Delta e_c &= \bar{e}_c - e_c \\
&= \int_c^{w_1} \bar{l}_c(t)\,dt - \int_c^{w_1} l_c(t)\,dt \\
&= \int_c^{w_1} e^{-\sum_{z=c}^{t} (\beta PM + 1)\mu_{z,z-c+s}}\,dt - \int_c^{w_1} e^{-\sum_{z=c}^{t} \mu_{z,z-c+s}}\,dt \\
&= \int_c^{w_1} \left(e^{-\sum_{z=c}^{t} \mu_{z,z-c+s}} e^{-\sum_{z=c}^{t} \beta PM\ \mu_{z,z-c+s}}\right) dt - \int_c^{w_1} e^{-\sum_{z=c}^{t} \mu_{z,z-c+s}}\,dt \\
&= \int_c^{w_1} \left(e^{-\sum_{z=c}^{t} \mu_{z,z-c+s}} \left[e^{-\sum_{z=c}^{t} \beta PM\ \mu_{z,z-c+s}} - 1\right]\right) dt \\
&= \int_c^{w_1} \left(l_c(t)\left[e^{-\sum_{z=c}^{t} \beta PM\ \mu_{z,z-c+s}} - 1\right]\right) dt
\end{aligned}
\tag{8}
$$

This specification has the disadvantage that the RR function is part of the exponent of the e-function. In order to simplify, with

$$
l_c(t) = e^{-\sum_{z=c}^{t} \mu_{z,z-c+s}},
$$

the following substitution is permissible :

$$
-\sum_{z=c}^{t} \mu_{z,z-c+s} = \ln\ l_c(t) \tag{9}
$$

Substituting (9) in (8) leads to

$$
\Delta e_c = \int_c^{w_1} l_c(t)\left[e^{\beta * PM\ * \ln\ l_c(t)} - 1\right] dt \tag{9'}
$$

To simplify further, the following linear approximation of (9') by means of a Taylor-approximation of degree 1 around 0 is used. The quality of the fit of this approximation is discussed below.

$$
e^{(\beta * PM)\ln\ l_c(t)} - 1 \approx (\beta * PM)\ln\ l_c(t) \tag{10}
$$

Thus the absolute change in life expectancy per person of a cohort c in year s is

$$\Delta e_c = (\beta * PM) \int_c^{w_1} l_c(t) \ln l_c(t) dt = (\beta * PM) H_c \quad (11)$$

where

$$H_c = \int_c^{w_1} l_c(t) \ln l_c(t) dt .$$

The change in life years for all persons of one cohort in grid cell x,y is obtained by multiplying Equation (11) by the size of the cohort $P_{c/x,y}$ and the length of the time interval for which demographic and mortality data are given. (For this study, data are available for five-years intervals.)

This leads to the change in life years lived for cohort c in grid cell x,y. As cohort data were obtained with reference to the aggregate national level, cohort size in a grid cell was calculated by weighting total population in a grid cell with the relative share of the given cohort in the national population:

$$\Delta L_c = P_{c/x,y} * \Delta e_t * i \quad (12)$$

where

$$P_{c/x,y} = P_{c/national} * \frac{P_{total/x,y}}{P_{total/national}} \quad (12')$$

where

ΔL_c	change in life years lived for cohort c in grid cell x,y
$P_{c/x,y}$	population in cohort c in grid cell x,y
$P_{c/national}$	national population in cohort c
$P_{total/x,y}$	total population in grid cell x,y (at least of age 30)
$P_{total/national}$	total national population (at least of age 30)
i	length of time interval

For all cohorts in a grid cell x,y the change in life years is expressed as the sum of the change in life years for the cohorts:

$$\Delta L_{x,y} = \sum_{c=w_0}^{w_1} \Delta L_c = i * (\beta * PM) * \frac{P_{total/x,y}}{P_{total/national}} \left(\sum_{c=w_0}^{w_1} H_c * P_{c/national} \right) \quad (13)$$

where

w_0	first cohort considered (here 30)
w_1	last cohort considered (here 95)

Dividing (13) by total population at least of age 30 in grid cell x,y leads to the average change in life expectancy in grid cell x,y.

$$\Delta E_{x,y} = \frac{\sum_{c=w_0}^{w_1} \Delta L_c}{P_{total\ /\ x,y}} = i * (\beta * PM\ ) \frac{\sum_{c=w_0}^{w_1} H_c * P_{c\ /\ national}}{P_{total\ /\ national}} \tag{14}$$

In order to calculate the average change in life expectancy for a country *A*, the change in life years in all grid cells of a country divided by total population is computed:

$$\begin{aligned} \Delta E_A &= \frac{\sum_x \sum_y \Delta L_{xy}}{P_{total\ /\ nat.}} \\ &= \frac{i}{P_{total\ /\ nat.}} * \sum_x \sum_y [(\beta * PM_{x,y}) * \frac{P_{total\ /\ x,y}}{P_{total\ /\ nat.}} \sum_{c=w_0}^{w_1} (H_c * P_{c\ /\ nat.})] \\ &= \frac{i}{P^2_{total\ /\ nat.}} * \sum_x \sum_y [(\beta * PM_{x,y}) * P_{total\ /\ x,y} (\sum_{c=w_0}^{w_1} H_c * P_{c\ /\ nat.})] \end{aligned} \tag{15}$$

where ΔE_A is the change in average life expectancy in country *A* expressed in years.

2.2.5 Error due to the linear approximation of the full model

As mentioned in the context of Equation 4, the methodology uses linear approximations for the hazard rate, i.e., of the relative risk and for calculating absolute changes in life expectancy according to Equation 10. This greatly speeds up the calculations since the second term in Equation 15 containing H_c can be pre-calculated and does not need to be computed for each scenario and grid cell.

It turns out that the linear approximation to the full model described above is reasonably good for the estimation of impacts in Europe. Figure 2 shows the estimation error for the "Current legislation" scenario for all grid cells. No clear bias in either direction can be detected.

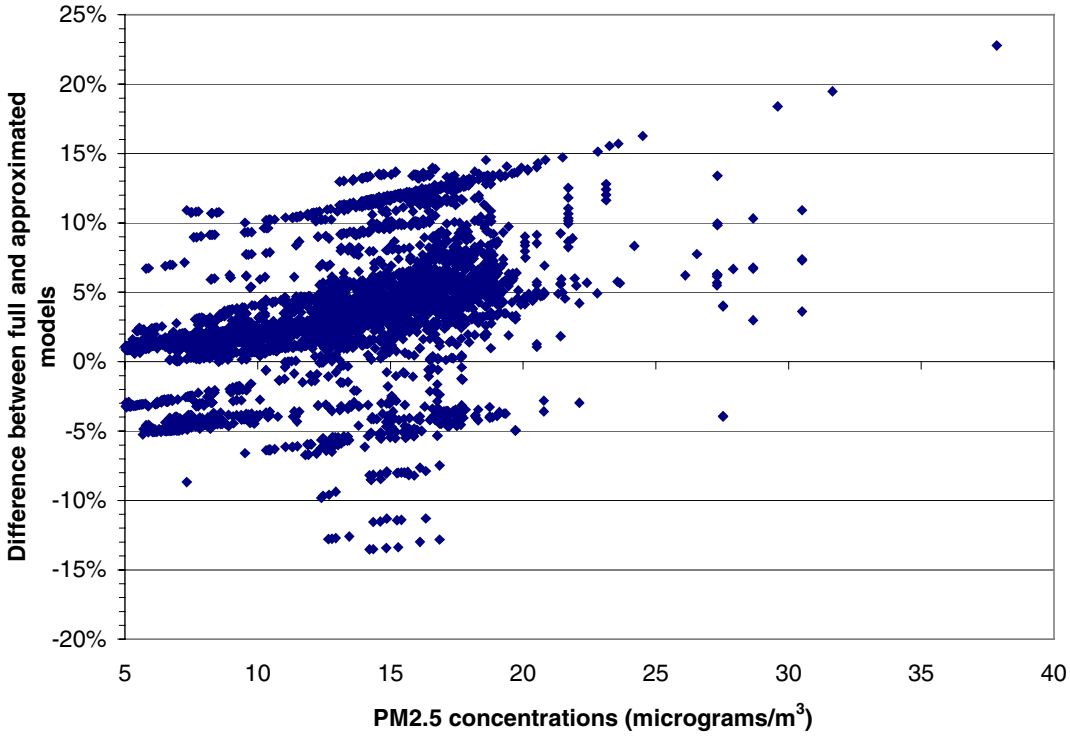

Figure 2: Approximation of the linear approximation model as a function of the change in PM for the CLE scenario in 2010

2.2.6 Transferability

A health impact assessment applies air pollution effect estimates derived from one population to estimate impacts in another (target) population, based on the assumption that these estimates can be transferred. Care must be taken if one cannot assume that the contribution of various causes of deaths is similar, if the mixture of pollutants differs, if the baseline health statuses of the populations are not the same or if exposure ranges do not overlap.

Currently, only the cohort studies listed in Table 4 are available and provide the basis for numerous impact assessments. Since all these cohort studies were conducted in the United States, the generalization of their results to populations in Europe and elsewhere is a concern. Recent studies have begun to explore effect modifiers that may explain the variation in air pollution effect estimates observed among locations in Europe and the United States (HEI, 2000; Katsouyanni *et al.*, 2001). However, results are not yet available and the present knowledge is quite limited, so that it is difficult to include other factors in a practical impact assessment at this point in time.

2.2.7 Extrapolations beyond the range of observational evidence

As pointed out by WHO (2001) caution must be used in extrapolating beyond the range of pollutant concentrations reported in the evidentiary study. The study of Pope *et al.* (2002) to which this assessment refers, covers annual mean PM2.5 concentrations from approximately 5[1] to 33.5 μg/m^3. The analysis in this paper finds, for the European situation, annual mean PM2.5 concentrations from 1 to 80 μg/m^3 (at a 50*50 km resolution). Although recent analyses suggest that there is no discernable threshold for the effects of particulate air pollution on longer-term average mortality from cardio-respiratory disease (Krewski *et al.*, 2000), this paper adopts a conservative assumption and does not extrapolate the calculation of health impacts below the lower range reported in Pope *et al.* (2002) i.e., below 5 μg/m^3. Note, however, that there are about 700 grid cells in Europe (13 percent of all grid cells), where calculated PM2.5 concentrations are below this threshold in 1990, and that this number will increase in the future. This implies that no benefits are calculated from reductions of PM2.5 concentrations below 5 μg/m^3. For the future, sensitivity analyses with modified thresholds are planned.

Similarly, there are 280 grid cells (5.2 percent of all total grid cells), where for 1990 PM2.5 concentrations are calculated to exceed the upper range of 33.5 μg/m^3 analyzed by Pope *et al.* (2002). For these situations the assumption is made that the linear response identified for the study domain does also hold, at least up to annual mean concentrations of 80 μg/m^3.

2.3 Estimates of ambient concentrations of fine particles

Fine particulate matter in ambient air is composed of a large variety of particles with different sizes and physical and chemical properties (e.g., Visser *et al.*, 2001). One may distinguish directly emitted primary particles and secondary aerosols that are chemically formed in the atmosphere from several precursor emissions. Primary particles originate from energy combustion, material handling, industrial activities, surface corrosion, and from natural sources (desert dust, sea salt, pollen, organic material, etc.). A certain fraction of secondary aerosols is of inorganic nature (ammonium salts of nitrates and sulphates) and is generated from sulphur dioxide (SO_2), nitrogen oxides (NO_x) and ammonia (NH_3) emissions, while secondary organic aerosols are a product of complex photochemical processes in the atmosphere involving, *inter alia*, emissions of volatile organic compounds (VOC).

This study estimates, for selected emission scenarios, fields of PM2.5 concentrations with the help of atmospheric dispersion models that describe the formation and transport of primary and secondary aerosols over the European continent. The European perspective is necessary because PM2.5, which has been associated by the evidentiary studies with adverse health effects, has a mean atmospheric residence time of about 50 hours, during which it can be transported over several hundreds up to 1000 kilometers away from its sources.

The study uses two atmospheric dispersion models to estimate the concentrations of various components of particulate matter over Europe. Fields of primary particulate matter (from direct anthropogenic PM emissions) are calculated with the EMEP Eulerian Primary Particulate Model (PPM), while preliminary estimates of secondary inorganic aerosols were computed with the EMEP Lagrangian model. It is foreseen to replace the calculations of the Lagrangian model with results from the forthcoming EMEP Eulerian model, as soon as this will be available.

---

[1] The exact value of the lower range of PM2.5 concentrations was not published in Pope *et al.* (2002). In Figure 1 (Pope *et al.,* 2002: p. 1136) the lower value for PM2.5 concentrations for 1999-2000 is around 5 μg/m.[3] The exact value will be used once more data are made available from the Pope study.

2.3.1 The EMEP Eulerian Primary Particle Model

At present, the EMEP Eulerian Primary Particulate Model (PPM) distinguishes two types of particles: particles with aerodynamic diameters smaller than 2.5 µm (PM2.5 or fine particles) and particles with aerodynamic diameters between 2.5 and 10 µm (coarse particles).

The atmospheric concentrations of primary PM10 have been calculated with a special version of the EMEP Eulerian dispersion model. The model uses the same horizontal, vertical and temporal resolutions and common description of advection and diffusion as the EMEP Eulerian acid deposition model (Olendrzynski *et al.*, 2000). At present, the model is used only for modeling the atmospheric dispersion of anthropogenic primary particles (PPM) that are considered chemically inert. Therefore, the chemical transformation routine of the acid deposition model has been neglected, but the dry deposition parameterization has been modified to allow for a distinction of removal processes according to particle size and land use type. The wet deposition parameterization has also been modified to allow for different in-cloud and sub-cloud scavenging parameters depending on particle size. The Eulerian PPM model sensitivity to the choice of dry and wet deposition parameterization is documented and analyzed in Tsyro and Erdman, 2000.

For the purposes of this paper, the PPM model was run for the emission scenarios described in Section 2.4. It should be noted that at present the PPM model only addresses anthropogenic emissions. Fine particulate matter from natural emissions, such as sea salt and wind blown dust, are not included in the calculations presented in this paper.

2.3.2 Air concentrations of secondary PM2.5 as presently calculated by the EMEP Lagrangian Model

A new Eulerian dispersion model for calculating acid deposition and the formation of secondary inorganic and organic aerosols is presently under construction. Until results from this new EMEP Eulerian model are available, the EMEP Lagrangian model has been used to derive very preliminary estimates of four secondary particles: sulphate, nitrate, ammonium sulphate and ammonium nitrate, which amount to typically 40-50 percent of PM2.5. While the EMEP Lagrangian model has not been explicitly designed to model particulate matter, there is a general correspondence between observations of total PM2.5 and the calculations derived from this model (Tarrason and Tsyro, 1998). However, this correspondence results from the overestimation of nitrate particulates by the model, which in turn compensates for underestimation of concentrations of ammonium and particulate sulphate and for the omission of secondary organic carbon matter.

Thus, the results obtained by now must be considered as illustrative. Improved estimates are expected from the forthcoming EMEP Eulerian model. Once available, they will be used to substitute the results from the Lagrangian model that are used here as placeholders for the health impact assessment.

For the calculations in this paper, linear transfer coefficients describing the formation and dispersion of secondary inorganic aerosols, relative to one unit of emissions, derived from the dispersion model were applied to the emission scenarios described in Section 2.4.

2.3.3 PM concentrations in urban areas

The presently available European-scale atmospheric dispersion models estimate PM2.5 concentrations with a spatial resolution of 50*50 km and are therefore considered representative for rural background concentrations. It is clear from monitoring data that concentrations within cities are usually higher than at regional background sites. Even within cities, certain gradients of PM2.5 levels are found, with higher concentrations at curbsides

than at typical urban background stations. As the evidentiary epidemiological studies identified relationships between mortality and PM concentrations measured at urban background concentrations, use of the results of these studies must obviously relate to urban background concentrations and not to PM levels at curb sites.

In absence of spatially more resolved dispersion calculations covering the European continent, the preliminary assumption was made that in urban background air primary particle concentrations are 25 percent higher than in the surrounding rural background air shed. This should reflect the higher exposure to primary PM emissions from traffic in cities. Due to the longer chemical reaction time of secondary particle formation, no differences in the levels of secondary particles (ammonium sulfate, ammonium nitrate, secondary organic aerosols) are assumed between urban and rural sites. Work is underway to derive more accurate estimates of PM concentrations in cities (see http://rea.ei.jrc.it/netshare/thunis/citydelta/).

2.3.4 Comparison with observations

Although it is not the purpose of this paper to validate calculations of the atmospheric dispersion models, a rough comparison between modeled and observed concentrations of PM2.5 should facilitate a judgment of the outcome of the health impacts. Such a comparison is complicated by the fact that (a) there are only very few monitoring results for PM2.5 available (most of them are rather recent), and (b) the calculations in this paper refer to emissions in 1990. Since then, significant reductions in primary PM emissions and in the precursors for secondary PM were implemented throughout Europe.

Table 5: Comparison of PM2.5 monitoring and model results

Country	Observed annual average PM2.5 concentrations	Calculated country average PM2.5 concentrations for 1990 emissions
Netherlands, various sites	14.2-19.9 μg/m^3 for 1998/1999, TEOM Visser *et al.*, 2001	35.1 μg/m^3
UK, Birmingham	13 μg/m^3 for 1995, TEOM, QUARG, 1996	14.2 μg/m^3
Switzerland, various sites	16-24 μg/m^3 for 2000, gravimetric method, EMEP, 2001	24.1 μg/m^3
Norway, Birkenes	4.2 μg/m^3 for 2001 dichotomous sampler, personal communication	4.0 μg/m3

This limited comparison could support a cautious conclusion that model results and monitoring data seem to be in a similar range. The overestimates apparent from Table 5 have to be interpreted remembering (a) that emissions have substantially declined between 1990, for which the calculations were made, and the end of the decade, from when the monitoring data originate, and (b) that TEOM measurements tend to systematically underestimate the nitrate fraction and thus total PM2.5 mass (typically a correction factor of 1.3 should be applied).

2.4 Emission scenarios

Losses in statistical life expectancy are calculated for three emission scenarios:

- the situation in 1990,
- the situation as it can be expected for the year 2010 assuming implementation of the presently decided emission control policies (the 'current legislation' (CLE) scenario), and
- the hypothetical situation in the year 2010, if all technically available emission control measures were fully implemented (the maximum feasible reductions (MFR) scenario).

Emission estimates are taken from the database of the Regional Air Pollution Information and Simulation (RAINS) model developed at the International Institute for Applied Systems Analysis (IIASA). With the exception of PM2.5, data for 1990 are identical to those used for the scenario calculations carried out for the negotiations of the Gothenburg Protocol and the Directive on National Emission Ceilings of the European Union (Amann *et al.*, 1999). The 2010 CLE scenario assumes implementation of these international agreements, while the MFR scenario explores the full scope of all technical emission control measures contained in the RAINS databases (Cofala and Syri, 1998a,b). For PM2.5, emissions estimates were taken from the recent, preliminary PM databases of the RAINS model (Lükewille *et al.*, 2001). PM2.5 emission data for each country are supplied in Table 6, the emissions of precursors to secondary inorganic aerosols in Table 7 to Table 9.

The preliminary estimates suggest that for the EU-15 present policies will bring primary emissions of PM2.5 down by 54 percent in the year 2010 compared to 1990. Thus, present legislation in the EU will largely exhaust the presently available technical control potential, which allows a 59 percent reduction. This is in stark contrast to the non-EU countries, where, mainly due to the economic decline and the following restructuring of the former centrally planned economies, primary PM2.5 emissions in 2010 are expected to be 48 percent below the 1990 level. There remains, however, in these countries a large technical potential for further reductions (up to –84 percent), which is not addressed by present legislation.

For SO_2, the Directive on National Emission Ceilings requests a 75 percent reduction of the sulphur emissions in the EU-15 between 1990 and 2010. The full technical potential, though at high costs, would allow for a 90 percent cut. In non-EU countries, SO_2 emissions are projected to decline by 56 percent up to 2010, with a technical potential similar to that in the EU-15.

In the EU-15, emissions of nitrogen oxides will be reduced with present legislation by about 50 percent in 2010, while technically 64 percent would be possible. In non-EU countries, a 28 percent decline is foreseen. For ammonia, the recent international agreements stipulate a 15 percent reduction in the EU-15 and a 21 percent cut in the non-EU countries, while the full technical abatement potential was estimated at about 40 percent.

It needs to be emphasized that these estimates must be seen as preliminary and 'work in progress' since they were not yet discussed with national experts. They are used here as illustrative estimates to test the methodology for calculating losses in life expectancy and need to be refined and validated in the future. In fact, combining preliminary estimates derived at different points in time caused certain inconsistencies in the reduction scenarios of some countries.

Resulting changes in ambient concentrations of PM2.5 are presented in Figure 3.

Table 6: Emissions of PM2.5 (in kilotons)

Country	1990	2010 CLE	2010 MFR
Austria	35	22	14
Belgium	36	19	19
Denmark	17	11	8
Finland	41	25	18
France	184	95	94
Germany	464	105	116
Greece	51	51	26
Ireland	13	6	6
Italy	127	68	60
Luxembourg	4	2	1
Netherlands	29	16	18
Portugal	22	19	11
Spain	100	79	63
Sweden	45	22	18
UK	152	70	65
EU-15	1320	611	537
Albania	13	8	2
Belarus	47	27	8
Bosnia-H.	36	23	5
Bulgaria	130	98	24
Croatia	18	12	4
Czech Republic	205	81	26
Estonia	54	17	5
Hungary	65	21	8
Latvia	14	8	3
Lithuania	20	10	3
Norway	13	9	7
Poland	333	259	92
Moldova	14	8	3
Romania	175	107	24
Russia	953	489	148
Slovakia	59	24	11
Slovenia	16	12	4
Switzerland	11	7	7
Macedonia	12	7	2
Ukraine	714	288	76
Yugoslavia	61	36	8
Non-EU	2961	1550	469
Total	4281	2161	1006

Table 7: Emissions of SO_2 (in kilotons)

Country	1990	2010 CLE	2010 MFR
Austria	93	39	31
Belgium	336	106	75
Denmark	182	55	19
Finland	226	116	70
France	1250	400	162
Germany	5280	550	443
Greece	504	546	91
Ireland	178	42	22
Italy	1679	500	200
Luxembourg	14	4	2
Netherlands	201	50	50
Portugal	344	170	36
Spain	2189	774	167
Sweden	119	67	53
UK	3805	625	422
EU-15	16398	4044	1844
Albania	72	55	7
Belarus	843	480	50
Bosnia-H	487	415	24
Bulgaria	1842	846	143
Croatia	180	70	18
Czech Republic	1873	283	265
Estonia	275	175	14
Hungary	913	546	296
Latvia	121	104	18
Lithuania	213	107	23
Norway	52	22	19
Poland	3001	1397	397
Moldova	197	117	19
Romania	1331	594	100
Russia	5012	2343	557
Slovakia	548	110	90
Slovenia	200	27	12
Switzerland	43	26	12
Macedonia	107	81	6
Ukraine	3706	1457	378
Yugoslavia	585	269	32
Non-EU	21599	9523	2477
Total	37998	13567	4321

Table 8: Emissions of NO_x (in kilotons)

Country	1990	2010 CLE	2010 MFR
Austria	192	103	81
Belgium	351	181	124
Denmark	274	127	90
Finland	276	152	88
France	1867	858	633
Germany	2662	1081	929
Greece	345	344	248
Ireland	113	65	42
Italy	2037	1000	728
Luxembourg	22	10	6
Netherlands	542	266	197
Portugal	303	255	152
Spain	1162	847	536
Sweden	338	148	134
UK	2839	1181	809
EU-15	13322	6618	4797
Albania	24	36	16
Belarus	402	255	115
Bosnia-H	80	60	22
Bulgaria	355	266	121
Croatia	82	87	37
Czech Republic	546	286	136
Estonia	84	73	27
Hungary	219	198	110
Latvia	117	84	59
Lithuania	153	110	61
Norway	220	156	125
Poland	1217	879	461
Moldova	87	66	25
Romania	518	437	185
Russia	3486	2653	1097
Slovakia	219	130	84
Slovenia	60	45	30
Switzerland	163	79	64
Macedonia	39	29	11
Ukraine	1888	1222	584
Yugoslavia	211	152	56
Non-EU	10170	7302	3427
Total	23492	13921	8224

Table 9: Emissions of NH_3 (in kilotons)

Country	1990	2010 CLE	2010 MFR
Austria	77	66	48
Belgium	97	74	57
Denmark	122	69	40
Finland	40	31	23
France	810	780	528
Germany	757	550	353
Greece	80	73	59
Ireland	127	116	111
Italy	462	419	282
Luxembourg	7	7	7
Netherlands	233	128	104
Portugal	77	73	52
Spain	352	353	225
Sweden	61	57	44
UK	329	297	218
EU-15	3631	3093	2149
Albania	32	35	25
Belarus	219	158	103
Bosnia-H	31	23	17
Bulgaria	141	108	86
Croatia	40	30	22
Czech Republic	107	101	72
Estonia	29	29	16
Hungary	120	90	73
Latvia	43	35	19
Lithuania	80	81	49
Norway	23	21	17
Poland	505	468	367
Moldova	47	42	29
Romania	292	210	206
Russia	1282	894	571
Slovakia	60	39	30
Slovenia	23	21	12
Switzerland	72	63	54
Macedonia	17	16	11
Ukraine	729	592	406
Yugoslavia	90	82	54
Non-EU	3980	3138	2237
Total	7611	6231	4386

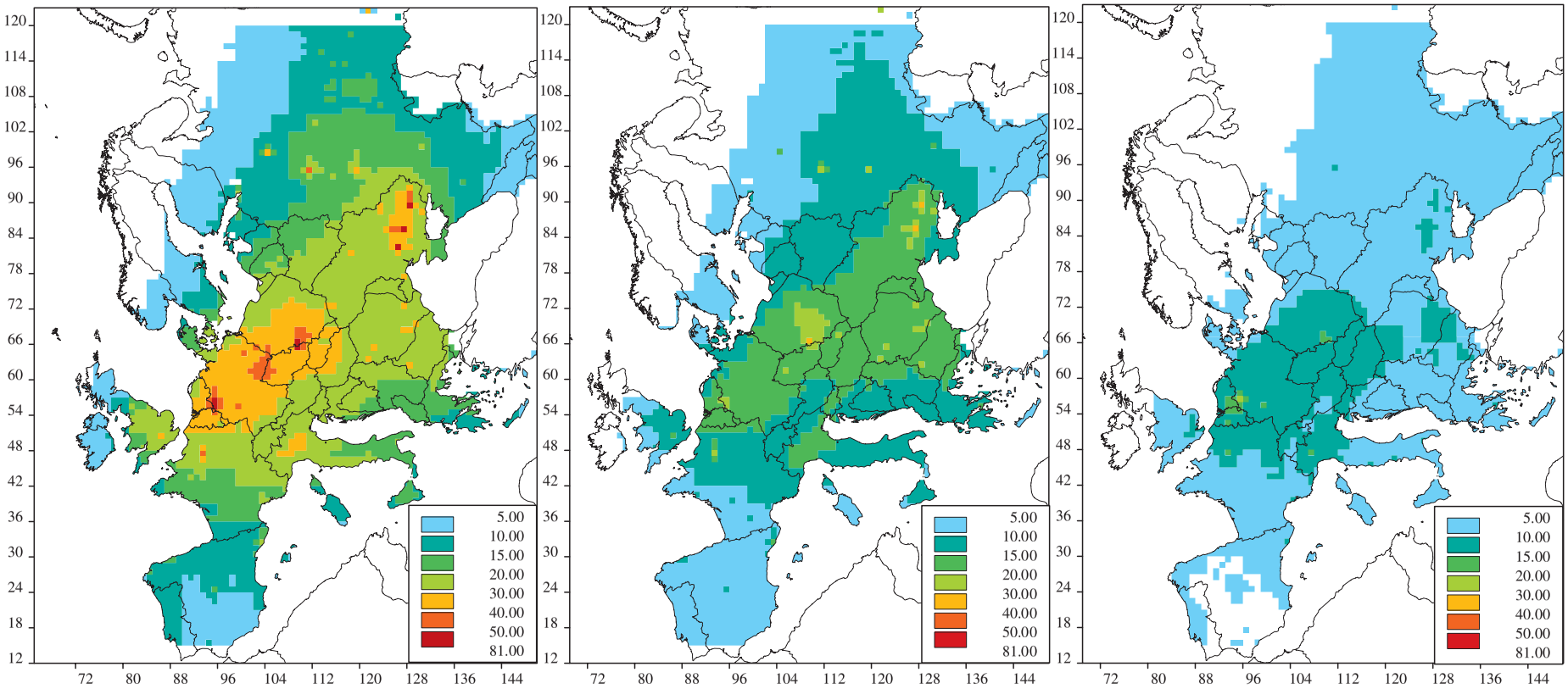

Figure 3: Preliminary estimates of rural background concentrations of PM2.5 in 1990, for the "current legislation" scenario in 2010 and for the technically feasible emission reductions in the year 2010

3 Results

With the methodology, data and assumptions outlined above losses in life expectancies were calculated for the three emission scenarios. Assuming that 1990 emission levels will be kept constant beyond 2010, the largest life shortening due to anthropogenic PM2.5 in ambient air is calculated for the Czech Republic (784 days), Germany (including the new Bundesländer, 733 days) and Netherlands and Poland (701 and 697 days, respectively, see Table 10). Least impacts are calculated for Scandinavian countries (Norway 93 days, Finland 165 days and Sweden 215 days). These preliminary calculations do not include natural sources such as windblown dust, thus they suggest low effects for the Iberian Peninsula (Spain 234 days and Portugal 211 days). These estimates need to be revisited with additional information on the contribution of natural sources, e.g., from the Sahara. On average, life expectancy of Europeans was reduced by 497 days.

By 2010, the implementation of the presently decided emission controls is calculated to improve life expectancy on average by about 218 days, so that PM pollution would cause life shortening of only 278 days on average. Largest improvements are expected for Germany, UK, the Netherlands, the Czech Republic and Belgium, where losses in life expectancy will be cut by more than 50 percent compared to 1990. Full implementation of technically available emission controls would restrict losses to about 205 days in the EU-15 and 178 days in the non-EU countries. This means that after implementation of the presently decided emission controls there remains scope for improving average life expectancy by about 50 days in the EU-15 countries and by 120 days in the non-EU countries through further technical emission controls.

Losses in statistical life expectancy on grid level are presented in Figure 4 for rural areas. A graphical representation of the results is provided in Figure 5.

The results discussed above relate to the statistical life expectancy of all people that are older than 30 years in 2010. These numbers are calculated with the assumption that the pollution level of the selected scenario will remain constant after 2010, so that each cohort will be exposed to this level until the end of its lifetime. This implies that the actual gain in life expectancy will be larger for the 30 years old cohort than, e.g., for the 80 years old cohort, and Table 10 lists the average for all cohorts older than 30 years. For comparison, Table 11 lists the gains in life expectancy of the cohort aged 30 in 2010.

Table 10: Preliminary estimates of reduced average life expectancy due to PM2.5 exposure in 1990, for the current legislation case in 2010 and for the maximum technically feasible emission reductions in 2010 (central estimates)

	Country average PM2.5 concentrations			(Population weighted country average) reduction in statistical life expectancy		
	1990	CLE	MFR	1990	CLE	MFR
	(annual mean concentrations, µg/^{m3})			(days lost)		
Austria	26.4	14.6	10.7	539	297	213
Belgium	34.5	17.1	13.7	684	342	278
Denmark	18.7	9.8	7.4	373	197	147
Finland	6.1	4.0	2.8	165	85	17
France	20.0	11.3	8.9	473	259	209
Germany	33.7	15.7	12.3	733	324	262
Greece	14.4	11.2	7.7	309	245	161
Ireland	7.8	4.2	3.4	172	34	0
Italy	20.0	12.1	9.3	436	258	197
Luxembourg	30.5	15.4	12.3	583	295	234
Netherlands	35.1	16.7	13.5	701	337	277
Portugal	9.3	6.9	4.6	211	157	91
Spain	10.6	7.3	5.2	234	162	96
Sweden	6.9	4.2	3.2	215	113	56
United Kingdom	14.2	7.0	5.5	433	202	155
EU-15 *)	19.2	10.5	8.0	497	258	205
Albania	17.6	12.6	8.0	342	244	153
Belarus	20.5	12.9	7.7	454	285	169
Bosnia and H.	21.1	13.6	8.5	429	278	172
Bulgaria	25.3	17.3	9.8	525	362	198
Croatia	22.7	14.0	9.2	482	295	194
Czech Republic	37.5	18.7	12.3	784	382	245
Estonia	13.5	8.3	5.2	306	180	93
Hungary	29.0	16.9	10.7	639	363	228
Latvia	15.4	9.8	6.2	384	244	153
Lithuania	18.9	11.7	7.3	402	250	156
Norway	4.0	2.6	2.1	93	29	0
Poland	31.1	17.7	11.0	697	405	241
Rep. of Moldova	26.3	16.5	9.6	524	331	192
Romania	26.8	17.4	10.0	598	388	217
Russian Federation	11.2	7.8	5.0	313	206	116
Slovakia	32.2	18.1	11.4	671	369	235
Slovenia	25.8	15.2	10.5	581	344	235
Switzerland	24.1	13.5	10.6	458	256	204
TFYR Macedonia	18.8	13.5	8.3	367	262	159
Ukraine	26.7	16.0	9.1	611	350	187
Yugoslavia	23.8	15.7	9.1	505	331	192
Non-EU *)	22.5	13.8	8.6	494	300	178
Europe *)	21.1	12.4	8.4	496	278	192

*) weighted average

Table 11: Losses in life expectancy due to particulate pollution for the age cohort 30-34 years in 2010 (central estimate, number of days)

	1990	CLE 2010	MFR 2010
Austria	621	342	245
Belgium	780	389	317
Denmark	430	227	169
Finland	184	94	19
France	546	298	241
Germany	832	368	298
Greece	355	281	185
Ireland	186	37	0
Italy	514	304	232
Luxembourg	657	332	264
Netherlands	798	384	315
Portugal	245	182	106
Spain	265	183	109
Sweden	242	127	63
United Kingdom	500	234	179
EU-15	552	291	231
Albania	367	263	165
Belarus	525	329	195
Bosnia and H.	479	311	192
Bulgaria	611	421	230
Croatia	550	337	221
Czech Republic	859	418	269
Estonia	362	213	110
Hungary	727	413	259
Latvia	402	256	161
Lithuania	505	311	192
Norway	106	32	0
Poland	763	443	264
Rep. of Moldova	571	361	209
Romania	684	444	248
Russian Fed.	348	229	129
Slovakia	770	423	269
Slovenia	699	414	283
Switzerland	539	302	240
TFYR Macedonia	411	293	178
Ukraine	681	390	209
Yugoslavia	600	393	229
Non-EU	561	341	201
Average	557	317	215

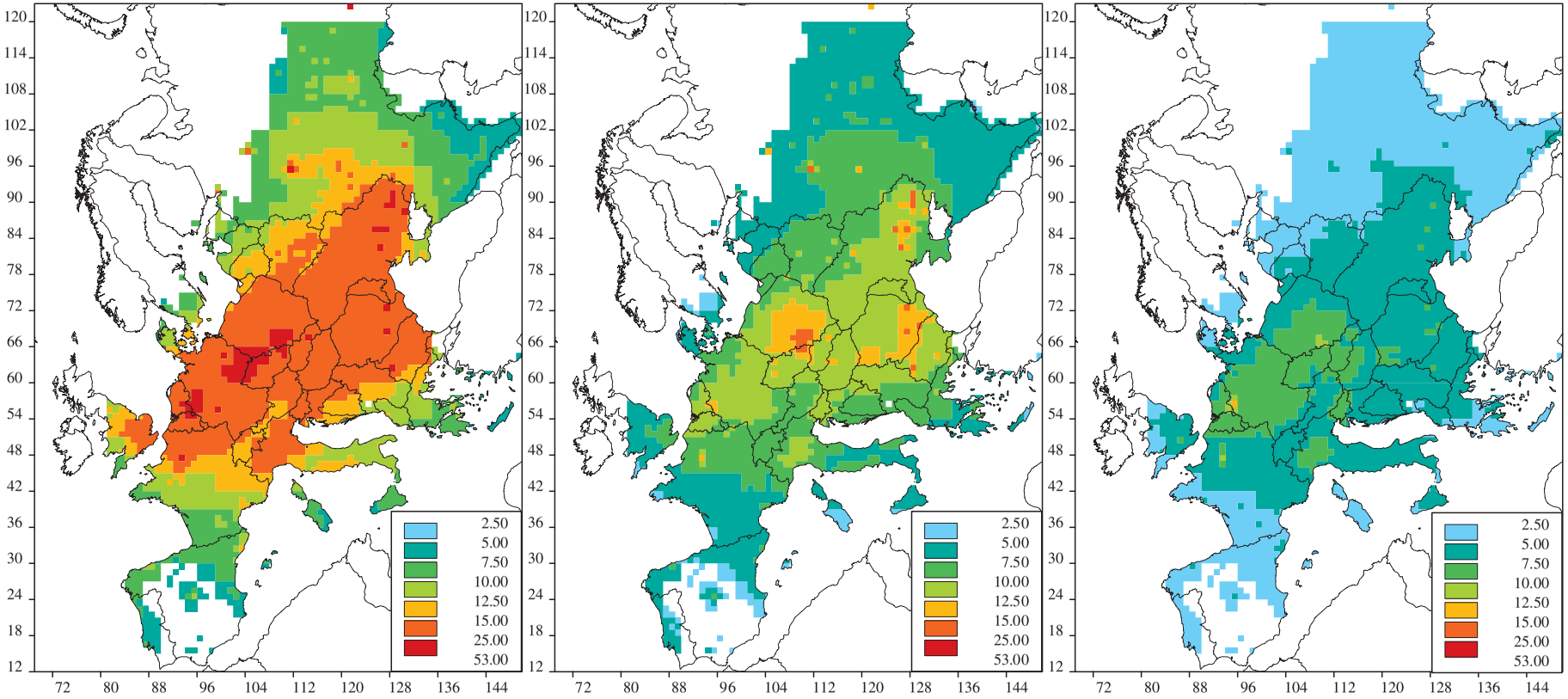

Figure 4: Losses in statistical life expectancy in rural areas due to particulate pollution, in 1990 (left panel), for the current legislation scenario in 2010 (central panel) and the maximum technically feasible reductions (right panel), in months

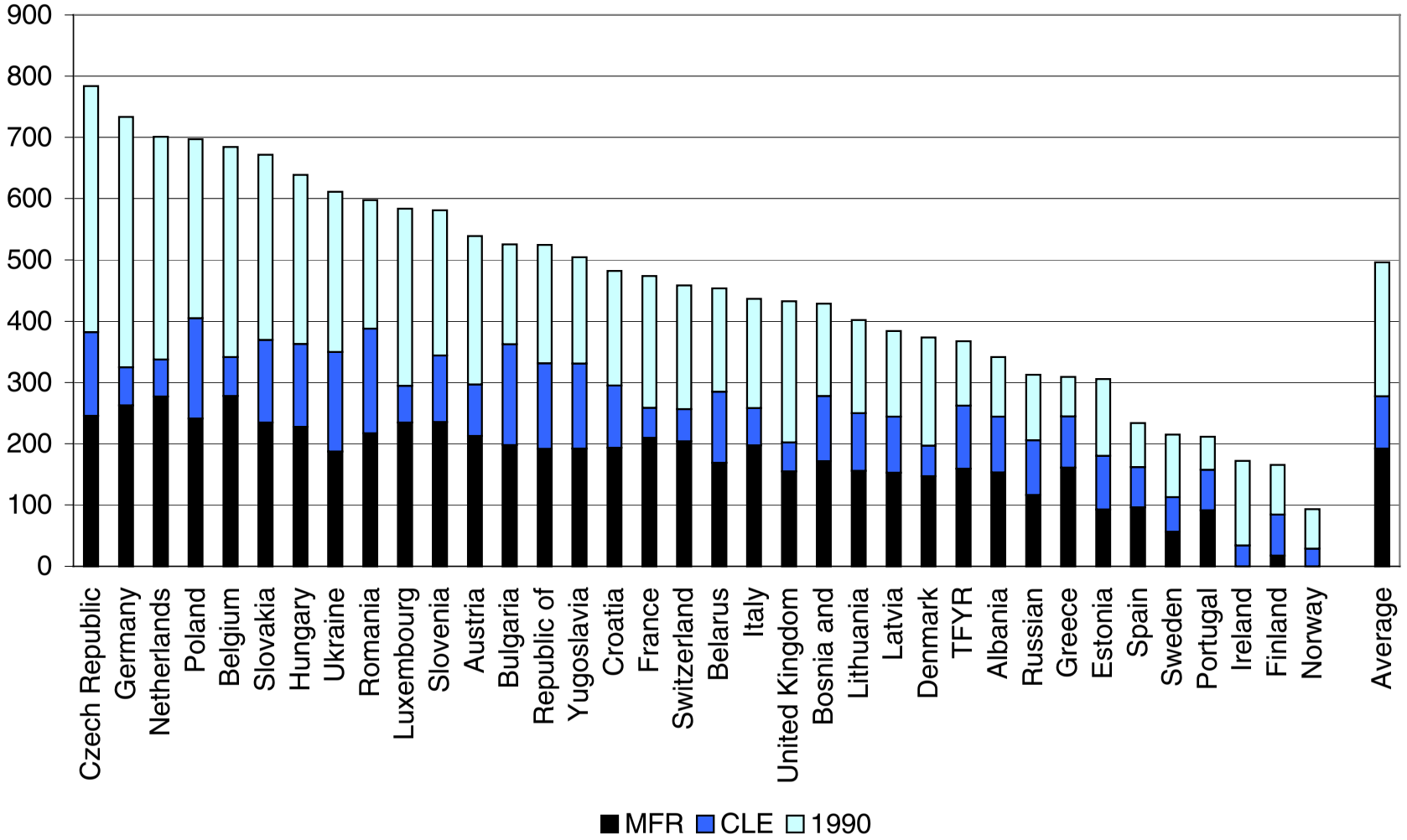

Figure 5: Losses in statistical life expectancy for the emission scenarios (in days)

4 Uncertainties

It is the objective of this paper to present a methodology for estimating losses in life expectancy due to air pollution at the European scale and to explore the potential order of magnitude of this effect. Many of the data, models and assumptions used for these illustrative calculations are preliminary and need refinement and further validation before robust quantitative conclusions could be drawn. This applies, *inter alia*,

- to the estimates of primary PM2.5 emissions in Europe,
- to the projections of emission levels of PM and other pollutants in the year 2010 in Europe,
- to calculations of the formation and atmospheric dispersion of primary and secondary aerosols in Europe,
- to estimates of ambient PM levels in urban air sheds,
- to the use of appropriate dose-response curves derived from epidemiological studies,
- to the question which property of particulate matter is causally linked with mortality.

Each of these aspects is associated with considerable uncertainties. By linking this information, the methodology to estimate losses in life expectancy combines these uncertainties. Suutari *et al.* (2001) developed a methodology to propagate uncertainties through a similar chain of model calculations aiming at determining ecosystems protection from alternative emission control scenarios. It was shown that, as long as the uncertainties in different elements of the model chain (e.g., the estimates of emissions and of ecosystems sensitivities) are statistically independent from each other, uncertainties do not accumulate, but compensate each other to a large extent.

In principle, this methodology could equally well be applied to the calculation of losses in life expectancy to quantify uncertainties of the overall results, although in practice such an implementation would take considerable time and resources. Instead, a partial sensitivity analysis was conducted using the upper and lower bounds of the 95 percent confidence interval of the relative risk function identified by Pope *et al.* (2002). Thus the sensitivity analysis explored the losses in life expectancy resulting from relative risks of 1.02 and 1.11, compared to the central estimate of 1.06 per 10 $\mu g/m^3$. Results suggest for 1990 the loss in average life expectancy ranging from 168 to 888 days with 496 days as the central estimate (Table 12). For the CLE scenario, the range extends from 94 to 497 days, while the maximum feasible reduction case results in 65 to 344 days.

Table 12: Sensitivity analysis: Reduced life expectancy due to PM2.5 exposure for the three emission scenarios, for the lower and upper ranges of the 95 percent confidence interval of the relative risk identified in Pope *et al.*, 2002.

	Lower confidence range (RR=1.02)			Upper confidence range (RR=1.11)		
	1990	CLE days	MFR	1990	CLE days	MFR
Austria	183	101	72	965	532	381
Belgium	233	116	94	1226	612	498
Denmark	127	67	50	668	352	263
Finland	56	29	6	296	151	31
France	161	88	71	848	463	375
Germany	249	110	89	1313	581	470
Greece	105	83	55	554	438	288
Ireland	58	12	0	308	61	0
Italy	148	88	67	782	462	353
Luxembourg	198	100	80	1045	528	420
Netherlands	238	115	94	1255	604	496
Portugal	72	53	31	379	282	164
Spain	79	55	33	418	290	172
Sweden	73	38	19	385	202	101
United Kingdom	147	69	53	775	362	277
EU-15 *)	169	88	70	890	462	367
Albania	116	83	52	612	438	274
Belarus	154	97	57	812	510	302
Bosnia and H.	146	94	58	768	498	307
Bulgaria	178	123	67	940	649	354
Croatia	164	100	66	863	528	347
Czech Republic	266	130	83	1404	684	440
Estonia	104	61	31	548	322	166
Hungary	217	123	77	1144	650	408
Latvia	130	83	52	688	437	274
Lithuania	137	85	53	720	448	280
Norway	32	10	0	167	51	0
Poland	237	138	82	1248	725	432
Rep. of Moldova	178	113	65	939	594	343
Romania	203	132	74	1070	695	388
Russian Federation	106	70	39	560	369	208
Slovakia	228	125	80	1202	661	420
Slovenia	197	117	80	1041	616	422
Switzerland	156	87	69	821	459	365
TFYR Macedonia	125	89	54	658	469	286
Ukraine	208	119	64	1095	626	335
Yugoslavia	171	112	65	904	592	345
Non-EU *)	168	102	61	885	538	319
Europe *)	168	94	65	888	497	344

5 Discussion and conclusions

5.1 Discussion

This paper introduces a methodology to estimate reduced life expectancy due to particulate pollution in Europe. It combines epidemiological evidence about a systematic association between fine particulate matter (PM2.5) and increased mortality with inventories and projections of emissions of PM2.5 in Europe and calculates the implications of mortality changes due to population exposure to PM2.5 concentrations on statistical life expectancy in the various European countries.

The paper also presents a preliminary implementation for Europe based on presently available models and data. This implementation should be considered illustrative, demonstrating that, in principle, all information required for the health impact assessment is available and to explore the order of the magnitude of effects. Many elements of the calculations presented in this paper have to be considered as preliminary placeholders. It is envisaged that within the next few years the work programs of the Convention on Long-range Transboundary Air Pollution and the Clean Air for Europe (CAFE) program of the European Commission will improve many important ingredients of the calculations. Work is progressing to enhance emission inventories of particulate matter, to refine the Eulerian dispersion models of aerosols and validate them with more monitoring data, to improve estimates of ambient levels of particulate matter in urban air, and to obtain comprehensive advice from WHO about the use of epidemiological evidence for health impact assessment. Such improved knowledge will reduce many uncertainties of the calculations presented in this paper.

However, there are other uncertainties, which are not expected to disappear as a result of the improved information that is expected to become available within the next few years. For instance, there might not be complete certainty about the causal factor in particulate matter leading to increased mortality, and a range of alternative hypotheses might prevail.

There are also a number of methodological uncertainties in the calculations of reduced life expectancy that will most likely not be completely resolved within the next few years. Questions about the transferability of results from the evidentiary studies conducted under conditions in the United States to the European situation, taking into account, e.g., differences between Western European, Eastern European and Mediterranean countries. This also applies to the range of PM pollution that is covered by the US studies. It turns out that considerable areas in Europe have significantly higher PM pollution levels than found in the US studies, and assumptions need to be adopted about the extrapolation of the response curves found in the US to the higher PM levels in Europe.

At the same time, the atmospheric calculations applied in this analysis suggest for areas in Scandinavia PM levels below the lower bound of the US studies (however, these preliminary calculations do not include natural emissions). The assumption of a lower cut-off threshold for PM effects (in order to remain within the validity of the statistical analysis of the evidentiary studies) has implications about potential benefits of improving air quality even at relatively clean sites. This needs to be discussed in view of the absence of a no-effect threshold concentration for PM as suggested in the literature (e.g., Krewski *et al.*, 2000).

Transferability is also an issue in the context of a potential confounding role of other pollutants present in European air sheds. This study calculates mortality effects exclusively for PM pollution. At the same time, certain effects are also demonstrated in the literature for sulfur dioxide (Pope *et al.*, 2002), ozone, and nitrogen oxides (Katsouyanni *et al.*, 2001). While care must be taken to avoid double counting of effects by adding up responses from multiple pollutants, some studies indicate potentially independent effects, e.g., for SO_2 and ozone.

As explained above, the calculations in this paper are limited to population of age 30 years and older. There is, however, some recent evidence about impacts of particulate pollution on

infant mortality (Bobak and Leon, 1999; Woodruff *et al.*, 1997), which could have very strong effects on statistical life expectancy.

5.2 Preliminary results

Keeping these imperfections in mind, the preliminary results of this assessment suggest life expectancy in Europe to be significantly shortened by particulate pollution, with the present assumptions between three months in Scandinavia and more than two years in central Europe. The 95 percent confidence interval of these estimates due to uncertainties in the evidentiary epidemiological studies ranges between one and five months in Norway and nine months and six years in Central Europe.

This situation is expected to profoundly change in the future due to the recently agreed emission controls. The Gothenburg Protocol of the Convention on Long-range Transboundary Air Pollution and the Emission Ceilings Directive of the European Union should bring significant reductions of the precursor emissions of secondary aerosols: SO_2 will be cut by 75 percent compared to 1990, NO_x by 50 percent and ammonia by 12 percent. Primary emissions of PM2.5 are expected to decline by 50 percent as a consequence of stringent controls for stationary and mobile sources.

These emission controls will reduce the average loss of life expectancy in Europe to somewhat more than nine months in Europe, spreading from one month in Norway to 13.5 months in Poland. Full application of all available technical control measures could further reduce these losses by another 25 percent.

The results obtained from this study can be compared with other work in the literature. A report of the Committee on the Medical Effects of Air Pollutants has applied a similar methodology to quantify long-term effects of particles on life expectancy (COMEAP, 2000), based on Hurley *et al.* (2000). This study focused on the UK and did not quantify scenarios of potential improvements in air quality, but analyzed gains in life expectancy for a hypothetical 10 $\mu g/m^3$ improvement of PM10. The study found that for the entire population alive in 2000, if the 10 $\mu g/m^3$ improvement were maintained for the rest of their lives, the population would gain between 4 and 26 million life years, which is equivalent to about 1-6 months per person. For comparison, the analysis presented in this paper suggests for the UK cohorts older than 30 years a range from 1.8 to 13.77 months with 7.6 months (231 days) as the central estimate for a country-average 5.6 $\mu g/m^3$ reduction of PM2.5 levels between 1990 and the CLE scenario. Such a 5.6 $\mu g/m^3$ improvement in PM2.5 is roughly equivalent to a 10 $\mu g/m^3$ change in PM10 concentrations, so that the numerical results can be directly compared with each other. Obviously, the exact magnitude of the effect depends crucially on the assumption of the relative risk taken by a particular study, which is slightly lower in the UK study conducted in the year 2000 than that of this paper, which relies on the recent 2002 study of Pope *et al.*

The effect of particulate air pollution on life expectancy was explored by Brunekreef *et al.* (1997). This study explores the response in statistical life expectancy for a Dutch cohort of 100,000, using a relative risk of 1.1 for a 10 $\mu g/m^3$ change in PM2.5. This results in a gain in life expectancy of 1.11 years. This is consistent with the gain of one year found for the Dutch population in this study, which is based on a relative risk of 1.06 found in Pope *et al.* (2002).

Nevalainen and Pekannen (1998) conducted a similar analysis for Finland. While the authors rely on the same evidentiary studies available at that time (i.e., Dockery *et al.*, 1993, Pope *et al.*, 1995), they explore the implications on life expectancy for Finland by using Finnish demographic data. They do not, however, estimate ranges of PM pollution that are realistic for Finland, but conduct the analysis for a hypothetical 10 $\mu g/m^3$ change.

5.3 Outlook: Loss of life expectancy as an additional endpoint in a multi-pollutant/multi-effect assessment

The RAINS model offers a framework to address multi-pollutant control strategies that simultaneously address several environmental endpoints. For the scenario analyses conducted for the Gothenburg Protocol of the UN/ECE Convention on Long-range Transboundary Air Pollution and the Directive on National Emission Ceilings of the European Union, the model identified cost-effective balances of emission reductions of SO_2, NO_x, VOC and NH_3 in order to reduce acidification, eutrophication and harmful effects of ground-level ozone (Amann and Lutz, 2000). With these emissions as precursors for the formation of secondary aerosols, health impacts of PM pollution could be additionally included in such a multi-pollutant/multi-effect framework (Table 13).

	SO_2	NO_x	NH_3	VOC	Primary PM
Acidification	√	√	√		
Eutrophication		√	√		
Ground-level ozone		√		√	
Health impacts from fine particles	√	√	√	√	√
	(via secondary aerosols)				

Table 13: The multi-pollutant/multi-effect framework of the RAINS model

While the calculations presented in this paper have to be considered as illustrative at the present stage, the availability of the methodology and its integration in the RAINS model opens the possibility to introduce gains in statistical life expectancy as a further environmental endpoint in the multi-pollutant/multi-effect analysis. Thereby, emission control strategies could be explored that balance emission controls over primary and secondary particles (and thereby address health effects from particulate matter), while keeping full account of benefits to and requirements from improvements of acidification, eutrophication and ground-level ozone.

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