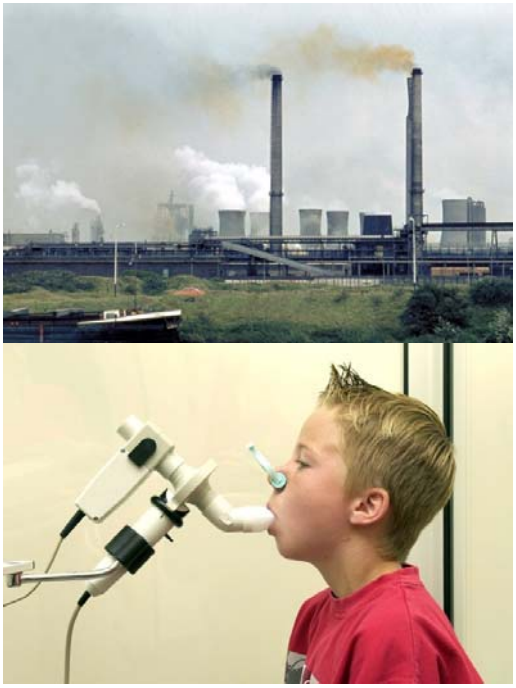
Health Impacts and Air pollution -
An exploration of factors influencing estimates of air pollution impact upon the health of European citizens



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European Topic Centre
on Air and Climate Change

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Summary

The great diversity of environments and lifestyles across Europe poses a challenge when it comes to estimating Europe-wide consequences of air quality upon the population. The size of communities, the nature of economic activity, the pollutant of concern, the geographical location – all of these factors and more besides will influence the effect air quality may have upon human health. Yet to date, the approach to estimating health impact for the whole continent has utilised a restricted spatial resolution of air quality variability (10km2), and simplified estimates of population distributions (static residential population densities). The challenge is to identify the main factors which will modify current estimates, to quantify the magnitude of such modifications, and to estimate the resolution needed to appropriately accommodate this diversity. As significant factors are identified and better quantified, the robustness of estimates increases.

This report addresses the following aspects:

1) The spatial scale of assessment
2) The influence of meteorological differences at street level
3) The influence of daily intra-urban migration on exposure to air pollution
4) The comparative health effects of finer particulate air pollution
5) The statistical description of the impact of particulates and of ozone.

Simultaneously, through case studies in Silesia, Athens, London and Oslo the report begins to contribute depth to our understanding of the impact of air quality upon health across Europe's various environments.

Assessment at a finer spatial resolution is shown to increase the estimates of total exposure experienced by a population. Determining exposure on the basis of air concentrations at residential addresses rather than total populations exposed to an urban average concentration is the first step in this direction. Similarly, improving temporal resolution improves our use of spatial information through description of intra-urban temporal population movement. Estimated total exposure increases.

Commuting is found to have real potential importance to exposure estimates. The daily movement of a city population daily towards the centre, including commuting on traffic corridors, increases the general exposure level. This typically means exposure to higher concentration brackets for a limited percentage of the population, rather than only longer exposure to existing brackets. Indeed, whilst the estimated increase in average urban exposure may be around 20%, this may be a misleading statistic as increased exposure and associated potential health effects are in reality focused on an identifiable target group. Although the proportion of the urban population thus affected may be restricted, the magnitude of the effect may translate into large absolute numbers across Europe as a whole.

Review of recent literature on the health impacts to exposure to $PM_{2.5}$ lends continuing support to the existing coefficient used for estimating mortality, of 6% per 10µg/m^3 of $PM_{2.5}$. Similar review finds evidence to suggest combined adverse effects of exposure to $PM_{2.5}$ along with exposure to ozone. When Europe-wide estimates are made, the numbers of total estimated premature deaths from exposure to $PM_{2.5}$ approximates those already estimated to result from exposure to PM_{10}. Indeed, it is found that for 10 Member States the Average Exposure Index lies above the 2015 binding value of 20 µg/m^3, in 5 Member States it lies at or slightly below this level, whilst for 12 Member States the average exposure index is clearly below.

Contents

Preface

This report begins exploration of the factors which modify the current estimates of the impact of air pollution on human health in Europe's urban areas. The work is an expansion of past Topic Centre activity in the mapping of population exposure to air concentrations, and the calculation of premature deaths.

There is a desire to investigate the robustness and sensitivity of existing estimates of health impact. Questions arise concerning the spatial resolution of estimates, the significance of detailed pollutant distributions in cities, population movements between areas of differing pollutant concentrations, the scale of morbidity impacts, and health impacts in areas with complex mixtures of pollutants.

This discussion paper is intended as an aid to determining the necessary focus for agency work in achieving still more robust estimates of health impact.

Acknowledgement
Valuable advice and comment has been voluntarily provided throughout to the team compiling this report by Dr. Michel Krzyzanowski of the World Health Organisation, in Bonn. The content of this report bears evidence of this input. The team wishes to express their deep thanks for this support.

1. Introduction

1.1. Purpose of this work

At present, the 10km x 10km grid cell averaged estimated air pollution concentrations are used by the European Topic Centre on Air and Climate Change (ETC/ACC) as the basis for estimating air pollution impact upon the health of European citizens. Although these estimates are essentially derived from monitored air quality data together with assimilation of model results as needed, their large spatial resolution, with consequent use of a city averages (average concentration at all operational (sub)urban background stations) without intra-urban gradients either in concentrations or populations raises uncertainty as to the accuracy of assessments. This report begins discussion of some of the principal factors.

At a minimum, there is a danger of underestimating exposure given the correlation that exists between population density and air pollution concentrations. But the picture is more complex than this. The gradients in populations and air quality in cities may be large. Populations move daily across zones of different pollution levels as well as commuting along highly polluted road corridors. The scale of averaging can soften the gradients which actually exist. Health impact may be expected from other pollutants than have been regularly assessed so far. And so forth.

This report seeks to explore these aspects, in order to establish the scale of departure from a uniform city or 10x10 km grid cell averaged exposure that may actually exist.

In particular, this report examines the following:

- **The effect of spatial scale of assessment**: A case study is presented from Silesia-Moravia on estimates of health impacts in a long standing heavy industrial centre evaluating the differences which arise when reducing the spatial scale of assessment from 10km to 1km.
- **Significance of micro-scale variability in pollutant concentrations**: Evaluation in Athens and London of the influence that the internal urban physical (micro)structure may have upon air concentrations and consequent health assessments.
- **Population distributions and mobility**: The consequence is examined in Oslo of careful representation of intra-urban air quality and population distributions, and of daily intra-urban movements on the population exposure distributions.
- **Impact of $PM_{2.5}$ in Europe**: Preliminary estimates using available monitoring data are presented of the comparative impact upon health of $PM_{2.5}$ and PM_{10} across Europe.
- **Recent progress in quantifying health impacts**: Recent literature on air pollution impacts on health have been reviewed, in particular coefficients of impact for $PM_{2.5}$, but also ozone.

1.2. Current approach to air quality Health Impact Assessment

Current Health Impact Assessments (HIA) for air quality deals with impact pathways – a quantified link between a pollutant and a defined health endpoint where the quantification reflects a broad consensus on the evidence for a reliable association. To date in the work of the ETC/ACC, the pollutants in focus has been PM_{10} and ozone, and mortality to chronic exposure has been the endpoint. The air quality data used as input to assessment has been interpolated concentration maps constructed at 10km x 10km resolution (see Horalek et al, 2008).

A significant aspect of current practice is that it is assumed that the population within these 10km grid cell is exposed to the same grid cell averaged concentration. Concentration and population density is

applied at 10km resolution so that gradients within a cell, differences in exposure for different population classes and indoor pollution have not been included in assessments so far.

For quantifying the effect of air pollution, the relative risk (RR) in a population is estimated by the concentration response function:

$$RR = \exp\left[B\left(C - C_0\right)\right]$$

Where C is the average air concentration to which the population is exposed, C_0 is a reference concentration (the background concentration that would exist without pollution or a concentration below which no health effects are to be expected), B is the estimated effect of the pollutant on the health outcome (e.g. total mortality excluding violent death) and is given as an increase in incidence per unit increase in concentration. In the assessments presented to date the reference concentration C_0 is set to zero.

Once the relative risks have been determined, the attributable fraction (AF) of a specific health effect from air pollution for the exposed population is:

$$AF = \sum P_i \left(RR_i - 1\right) / \sum P_i RR_i$$

where Pi = the proportion of the population at exposure category i
RRi = the relative risk in exposure category i

When the total population is considered with only one exposure level, this simplifies to:

$$AF = \left(RR - 1\right) / RR$$

The expected total number of cases of premature mortality due to air pollution is given by:

$$E = AF \cdot MR \cdot Pop$$

where *E* is the expected number of deaths due to outdoor air pollution,

MR is the population incidence of the given health effect (i.e. cases per 1000 people per year) and

Pop is the relevant exposed population for the health effect.

National demographic data (absolute numbers, age/sex distributions) are taken either directly, or after downscaling from regionalised level to the national level using data of the World Population Prospects (e.g. UN, 2005). Similar age distributions for each grid cell within a country are assumed. Information on baseline incidences is obtained from the WHO Burden of Disease project (WHO, 2004; Mathers and Loncar, 2006). *MR* is estimated using age and sex dependent baseline incidences. An example of summarised results is given in table 1.1.

Table 1.1 Premature deaths attributable to exposure to ambient PM_{10} concentrations, using methodology described in section 1.2.

country	PM_{10}
Austria	6805
Belgium	11847
Bulgaria	14429
Cyprus	1195
Czech Republic	13385
Germany	77061
Denmark	4525
Estonia	1116
Spain	42795
Finland	2639
France	37965
Greece	15829
Hungary	15865
Ireland	1524
Italy	77667
Lithuania	3043
Luxembourg	257
Latvia	2313
Malta	449
Netherlands	16673
Poland	39049
Portugal	12030
Romania	33550
Sweden	5575
Slovenia	2064
Slovakia	5655
United Kingdom	51537
Albania	2754
Andorra	39
Bosnia and Herzegovina	3755
Croatia	6700
Iceland	92
Lichtenstein	23
Norway	3096
San Marino	31
Serbia and Montenegro	17735
Switzerland	4963
TFYR Macedonia	2813
EU27	496842
Total	538843

2. Intra-urban audits: exploring the differences within cities

This chapter presents three analyses each employing numerical air pollution modelling to evaluate:

- the significance of spatial scale upon impact estimates,
- the potential role of microscale air concentration differences on impact estimates
- the significance of urban mobility on exposure

2.1. Decreasing the spatial scale of assessment from 10km to 1km: Health impact of air pollution in the Moravian-Silesian Region

2.1.1. Introduction

The Moravia-Silesian region (MSR) is one of the most polluted regions of both the Czech Republic and Europe. PM10, benzo(a)pyrene (BaP) and benzene ambient concentrations exceed EU limit and target values. The neighbouring Polish part of Silesia also belongs to this highly polluted region, although limited data from this region hampered extension of this case study to Poland.

The Moravian-Silesian region (5445 km^2-7% of the Czech Republic) is in the north east on the borders with Poland and Slovakia. On the west is the massif of the Hrubý Jeseník Mountains., incl. the highest peak of the region Praděd, 1492 m. The middle of the region is the lowland of the Opava Plain and Ostrava Basin (incl. the lowest point of the region - outflow of Odra River,198 m), with agriculture, industry, biggest towns and highest population density. The south-east is the Beskydy massif (highest peak Lysá hora, 1323 m), with agriculture (> 50 %) and forests (> 35 %).

The region is located on the historical transport route running north-south from the Baltic to the Mediterranean. Today, road transport is characterised by a dense net of regional and local roads and connections to Poland and Slovakia. The industrial tradition is long, being one of the most important industrial regions in Europe since 19th century. In 1763 coal mining in Ostrava was opened, in 1828 the first ironworks were founded. In the 2nd half of the 19th century the region underwent dynamic industrial development of coal mining, metallurgy, steel industry, and engineering. At the turn of the 20th century Ostrava city was the industrial centre of the Austro-Hungarian Empire and this period saw a remarkable growth in population. Since 1989 restructuring and revitalization of heavy industry has taken place, with the closure of many coal mines, inflow of domestic and foreign investments, and growth of the automobile industry.

Demography: Population density is about 230 inhabitants/km^2. The most populated city is the capital of the region - Ostrava city, 310 741 inhabitants. Almost 62 % of citizens live in towns with a population over 20 000 inhabitants and about 25 % of inhabitants live in settlements with population lower than 5 000. At the end of 2006 the region had 1 249 290 inhabitants (Czech Statistical Office, final data). The number of live born children (12 381) is lower than the number of deaths (12 657), meaning a decline of 0.2 per 1000 annually. Infant mortality (deaths under 1 year per 1 000 live births) fell to 3.4 per mille in the Czech Republic as a whole, but in Moravia-Silesia increased to 4.1 per mille. Neonatal mortality in the Czech Republic (deaths under 28 days per 1 000 live births) increased to 2.3 per mille and in Silesia-Moravia was 2.7 per mille. The number of stillbirths per 1 000 total decreased to 3.5 (from 3.8 in in 2005), above the Czech Republic average of 2,8. In the region, 12 657 inhabitants died in 2006, a crude mortality rate of 10.1 per1 000 inhabitants, close to the overall Czech value of 10.2 (IHIS, 2007).

2.1.2. Air pollution Emissions

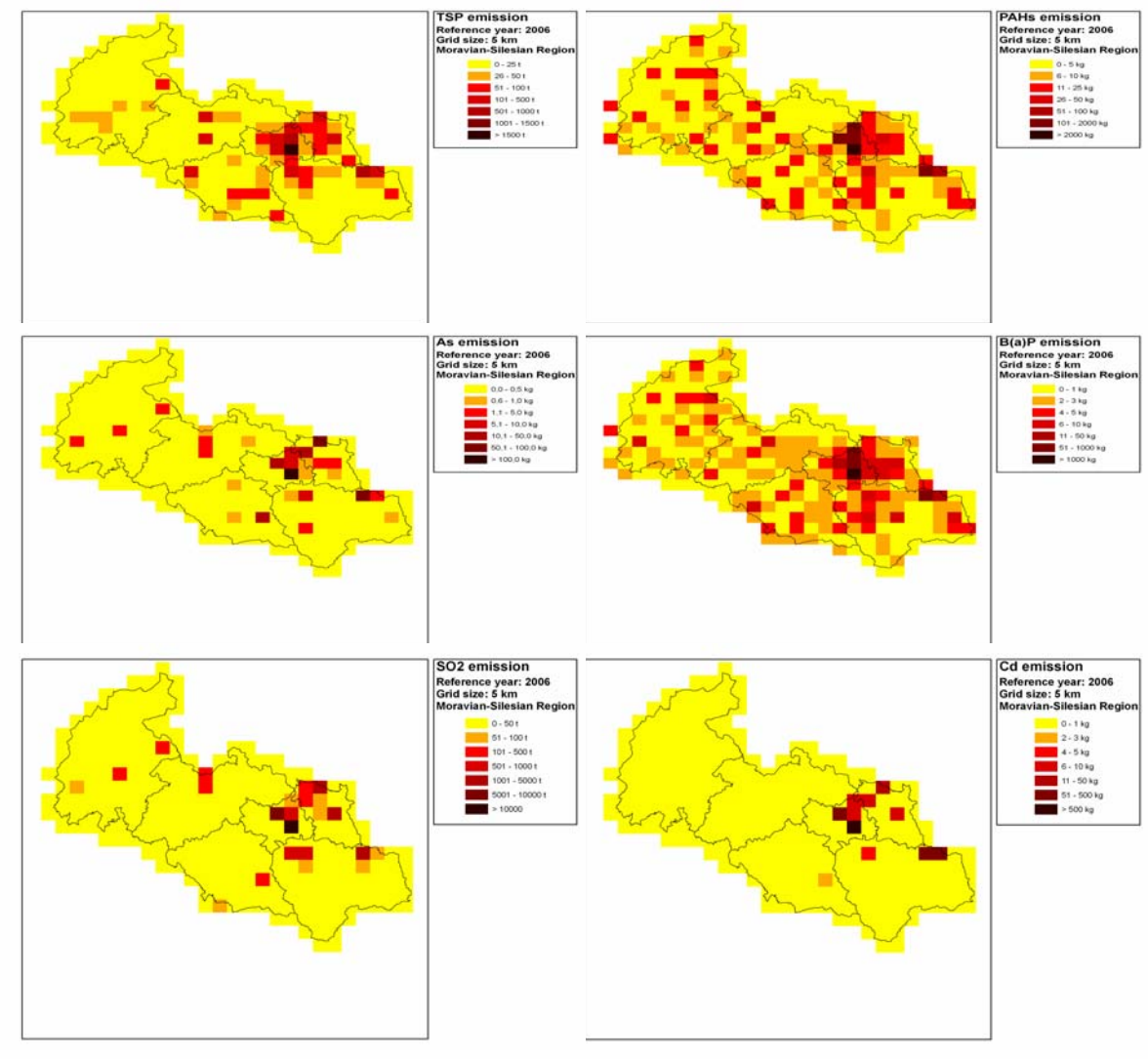


Figure* 2.1 *Emission fluxes from of TSP, PAH, As, BaP, SO2 and Cd in the Moravian-Silesian region, 2006.

Figure 2.1 presents emissions of selected pollutants on a 5 km grid in Moravia-Silesia in 2006. The highest emission fluxes correspond to areas with the highest concentration of heavy industry in Ostrava, Karviná, Třinec and Bohumín.

2.1.3. Ambient air quality

The Moravian-Silesian region is the most polluted region in the Czech Republic, and is amongst Europe's most polluted areas (Figures 2.2). The poor quality of ambient air in Moravia-Silesia results from the high concentration of heavy industry in densely populated cities (Ostrava, Karviná, Havířov, Český Těšín and Třinec) and specific climatic conditions influenced by orography support frequent inversions with restricted dispersion. In 1970's and 1980's PM concentrations were extremely high, and whilst during the 1990's PM levels decreased (Figure 2.3), during several recent winters an increasing trend of PM concentrations in the Ostrava-Karviná area has been recorded (Figure 2.4). PM_{10} ambient air quality thresholds are exceeded in the long–term and regularly at almost all monitoring stations in Moravia-Silesia. In 2006 PM_{10} concentrations were monitored in 25 locations. Exceedance of the PM_{10} 24-hour limit value occurred most frequently at stations in the districts

Karviná and Ostrava-město, and in several parts of the Frýdek-Místek, Nový Jičín and Opava districts. Exceedances of the PM_{10} annual limit value (40 µg.m^3-3) were also recorded mostly in the above districts.

The localities which measured $PM_{2.5}$ in 2006 in Moravia-Silesia rank among the localities with the highest measured concentrations in the Czech Republic. In Věřnovice the annual average concentration was 50.4 µg.m^{-3}, in Ostrava-Přívoz 44 µg.m^3, in Ostrava-Zábřeh 35.1 µg.m^3 and in Ostrava-Poruba 31.4 µg.m^3. It is evident that all localities would markedly exceed the proposed limit value.

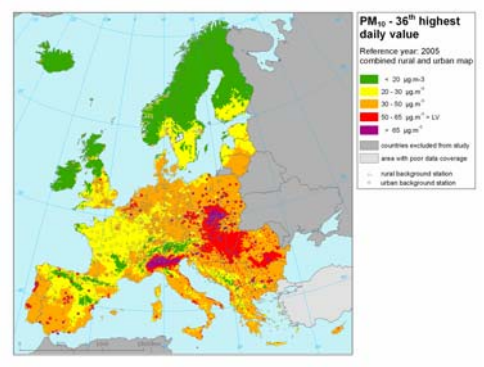


Figure 2.2 Combined rural and urban concentration map of PM10 – 36th maximum daily average value, year 2005. (EEA, 2008)

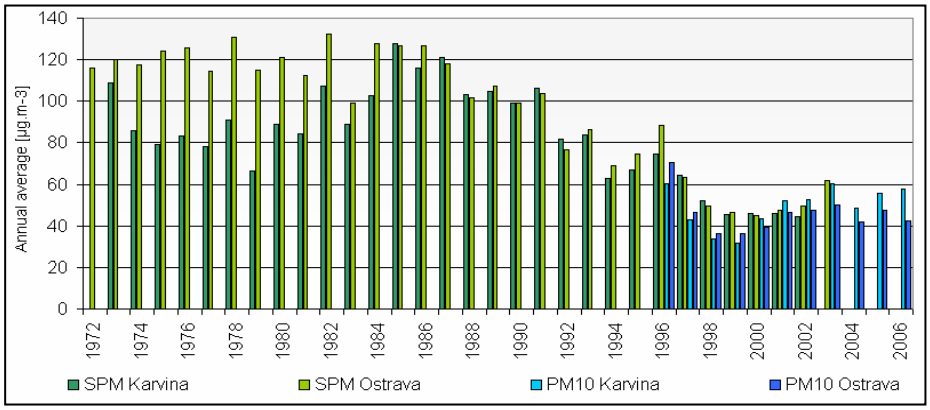


Figure 2.3 Annual PM averages, 1972-2006

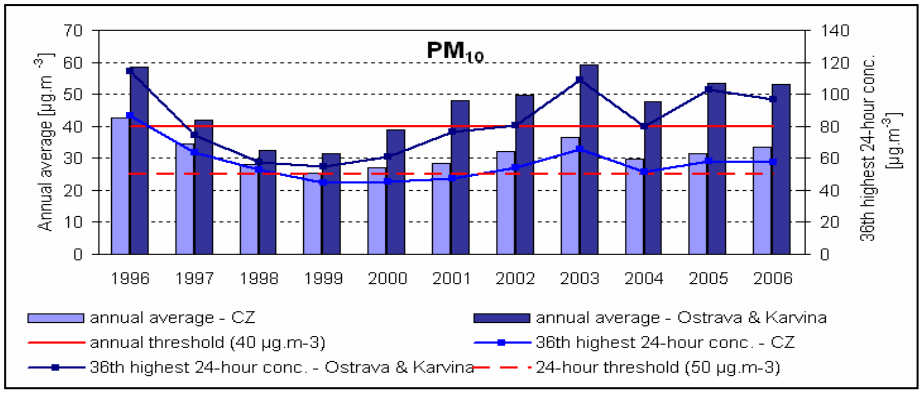


Figure 2.4 Annual PM characteristics comparing the situation in Ostrava and Karvina with the in the Czech Republic as a whole, 1996-2006

2.1.4. Population exposure and health impact in Moravia-Silesia

Population exposure to PM_{10} annual average concentration in 2006 for depicted concentration classes and for the Moravian-Silesian region (MSR) is presented in Table 2.1.

Table* 2.1 *Population exposure to PM10 annual average concentration in 2006 in MSR

Grid size [km x km]		*Concentration classes [µg.m^{-3}]*						*Population weighted PM_{10} annual average concentrationl*
		0-10	*10-20*	*20-30*	*30-40*	*40-45*	*>45*	
1x1	number of population	**0**	**12 911**	**189 653**	**717 067**	**320 565**	**620 920**	41.2 µg.m^{-3}
	% of population	**0.0%**	**0.7%**	**10.2%**	**38.5%**	**17.2%**	**33.4%**	
10x10	number of population	**0**	**12 007**	**234 325**	**805,756**	**228 339**	**580 689**	39.9 µg.m^{-3}
	% of population	**0.0%**	**0.6%**	**12.6%**	**43.3%**	**12.3%**	**31.2%**	

These estimates are based on a detailed mapping of the PM_{10} air quality indicator for the region (Figure 2.5) and are prepared as an extract from the PM_{10} annual average maps for the Czech Republic (CHMI, 2007). Preparation of these maps followed the same methodology as presented in Horalek et al., 2007.

Population exposure analysis was done at a 10x10 and at a 1x1 km grid size to estimate influence of mapping grid size on estimated population exposure. The estimated population exposure is higher for the finer grid. With increasing grid size the estimates of population exposure are evidently more biased. Within a grid cell of the larger size the areas with high concentrations gradients are 'smoothed' by cleaner areas which have lower population density, thus reducing the spatial peaks.

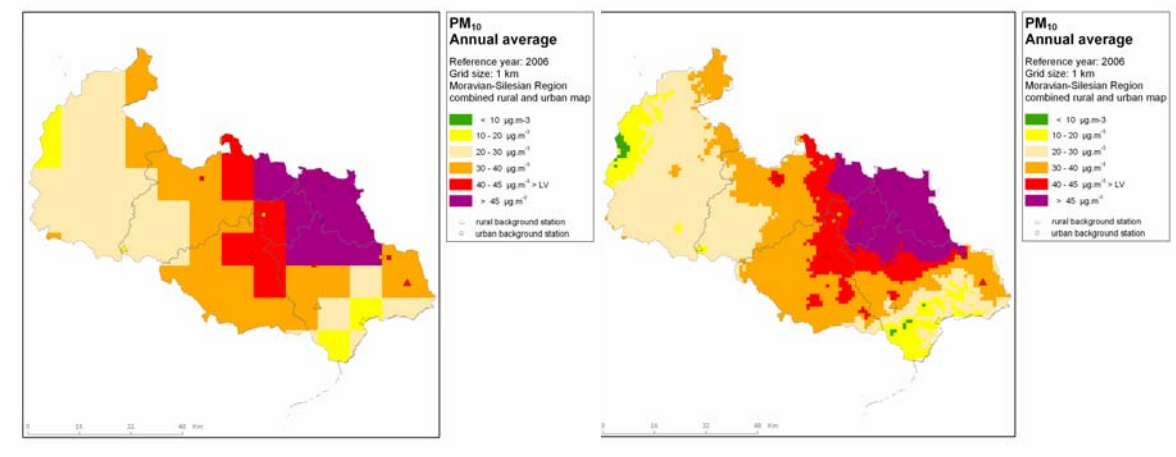


Figure 2.5 PM10 annual average concentration fields in Moravia-Silesia assessed in 10(left) and 1(right) km grid size, 2006

In the health impact assessment for the analysed region the number of premature deaths attributable to long-term exposure to PM_{10} was estimated using a similar approach as described in Horálek et al. (2007) and (2008). A relative risk of 4.3% per 10 µg.m-3 PM_{10} for total mortality (excluding violent deaths, adults 30 years and older) was used (Künzli et al., 2000). Total mortality rate for the region per 1000 inhabitants was 10.1 for the year 2006 according to the Institute of Health Information and Statistics of the Czech Republic (IHIS, 2007). An assumed uniform non-anthropogenic background concentration of 5 µg/m3 was subtracted.

Mortality, *mi*, in a grid cell *i* attributable to PM_{10} concentration, *ci*, over 'non-anthropogenic' background concentration of PM_{10}, *cb,* was calculated according to (1) (Ostro, 2006):

$$mi = (ci-cb)*0.1*RR*MR*0.001*popi \quad (1)$$

MR mortality rate per 1 000 inhabitants (10.1 for the Moravian-Silesian region);
RR relative risk 4.31(2.6–6. 1) % to the overall mortality (Künzli et al., 2000) per 10 $\mu g.m^{-3}$
c_b 'non-anthropogenic' background concentration of PM_{10} (5 or 10 $\mu g.m^{-3}$);
pop_i population in a grid cell *i.*

The number of premature deaths NDP per million attributable to PM_{10}, calculated according to eq. (2)

$$NPD = \frac{1.10^6 \cdot \sum_{i=1}^{N} m_i}{\sum_{i=1}^{N} pop_i} \quad (2)$$

for the analysed region for 2006 is presented in Table 2.2.

Table* 2.2 *Number of premature death per million in Moravia-Silesia attributable to PM10

PM_{10} 'nonatropogenic' background	Grid size *[km]*	
	1x1	10x10
5 $\mu g.m^{-3}$	**1573.3**	**1514.8**
CI	951.3-2231.9	915.9 – 2148.9
10 $\mu g.m^{-3}$	**1356.1**	**1297.72**
CI	820.01-1923.9	784.6 – 1840.9

The assessment of air pollution impact on health of the Moravia-Silesian population, and especially for the Ostrava city, carried out by the Institute of public health estimated a shortening of average live expectancy of about 2 years, whereas for the Czech Republic as a whole the shortening is about 1 year (Šebáková et al., 2008). Life expectancy in this region: men 71.86 (73,45 in the Czech Republic) years (a difference 1.6 years) ; women 78.84 (79.67 in Czech Republic).

The individual lifetime cancer risk (ILCR) from exposure to carcinogenic pollutants is higher than 1x10-6 for current concentrations of arsenic, benzene and namely PAHs. The highest morbidity due to acute respiratory illnesses in children up to 6 years has been reported during recent years in the Ostrava region.

2.2. Micro-scale intra-urban differences in air quality: Assessment of the potential health impact in London and Athens.

2.2.1. Introduction

Whilst numerical predictions from air quality modelling of annual average pollutant concentrations are a typical input to health impact assessment, the substantially varying physical characteristics within cities are not usually accurately resolved by this modelling. The question is whether this lack of resolution has significance when estimating urban health impact, or whether a health impact based on urban background concentrations is a close enough approximation.

This study will present results from an assessment of two different locations within Athens and one location in central London, using a regional-urban-local model cascade, namely the EMEP, OFIS and MIMO models. The main objectives of this activity are the following:

- To establish the differences in assessing the health impacts of air pollution at a city scale compared to a neighbourhood scale.
- To gain an insight of the effect of the intra urban differences in building geometry, meteorology, and local emissions on the prediction of pollutant concentration levels.

Urban background concentrations for the cities of London and Athens were calculated using the model OFIS, which utilises regional background concentration levels from the EMEP model. Concentration levels of selected pollutants at a microscale level in the selected locations in the city of Athens, were then calculated with the use of MIMO (Ehrhard et al., 2000) for the case of the city of Athens and ANSYS CFX (http://www.ansys.com) for the case of the city of London. Both are typical Computational Fluid Dynamics (CFD) models.

2.2.2. Study sites and outline of methodology

In the city of Athens, two representative locations with dimensions of approximately 400 m were selected. The first, located near the shore of Piraeus, represents a typical case of densely-built urban sea-front with multi-store buildings, numerous urban canyons and a large open area at the middle (Figure 2.6a). The second, centred around Patision and Fokionos Negri streets in downtown Athens, is an example of very densely built area with poor ventilation along its lateral boundaries and an orthogonal configuration of roads carrying a significant traffic load during most of the day (Figure 2.6b). For both areas computational grids were constructed based on 3-dimensional geometrical data of the building and road structure (Figure 2.7).

The selected area in central of London is in the financial district, the so-called “City”. Due to the complexity of the required mesh, the commercial ANSYS™ ICEM CFD 5.1 (http://www.ansys.com) mesh generating code was utilized, and an unstructured computational mesh of 2500000 cells was set up with sufficient refinement near buildings to resolve the important features of the flow (Figure 2.8).

For both city cases, 16 different wind directions were simulated. The average concentration levels within every individual wind direction were interpolated to obtain the average concentration levels for $PM_{2.5}$ and PM_{10} at a step of 1^o. These values were further interpolated to obtain results for a range of wind speeds between 1 ms^{-1} and 15 ms^{-1}. These results were then correlated with wind direction and speed from mesoscale model calculations to create an annual time series and to calculate the yearly average concentration. It should be noted that calculated concentration levels were averaged over the horizontal and vertical structure upto roof level in order to include potential impacts on those residing in the upper levels.

(a) (b)

Figure 2.6 The two locations selected in the case of the city of Athens: (a) shore area close to Piraeus port and (b) area located in the city centre

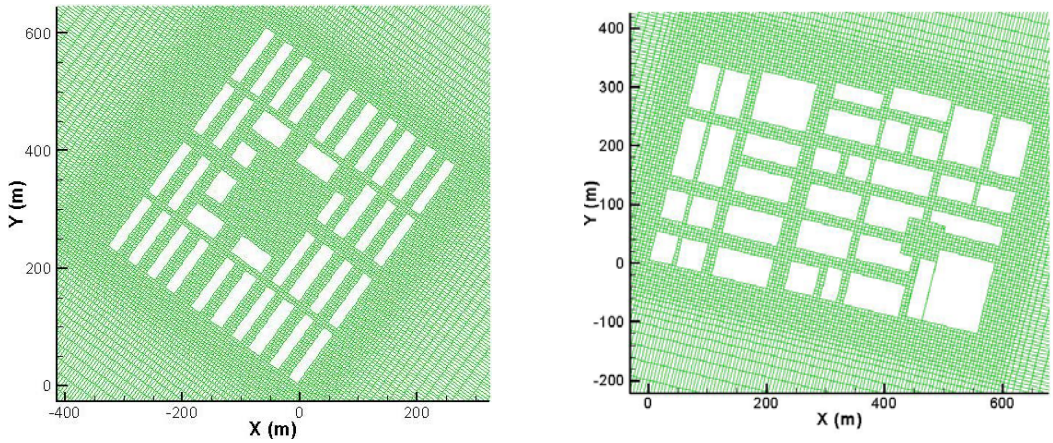
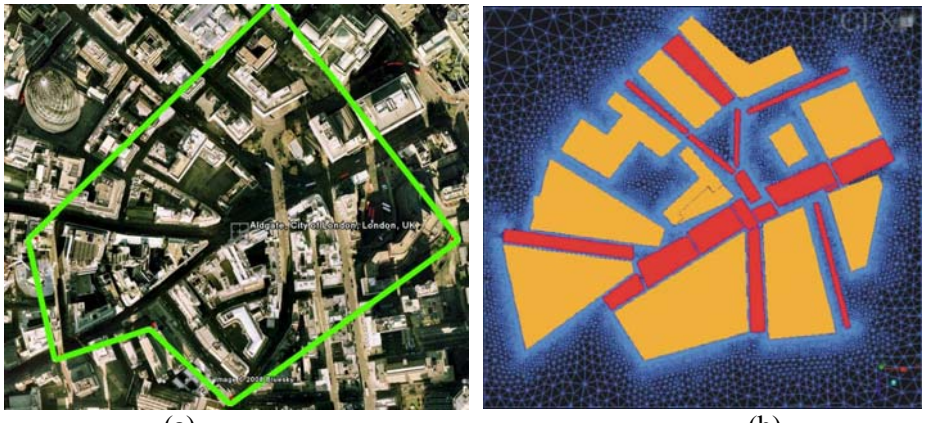


Figure 2.7 Computational domain and mesh for the two selected locations in the city of Athens



(a) (b)

Figure 2.8 (a) Bird' s eye view of the the selected urban area in central London and (b) computational domain and mesh

2.2.3. The results

Figures 2.9, 2.10, and 2.11 present the calculated concentration levels at a horizontal level of 3 m above the ground indicatively for two different wind directions, namely North and North - West. Numerical results for the predicted monthly concentration levels for all three cases considered are presented in Figure 2.12. The results shown in this figure include both the contribution of traffic emissions and other sources, as well as the contribution of the urban background. The calculated

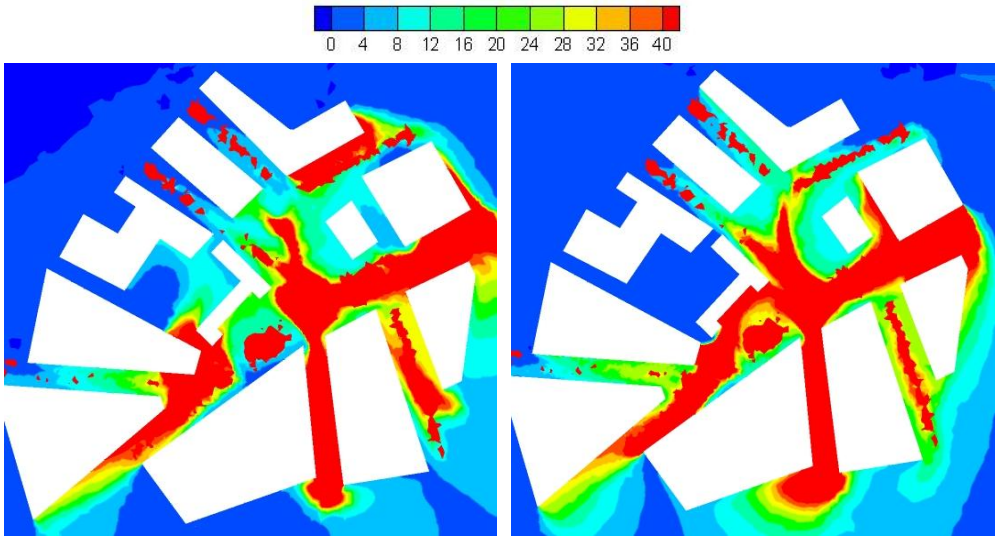


Figure 2.9 Calculated $PM_{2.5}$ concentration levels in the case an area in central London when the wind is approaching (a) from the North and (b) from the North West.

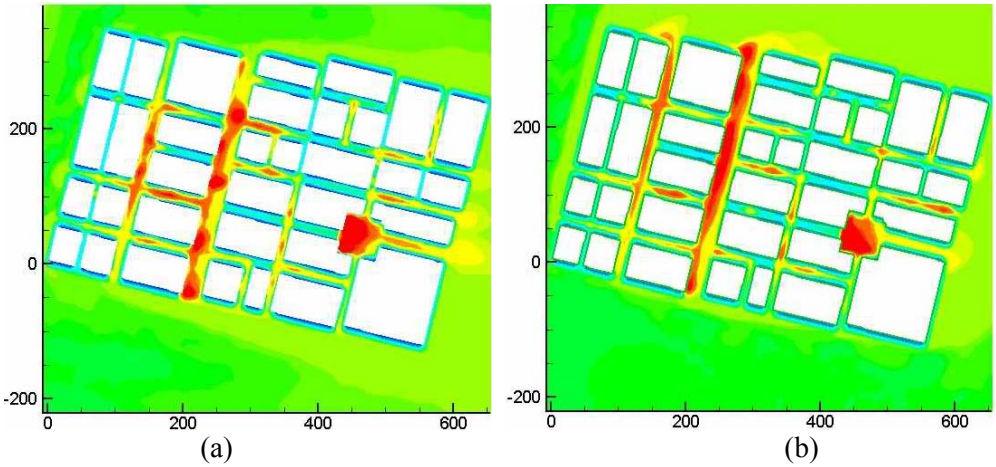


Figure 2.10 Calculated $PM_{2.5}$ concentration levels in the case of Patision Street area when the wind is approaching (a) from the North and (b) from the North West

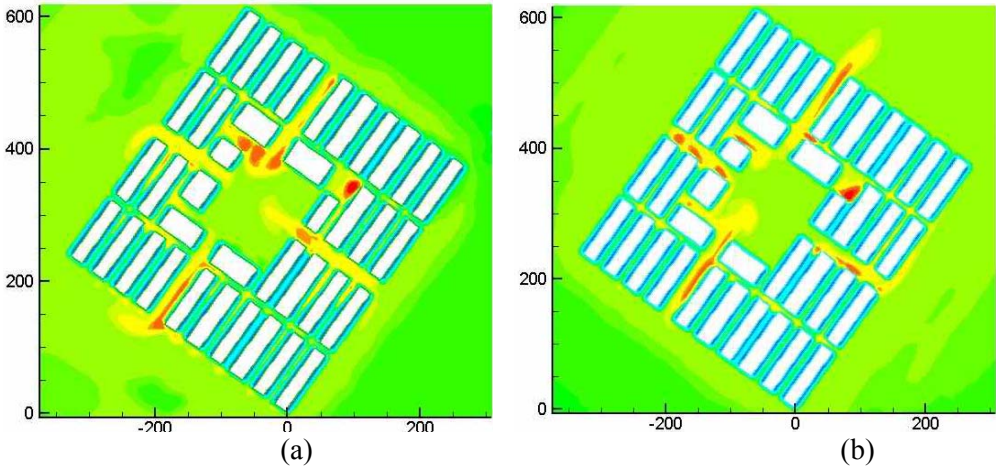


Figure 2.11 Calculated $PM_{2.5}$ concentration levels in the case of Piraeus harbour area when the wind is approaching (a) from the North and (b) from the North West

annual concentration levels reveal that both locations in Athens suffer from considerably higher $PM_{2.5}$ concentrations compared to the selected location in central London. The annual average concentration levels in Patision Street (39.7 ug m^{-3}) area are approximately 53% higher compared to London (18.71 ug m^{-3}), while those in the Piraeus harbour area (31.5 ug m^{-3}) are 40% higher compared to London.

These differences in the estimated concentration levels between the selected locations in the two cities can be attributed mainly to the fact that the two locations in Athens comprise of narrower streets which enforce entrapment phenomena combined and a slower rate of renewal of air inside the streets due to the lower average wind speed compared to the London case.

Furthermore, a comparison of the estimated annual concentration levels between the two selected location in the greater Athens area show considerably higher annual $PM_{2.5}$ concentrations in the case of the Patision Street area, of the order of 20%. Although the Piareus harbour area, should be affected more by shipping emissions, due to its close proximity to the sea shore the area is more efficiently ventilated.

2.2.4. Health impacts assessment- YOLL

The present analysis uses a risk estimate approach in terms of Relative Risk (RR - ratio of incidents observed at two different exposure levels) based on epidemiological studies. Quantification of health effects is usually expressed as the linking of two components; (i) RR, typically giving the rate of change in health endpoint per unit change in pollutant and (ii) the background rate of health effect in the population at risk. The result of the analysis is the quantification of the expected health burden in the target population, expressed in terms of the number of cases or Years of Life Lost (YOLL) attributable to the exposure (Krzyzanowski et al., 2002). YOLL is a meaningful and appropriate impact indicator for all risk factors, even those that are not observable as the cause of an individual death. In the frame of this analysis, the estimated loss of life expectancy is expressed in terms of the annual mean years of life lost (YOLL).

The present analysis is based on the RR from Künzli et al. (2000) and based on in Pope et al. (1995) (and its later extended follow-up (Pope et al., 2002)). Regarding YOLL in the present analysis the estimation of ExternE methodology is adopted (based on Pope et al. estimates).

$$R_{CM,PM_{10}} = 4.0 \cdot 10^{-4} \frac{YOLL}{year \cdot receptor \cdot \mu g / m^3}$$ for PM_{10} applicable to entire population.

Results for the potential annual average YOLL due to exposure to $PM_{2.5}$ concentrations are presented in figure 2.13, including the individual contributions of the urban background and of the other sources. For inhabitants in Peiraias harbour area, approximately 210 YOLL per year per 10,000 inhabitants can be attributed to exposure to $PM_{2.5}$, which translates to 19 months loss of life expectancy per capita for a life expectancy of 75 years. As regards the Patision street area, approximately 266 YOLL per 10,000 inhabitants can be attributed to exposure to $PM_{2.5}$ (24 months loss of life expectancy per capita for a life expectancy of 75 years). In central London, an estimated 125 YOLL are attributed to exposure to $PM_{2.5,}$ corresponding to an average of 11 months of life expectancy per capita for a life expectancy of 75 years. As the calculation of YOLL is directly related to air concentrations, reference to figure 2.12 indicates that the magnitude of health impact varies during the year.

The impacts described are applying hotspot situations to estimate the potential impact upon the population, and thus it is evident that locally elevated $PM_{2.5}$ concentrations have potential to significantly increase loss of life expectancy. It should be noted that the two areas are only a few kilometers apart. Therefore, when estimating the health effects of air pollution exposure is ideally based on the time individuals spend in specific areas.

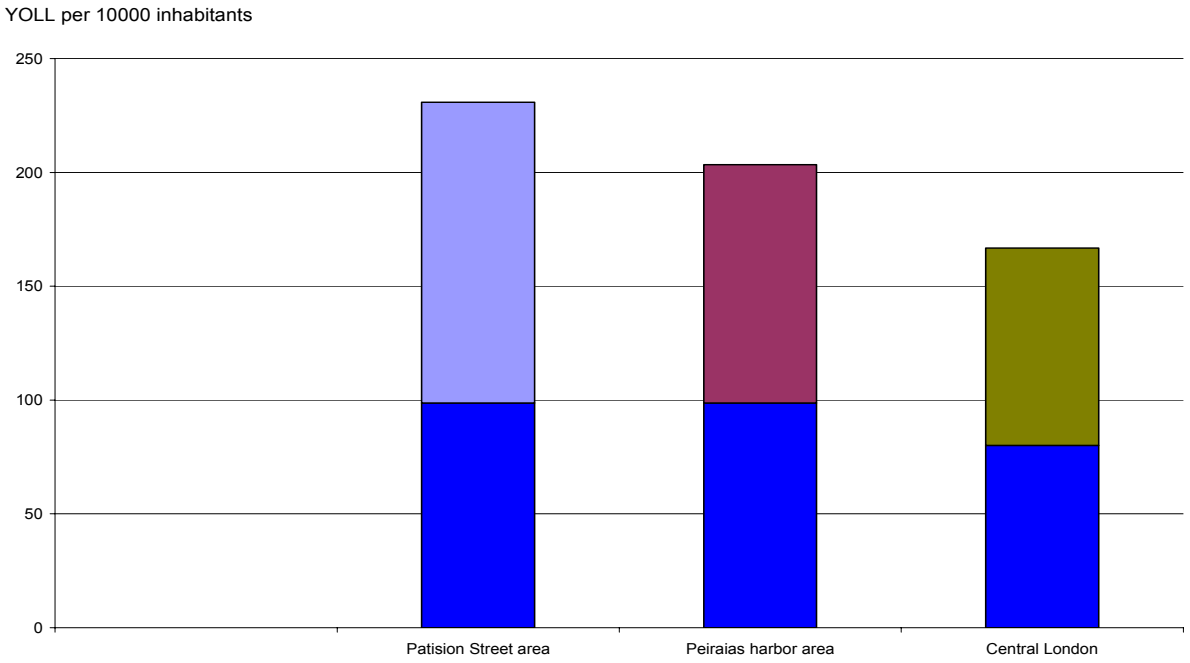


Figure 2.13 Annual mean potential YOLL for chronic mortality due to exposure to $PM_{2.5}$ for the three selected location in the cities of Athens and London per 10,000 inhabitants. Urban background contribution denoted by blue.

2.2.5. Health impacts assessment - morbidity

In this analysis the approach of Hurley et al. (2005) and ExternE (2005) is followed to estimate cases of bronchitis arising from air pollution exposure. Based on the study of Abbey et al. (1995), the approach takes into account remission rates in estimating background incidence rates. Hurley et al. (2005) derived a function as follows:

New cases of chronic bronchitis yr^{-1}
= 26.5 (95% CI -1.9, 54.1) per 10 $\mu g/m^3$ PM_{10} per 100,000 adults aged 27 years+.

Figure 2.14 presents the annual average number of chronic bronchitis cases due to exposure to $PM_{2.5}$ concentrations for the selected locations in London and Athens. Approaching 16 cases of chronic bronchitis per year can be attributed to exposure to $PM_{2.5}$ in Patision street area and approaching 14 in Piraeus harbor area compared to an estimated 11 cases in London.

Across Athens approximately 6.5 cases of chronic bronchitis per 10,000 inhabitants can be attributed to long term exposure to urban background levels. However, the total number of cases is considerably increased when considering the local contribution of the road traffic and other sources. Approximately 8.7 cases of chronic bronchitis can be attributed to long term exposure to $PM_{2.5}$ concentrations from road traffic and other sources in Patision street area per 10,000 inhabitants and 7 in the Piraeus harbour area. Correspondingly, approximately 5.3 of cases of chronic bronchitis can be attributed to long term exposure to urban background levels in London, per 10,000 inhabitants, and at the selected location an additional 5.7 cases due to long term exposure to locally elevated concentrations. The proviso is that the impacts described applying hotspot situations to estimate the potential impact upon the population.

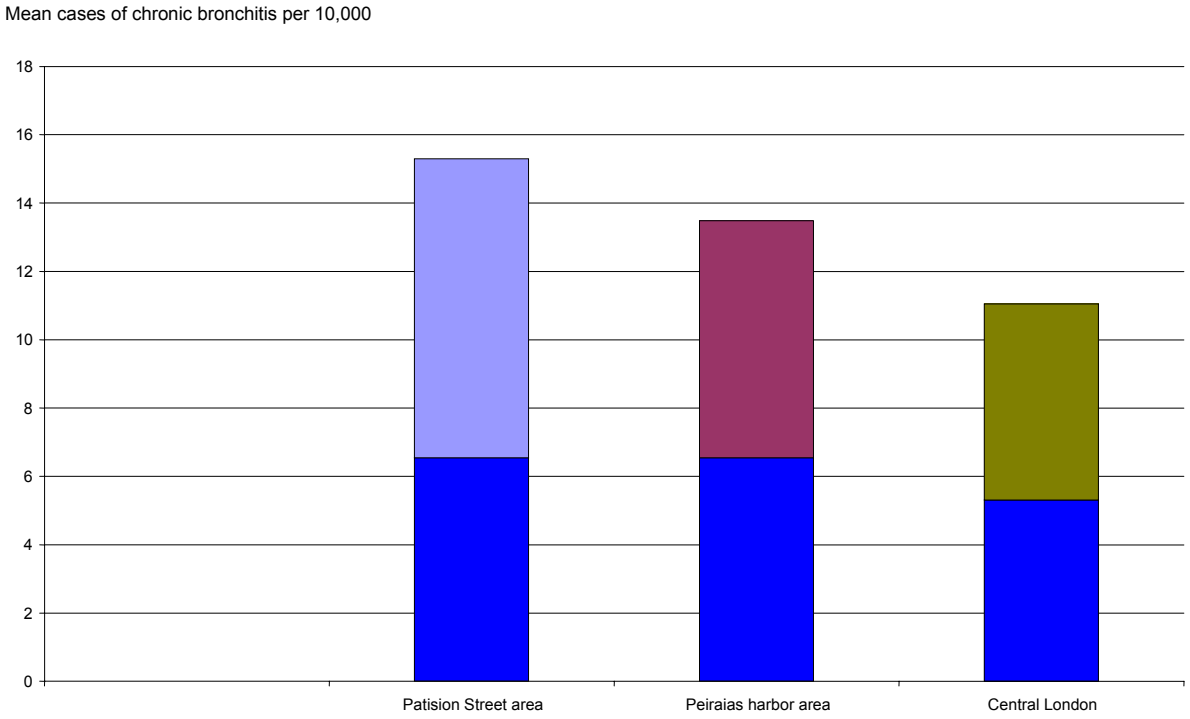


Figure 2.14 Annual mean cases of chronic bronchitis for the three selected locations in the cities of Athens and London per 10,000 inhabitants. Urban background contribution denoted by blue.

2.2.6. Synthesis and future steps

In urban areas air pollution concentrations clearly can vary significantly between selected locations. Whilst the potential importance of this for health impact has been illustrated by worst case analysis, the actual health impact upon the population will depend on the periods of time spent in hotspot locations, and by the proportion of the population thus affected. The need for accurate time exposure description of a population has been noted. This subject is dealt with in section 2.3 below.

Air concentrations may vary between locations depending on the geometrical characteristics of the main features. Ideally, when applying numerical models for assessment of health impacts, these effects should be resolved, although the tools for such resolution are often either unavailable or impractical for many practitioners. The implication of the two sites in Athens, and a point for future clarification, is that if emission density in the two areas were to be similar, and background meteorological ventilation are similar, that building structure and its effect on air flows may have a major effect on resulting air pollutant concentrations. It will then be important in deriving benefit from CFD modelling to clarify the relative importance of wind direction, building geometry, and emission density, and to explore the generalisation of such findings for European scale application.

2.3. The relevance of intra-urban movements for exposure: Oslo

2.3.1. Introduction

Oslo is situated at 60˚N 11˚E, near sea level and at the end of a 100 km long fjord which is surrounded by forest covered hills rising to 500m ASL. Normal average monthly temperature vary from– 5 ˚C in January to +17 ˚C in July. The air concentrations of PM_{10}, $PM_{2,5}$ and NO_2 can be high under specific meteorological conditions, particularly cold winter days, with low wind speeds and strong inversions. Peaks of PM_{10} also typically occur during dry periods in spring time due to suspension of road dust. The most important sources for PM_{10} are road traffic and domestic wood burning. Despite policies to discourage their use, still about 20 % of vehicles use studded tyres, whilst wood burning has been a traditional for heating in winter, a practice which continues. It has been estimated that in 2005, 45 % of Oslo inhabitants were exposed to PM concentrations in excess of National Target limit values, see table 2.3. Traffic was responsible for about 60% of these exceedances, with 15 -20 % due to wood burning for domestic heating (Slørdal et al., 2007)

The city of Oslo has around half a million inhabitants. The city is divided into 15 administrative districts, 5 of them forming the inner city and the rest the outer city. About 60 % of the working population lives in the outer city, whereas over 60% of workplaces are registered in the inner city (Statistics Oslo,2007). A number of people travel to work in Oslo from the surrounding areas; there are more than twice as many entering people entering Oslo to work each day than leaving. The overall picture is thus that the population is shifted towards the city centre during working hours. In this case study the sensitivity of exposure estimates to this population movement was considered. Comparison was made between estimated PM_{10} exposure considering home address and an exposure estimate taking into account the daily population movement within the city.

Table 2.3 National target and EU limit values (µg/m3) for PM_{10} with respect to human health

	24 hours	Year
National target (number of exceedances allowed)	50 (25 per year) 50 (7 per year*)	
EU (number of exceedances allowed)	50 (35 per year) 50 (7 per year*)	40 20 *

* Guidelines, should be reached by 1.1.2010

2.3.2. Concentration fields; spatial resolution and population weighting

The AirQUIS application (AirQUIS, 2007) was used to model both concentration fields and the concentrations at specific receptor points. The model grid for Oslo has a 1x1 km resolution, and covers an area of 18x22 km. Hourly PM_{10} concentrations calculated at receptor points were used to estimate within-day variation for individuals at different locations in the city. The first week of March 2005 was chosen randomly as a " case study week", to limit data to manageable quantities in this initial study. Plots of the average concentration fields for this week in March, and for the whole year, 2005 are given in figure 2.15.

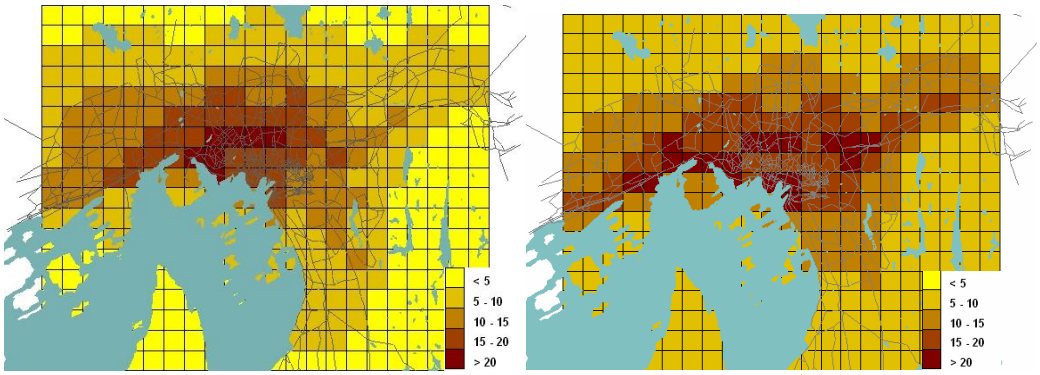


Figure 2.15 Maps of the average concentration of PM10 (µg/m3) in Oslo for the first week in March 2005 (left) and the whole year (right). Grid cell size: 1x1 km

We see that concentrations for the selected week were in general lower than the annual average, especially to the east. The averaged grid concentrations for the week and the year are 7.5 µg/m^3 and 10.0 µg/m^3 respectively (see table 2.5), whereas the population weighted averages are somewhat higher at 12.5 µg/m^{3}and 14.5 µg/m^3 respectively. In table 2.4, averages are also given for the inner city and the outer city separately. The differences seen between the grid average and the population weighted average concentration (+45% annually, +65% for the selected week) comes from the fact that the grid covers low populated and low concentration areas like forests and parts of the fjord. The differences in the averages when improving spatial resolution (division into inner and outer city) are very marked. Whilst it is expected that a finer grid might better reveal concentration differences, what is interesting is that population weighting in addition to improved resolution usually increased estimated experienced concentrations. This is also so in outer city areas where spatial resolution alone would otherwise decrease estimated concentrations.

Table 2.4 Population and spatially weighted mean concentrations for the first week in March and the year 2005, and percentage departure from baseline.

	Weekly Mean (µg/m^3)			Yearly Mean (µg/m^3)		
Averaging Method	Whole grid	Inner city	Outer city	Whole grid	Inner city	Outer city
Grid average conc.	7.5	18.2	6.9	10.0	19.9	9.4
departure from baseline	Baseline	+143%	-8%	Baseline	+99%	-6%
Pop. weighted conc.	12.4	19.0	9.9	14.5	19.8	12.5
departure from baseline	+65%	+153%	+32%	+45%	+98%	+25%

2.3.3. Statistics on population movements

Population data and statistics are available in details only for the administrative city of Oslo. The case study has therefore been restricted by assuming the Oslo boundaries closed and only the internal movements will be considered. The number of inhabitants (526 228) is initially distributed on the grid according to home addresses and this will be the reference method to calculate exposure. The change to this reference method will be to include some movement of the employed part of the population, assuming all other groups to be “at home”. In Oslo it is, for example, most common to have school or day-care / kindergarten in the proximity of the home.

Statistics on the relation of registered work place based on home address for the employed part of the population have been studied (Statistics Oslo). The conclusion is that one can estimate a shift of 20% of the outer city inhabitants to the city centre during work hours, this is 15 % of the total population. The others work in the neighbourhood of their home and we then assume their work exposure to be similar as at the home address. This is of course a crude simplification of the overall placements and movements in Oslo, especially in that movements from neighbouring administrative regions are not included. Thus, the estimates are for Oslo city and not for the greater Oslo urban area, accepting that these may be anticipated to contribute notable numbers of people who travel from low concentration home locations to inner city working locations each day. However, it does represent a first and solidly based approximation of the main shifts that occur within the administrative city for which population statistics were available, and for which across Europe figures may be most readily found.

2.3.4. Daily routines and individual movements

To personalize the travellers from the outer city to the inner city, 19 daily routine scenarios have been defined. Each "person" has an 8 hour work day with one hour travel to and one hour travel from work. The rest of the time is spent at the home location. Receptor points where placed at the different locations; work, home, travel to and travel from. The daily routine concentrations for one person were then created by picking values from the corresponding receptor points. Several receptor points were used to trace the travel to and from work, which were averaged to give one concentration value for the hour of travel. No difference in the daily routine was included, making weekdays and weekends the same.

The exact locations were chosen subjectively using best judgement and knowledge of the city to spread the home locations over the outer city, work places in the inner city and travel paths along the road net, so that a variety of possible placements would be represented. The resulting averages for our case study week are given for the 19 persons in table 2.5. In this, it should be noted that the receptor point values might vary from the grid cell values, depending on precise proximity to sources, such as roads.
For each column in the table the max values and minimum values are marked in pink and green respectively. The averages for each column are given in the last row, and person 12 is rather close to this average. Person 5 has as much as 159% increase of the weekly mean with her daily routine compared to home, where as person 6 has a decrease of 12 %. Person 6 has a very high home receptor point concentration which is rather different from the value in the corresponding grid square. This is because the home location is close to a major road and explains as well why there is a decrease in the average for the daily routine compared to the home concentrations. The comparison of the daily routine to the home concentrations for the week in March are plotted for person 5 and 6 in figure 2.16.

The plots show a fairly large variability from day to day. Some days the home and daily routine concentrations are quite similar, but other days the daily routine gives hourly values more than 100 times larger, thus indicating the potential for repeated acute exposure. Person 6 has the max hourly value during the first 24 hours, whereas person 5 has the max value on the 5th day (hours 96-120). The daily routine concentrations of person 5, 6 and 12 are compared in figure 2.17 for two time periods. To the left, the first 48 hours of the week, the three persons have similar exposure, at least for most of the period. In the plot to the right however the situation is different and person 5 has some high peaks with concentrations far above the two others. Representation of actual movements clearly has a major influence on estimated departure from the baseline case of exposure described solely by home location.

Table 2.5 The average PM_{10} concentrations the 19 persons are exposed to.

"Person"	Averaged Concentration, with daily routine ($\mu g/m^3$)	Averaged concentration at home receptor point ($\mu g/m^3$)	Difference between home and daily routine ($\mu g/m^3$)	Difference between home and daily routine (%)	Grid concentration for home, when different from receptor point ($\mu g/m^3$)
1	14.8	8.9	5.9	66	
2	10.5	6.1	4.4	73	
3	10.3	5.0	5.4	107	
4	12.9	9.8	3.1	31	
5	20.3	7.8	12.4	159	
6	14.5	16.5	-2.0	-12	9.0
7	12.6	9.1	3.5	37	
8	13.4	9.5	3.9	41	
9	14.0	12.7	1.3	10	
10	16.8	13.6	3.2	24	11.6
11	17.1	14.9	2.0	15	
12	12.5	8.6	3.9	46	
13	13.5	7.9	5.6	72	
15	11.3	5.1	6.2	122	
15	13.9	8.5	5.4	64	
16	24.6	17.3	7.3	42	
17	13.5	10.7	2.8	26	
18	10.1	5.6	4.5	82	
19	17.2	18.9	-1.7	-9	
Average	14.4	10.3	4.1	52 (39)*	

*If the average for the 19 persons is used (4.1/10.3)

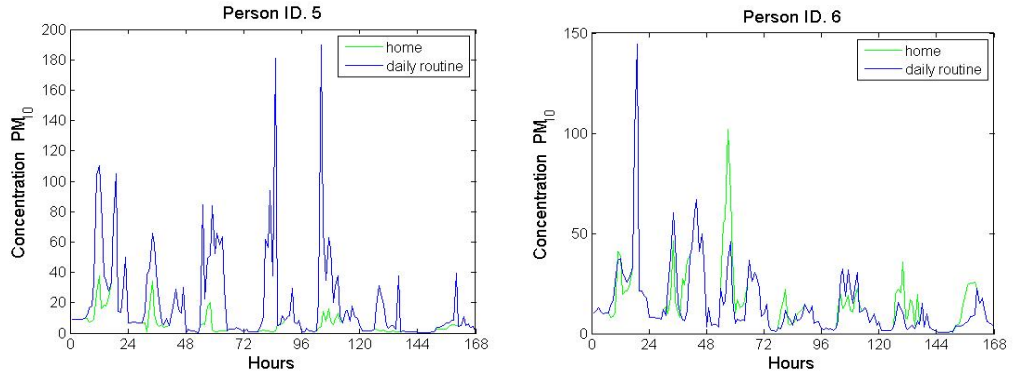


Figure 2.16 Concentrations at home compared to concentrations during the daily routine

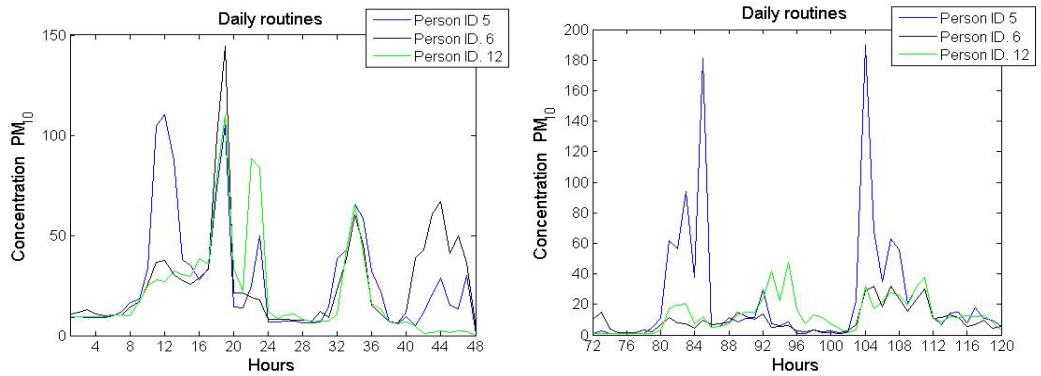


Figure 2.17 Comparison of the daily routine concentrations for person 5 6 and 12

2.3.5. Influence of movements on total population average exposure

The moving group of 20% of the population in the outer city has been given four possible exposure outcomes to estimate the influence of the movement on the population weighted mean for the entire Oslo population. The exposure increase given to the group where based on the results from the 19 persons and on the grid averages. The four outcomes were:

1. Assume a 50 % increase in average exposure concentration compared to their home grid concentration; 50 % approximates the average increase for the 19 scenarios.
2. Assume a 100 % increase in average exposure concentration compared to their home grid concentration; 100% is a high end increase amongst the 19 scenarios.
3. Assume an increase of 14.4 $\mu g/m^3$; the average increase for the 19 person scenarios
4. Assume the inner city daily average value (19.0 $\mu g/m^3$) instead of the home address value.

The effects upon population estimates are given in table 2.6.

Table .2.6 Percentage of the population in each concentration interval depending on how the moving 20% of the outer city is treated. Including also the final population weighted mean

Concentration interval ($\mu g/m^3$)	0-5	5-10	10-15	15-20	20-25	Mean ($\mu g/m^3$)
Initial	5	40	21	22	12	12.4
With 50% increase	4	36	24	23	13	13.2
With 100% increase	4	35	22	25	14	13.9
Concentration of 14.4 ($\mu g/m^3$)	4	32	32	20	12	13.1
Concentration of 19.0 ($\mu g/m^3$)	4	32	18	34	12	13.8

2.3.6. Discussion of the results

The conclusion is that moving a part of the city population to the centre increases the general exposure level. An increase from 12 % to 14 % of the population exposed to concentrations above 20 µg/m3 (in table 2.7) means that more than 10 000 would enter the highest exposed group. Even if the change is modest when looking at the whole of population for the week average the personal variations are high. Some high hourly values, typically when travelling along the roads, might increase the daily means such that we could expect to see a large increase in the number of days above the limit values when considering one year, even if the annual mean would have a limited increase.

This, however, likely understates the actual exposure status. Population statistics were only for within-city movements, and details were unavailable for those commuting into the administrative city region. In gross terms, it is known that over 368,000 people have a working place in Oslo city, and that over 120,000 travel to these working places each day from outside the city boundaries. Thus, one third of Oslo's working population could not be represented in this study, and Oslo's daytime population will be ca. 20% higher than the resident population at around 600,000. Furthermore, these people are largely commuting from low air concentration low population regions, and may be anticipated o be employed in central areas of Oslo, rather than outer city locations. Using the 19 person scenarios to

estimate exposure for the commuter group is uncertain, precisely because none of the 19 are long distance commuters. At the high end, person '5' (see table 2.7) experiences the greatest increases (almost 160% rise) travelling from the forest edge on the city limits to the centre. Somewhat lower, but still substantial, the average increase in experienced air concentrations amongst the 19 person scenarios having home addresses in low concentration sites is approximately 75%. Thus, an increase of 100% for commuters may be reasonable. Expressed as an average for the city population, this would mean a 20% increase in experienced air concentrations, but the question must be asked whether such averaging really expresses potential health impact when the impact is actually far greater upon a clearly identifiable group. Such reasoning also applies to others e.g. the relatively immobile, such as children and retired people. The next stages of this work in Oslo would be to extend the region for which population movements can be traced, and to identify the groups of people which can be identified and for which certain movement patterns with commensurate changes to exposure can be recognised. The work to date has indicated that scaling is possible in order to gain better insight into actual exposure, and that the numbers affected by large increases may be substantial both in absolute and percentage terms.

3. PM2.5: Preliminary assessment of the health impact in Europe

3.1. Estimating the $PM_{2.5}/PM_{10}$ ratio

Estimates of the ratio are derived from co-located $PM_{2.5}$ and PM_{10} measurements extracted from AirBase for the period 2004-2006, this containing air quality information submitted by 35 European countries following the *Exchange of Information decision* (EU, 1997). The EoI requires submission of validated data, and hence PM_{10} and $PM_{2.5}$ data have been used without further processing. Similarly, it is thus assumed that PM data has been corrected for non-reference methods when needed. Supporting information, however, is often incomplete, unavailable or not up-to-date, and this lack of information hampers intercomparison of results. Conclusions regarding $PM_{2.5}/PM_{10}$ relation should be handled with caution in light of this uncertainty.

Based on AirBase information, data from 233 stations and 437 annual time series fulfilling the following criteria were selected:

- -) The PM measurements should be co-located.
- -) data coverage of 75%: at least 274 days p.a. valid daily values for both PM_{10} and $PM_{2.5}$
- -) annual correlation between co-located $PM_{2.5}$ and PM_{10} daily averages at least 0.7. Correlations show no interannual variation (Horalek *et al.,* 2008) and are on average 0.86 – 0.88. Lower correlations (R=0.81) are observed at traffic stations related to direct PM_{10} emissions (e.g. tyre and brake wear, resuspension, winter sanding). When the correlation is < 0.7 it is assumed the data is unrepresentative, and it is excluded.

Additionally, four time series at two traffic stations (in Iceland and France) were excluded as unrealistic ratios larger than one were observed. The $PM_{10}/PM_{2.5}$ ratio is calculated as the slope of a linear regression of daily concentrations, with averaging over days with simultaneous PM_{10} and $PM_{2.5}$ measurements.

In Figure 3.1 the annual mean concentrations of PM_{10} and $PM_{2.5}$ are given as function of the station classification. The wide spread in $PM_{2.5}$ / PM_{10} ratios indicates the ratio clearly depends on the type of station. Europe-wide, ratios are in the range of 0.4 - 0.8. At rural and urban stations they are 0.62-0.77, whilst traffic locations have a lower ratio (0.58) indicating a small contribution of locally emitted PM_{10} to the total. Results are presented in Table 3.1 and Figure 3.1 for four different European regions (Holarek *et al.* 2008) (countries without co-located PM_{10} and $PM_{2.5}$ data are printed in *italic*):

1. Northern Europe: Norway, Sweden, Finland, *Estonia, Lithuania, Latvia,* Denmark, *Iceland*
2. North-western Europe: United Kingdom, Ireland, *the Netherlands*, Belgium, *Luxembourg*, France north of 45 degrees latitude
3. Central and Eastern Europe: Germany, Poland, Czech Republic, Slovakia, Hungary, Austria, *Switzerland, Liechtenstein*
4. Southern Europe: France south of 45 degrees latitude, Portugal, Spain, *Andorra, Monaco,* Italy, *San Marino, Slovenia, Croatia, Greece, Cyprus, Malta, Albania, Bosnia Herzegovina,* Bulgaria, *Romania*.

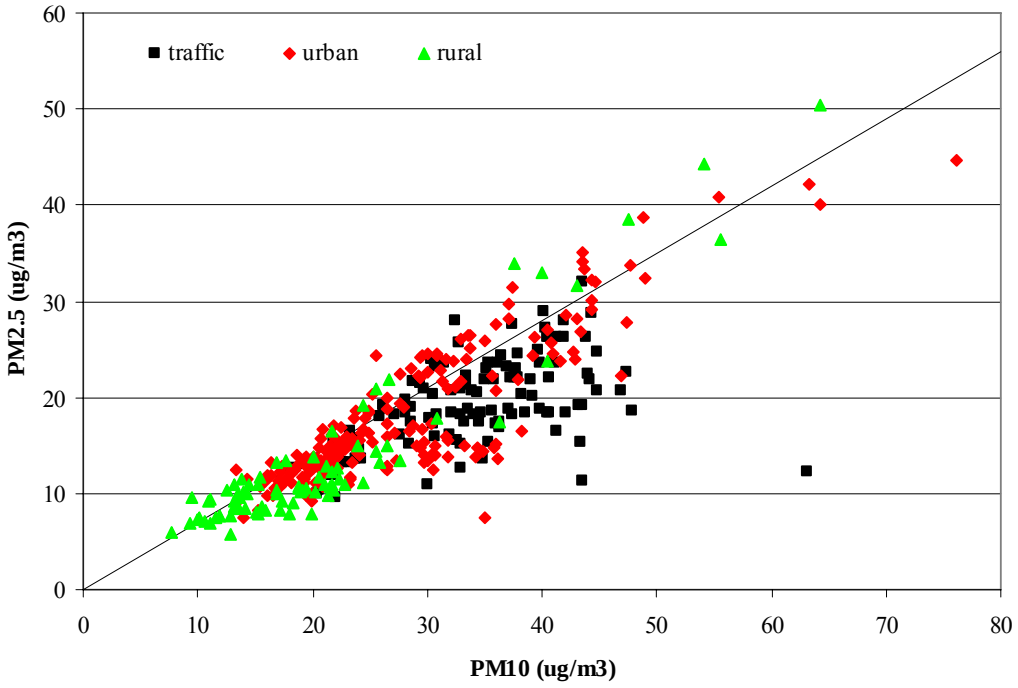


Figure 3.1 Annual mean concentrations of PM10 and PM2.5, period 2004-2006. The line corresponds with a PM10/PM2.5 ratio of 0.7.

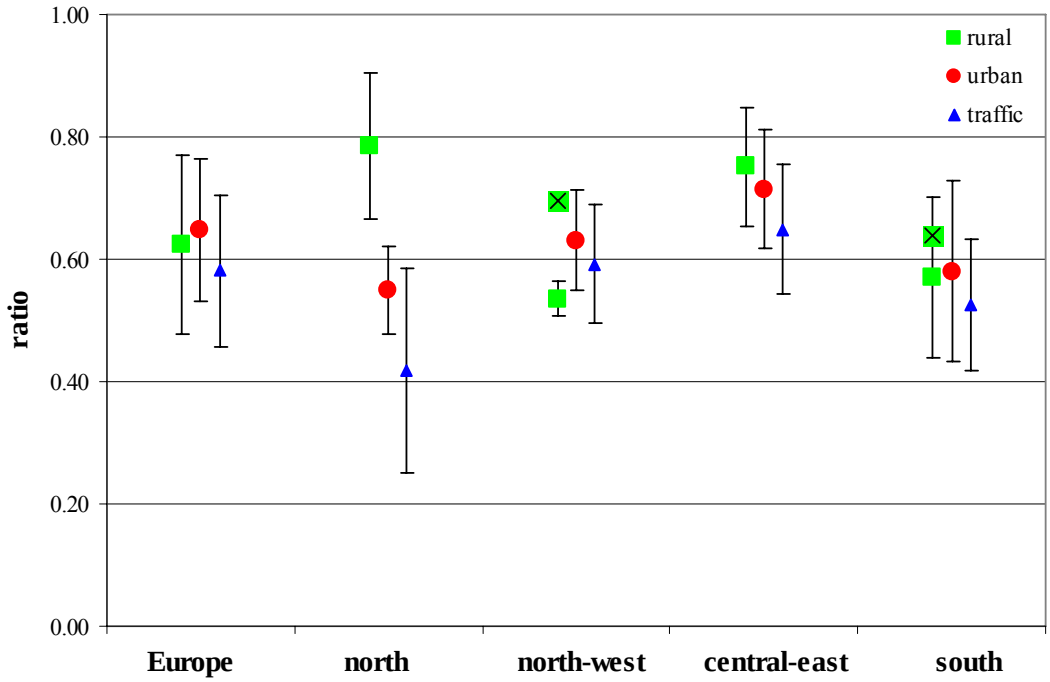


Figure 3.2 PM2.5 / PM10 ratios averaged for Europe and averaged per region and station type. The error bars indicate plus/minus one standard deviation. The marked rural dots correspond to the adjusted ratios, see text for explanation.

Table 3.1 PM2.5 / PM10 ratios and available number of time series as function of region and station type. The second value corresponds to the adjusted rural ratio (period 2004-2006)

	$PM_{2.5}$ / PM_{10} ratio			number of time series		
region	rural	urban	traffic	rural	urban	traffic
North	0.78	0.55	0.42	5	11	6
North-West	0.53/0.69	0.63	0.59	8	78	32
Central-East	0.75	0.71	0.65	20	73	41
South	0.57/0.64	0.58	0.53	48	39	38
Europe	0.62	0.65	0.58	81	201	117

In Northern, and in Central and Eastern regions, there is a tendency for lower ratios in the order rural-urban-traffic, indicating increased locally emitted coarse particles at urban/traffic sites. No such tendency is found in North-western and Southern Europe. Rural stations in North-Western Europe have surprisingly low ratio compared to those at urban and traffic sites in this region. The low numbers and geography of available time series may play a role here: 8 rural time series (6 UK and 2 Belgium against urban time series measured mostly in France (64 from the 78 total). In Southern Europe rural background stations are mostly located on the Iberian Peninsula (45 from the 48 time series). One possible explanation for a low rural ratio might be an important contribution of mineral (Sahara) dust.

The representativeness of the rural ratio in the North-West and South regions is questionable and, therefore, in a more detailed analysis the differences between a rural station and a close-by (less than 75 km) urban background station have been examined. Only 17 rural stations could be linked with urban background stations within the required distance. On average, the $PM_{2.5}/PM_{10}$ ratio at a rural background station is 10% larger than at the nearby located urban stations: rural ratios in these two regions have thus been adjusted by 10% (Figure 3.2).

3.2. Preparing $PM_{2.5}$ maps

The $PM_{10}/PM_{2.5}$ ratios have been applied to infer from the PM_{10} data a European $PM_{2.5}$ map. With the limited number of operational $PM_{2.5}$ measuring stations there is no alternative means at the moment to this approach. Measured PM_{10} concentrations are supplemented with data from the EMEP model, with corrections for altitude field, wind speed and surface solar radiation. Separate urban and rural PM_{10} concentration fields (Horalek *et al.*, 2008) were used, the final map being a population density weighted average. Three versions of the $PM_{2.5}$ maps have then been created, by applying the different European, regional, and country specific ratios to the PM_{10} maps. The final $PM_{2.5}$ map uses weighting from Horalek *et al.* (2008).

Independent validation of the maps is difficult with most available $PM_{2.5}$ monitoring data used to estimate the $PM_{2.5}/PM_{10}$ ratios. Only 15 $PM_{2.5}$ stations not co-located with PM_{10} stations, plus two EMEP stations not in AirBase, can be used. Figure 3.2 gives a scatter plot of the observed concentrations against the interpolated values in the corresponding 10x10 km cells, and table 3.2 gives summary statistics. Selected stations are all rural or (sub)urban background stations; traffic or industrial hot spot locations are not resolved in the interpolated maps. The procedure seems to smooth the monitoring data: at low levels (below 20 $\mu g/m^3$) there is some overestimation whereas higher levels are underestimated. Differences between the three approaches are not large. The European ratios give the worst and the country ratios the best fit. A map based on country ratios is hampered by specific ratios being unavailable for all countries. Therefore, regional ratios were used. The final $PM_{2.5}$ concentration map is given in Figure 3.3.

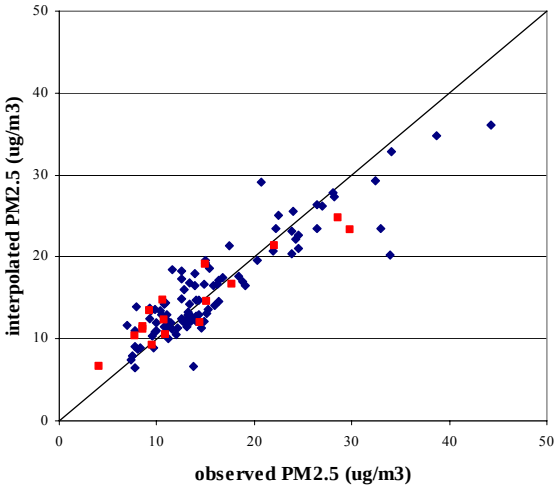


Figure 3.2. Comparison of observed and interpolated $PM_{2.5}$ concentrations. The blue dots corresponds to stations used in the estimation of the concentration ratios; the red dots correspond to additional monitoring stations.

Population and area weighted averages are compared in Table 3.3 alongside average PM_{10} concentrations. The $PM_{2..5}/PM_{10}$ ratio is about 0.6-07 but varies by country. Differences in concentrations arising from the three ratios are relatively small - <5% for population weighted concentrations, and <10% for area weighted concentrations. At the country level differences are larger, at up to 20-30% (e.g. Portugal).For the EU27 greater differences are seen in population weighted *.v*. area weighted concentrations, the former being about 40% higher than the latter.

Table 3.2. Comparison between observed PM2.5 concentrations and the interpolated grid cell values using European, region and country specific PM2.5/PM10 ratios. Left -results for all stations; right- results for stations not included in the parametrisation

	europe	region	country	europe	region	country
RMS	3.71	3.22	3.11	2.65	3.03	2.50
Bias	0.30	-0.26	0.19	-0.42	-0.53	-0.59
R2	0.871	0.903	0.908	0.934	0.936	0.953
N	114	114	114	17	17	17

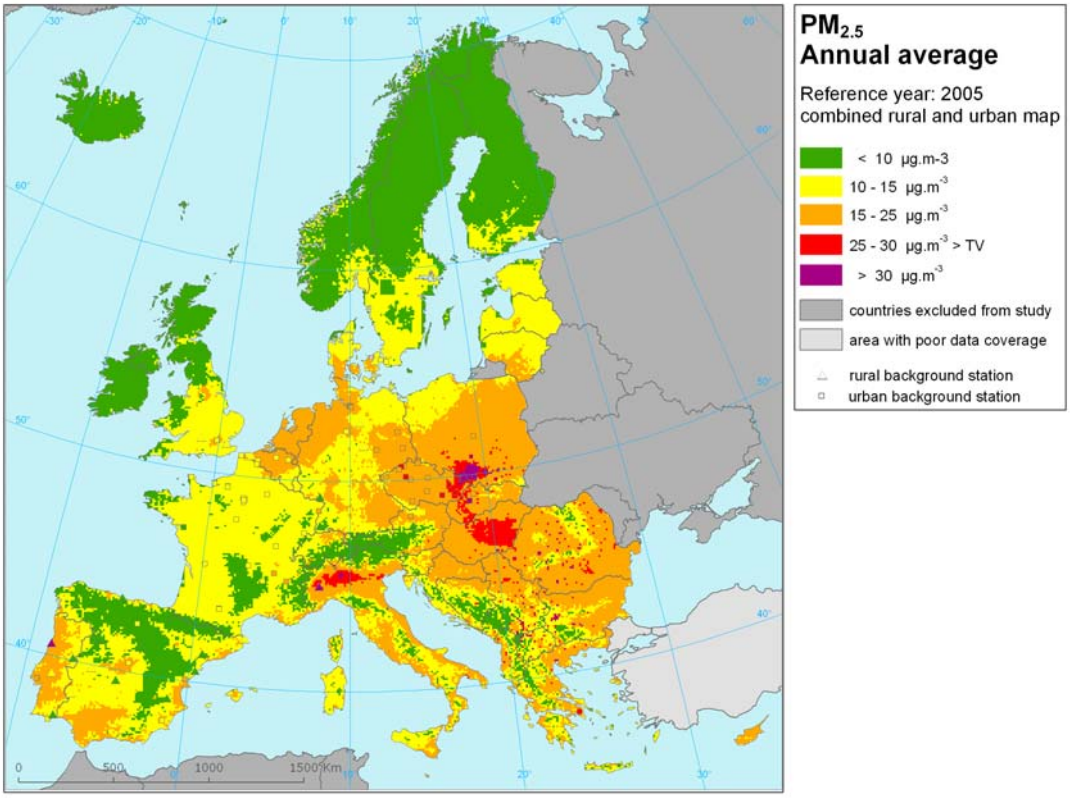


Figure 3.3 PM2.5 concentration map, annual average, year 2005. The map is based on the combination of scaled rural and urban PM10 maps using region specific PM2.5/PM10 ratios, see text for further details

3.3. Estimated exposure to $PM_{2.5}$

The recent Air Quality Directive (EC, 2008) sets health based standards and objectives for $PM_{2.5}$, with an annual mean concentration of 25 µg/m^3 as target value for 2010 and as a limit value for 2015. Additional objectives target exposure of the population to fine particles. These are set at the national level and are based on the average exposure indicator (AEI). The AEI is the averaged level measured at urban background location throughout the territory of a Member State and is to reflect population exposure.

The $PM_{2.5}$ maps constructed here have been used to explore these standards at a sub-national scale. The area of exceedance is simply estimated by the number of 10x10 km grid cells with concentrations above 25 µg/m^3. Typical hot spots, for example, heavily trafficked situations, might be neglected in this way. The three approaches generally give similar results (Figure 3.4), with no exceedance calculated in 15 Member States (local hot spots might not be reflected). In the remaining 12 MS the exceedance area ranges between 3% and over 30%. In Italy, Czech Republic, Slovakia and Hungary the three approaches give strongly varying estimates, the largest difference observed for Hungary where European specific ratios give an area of exceedance less than 1% compared to a 33% estimate from the other methods. This is due to concentrations around the target value over much of the country so that a relatively small difference in urban ratios (0.65, 0.71 and 0.72 for the European, region and country specific approaches) is sufficient to bring large areas above the target value. The averaged concentration in exceedance areas has little dependance on the chosen ratios.

Table 3.2 Population and area weighted PM10, and PM2.5 concentrations using three PM2.5/PM10 ratios

	population weighted				area weighted			
country	PM_{10}	PM_{25} (a)	PM_{25} (b)	pm_{25} (c)	PM_{10}	PM_{25} (a)	PM_{25} (b)	PM_{25} (c)
Austria	23.5	15.0	17.2	18.4	16.6	10.4	12.4	13.6
Belgium	28.9	18.6	18.7	14.7	25.6	16.2	17.0	13.3
Bulgaria	37.0	23.6	22.3	23.6	25.2	15.8	16.0	16.1
Cyprus	37.9	24.3	22.6	22.6	28.6	17.9	18.2	18.2
Czech Republic	31.5	20.1	23.1	23.5	27.5	17.3	20.5	20.4
Denmark	19.8	12.6	13.3	12.9	17.2	10.8	13.1	13.0
Estonia	16.4	10.4	10.8	10.8	13.8	8.7	10.8	10.8
Finland	13.3	8.4	9.1	8.5	10.0	6.2	7.8	7.1
France	19.1	12.2	12.3	13.2	16.7	10.5	11.2	12.4
Germany	22.1	14.1	16.0	15.3	20.0	12.6	14.8	14.2
Greece	34.8	22.3	20.8	20.8	22.7	14.2	14.4	14.4
Hungary	33.5	21.2	24.6	24.6	31.5	19.7	23.6	23.6
Ireland	11.5	7.3	7.6	7.6	8.6	5.4	5.9	5.9
Italy	32.8	21.0	19.6	23.8	24.3	15.3	15.2	18.0
Latvia	18.7	11.9	12.4	12.4	15.9	10.0	12.4	12.4
Lithuania	20.3	12.9	13.6	13.6	18.3	11.4	14.2	14.2
Luxembourg	18.4	11.7	12.1	12.1	17.4	10.9	11.9	11.9
Malta	36.5	23.6	21.3	21.3	32.6	21.0	19.2	19.2
Netherlands	29.1	18.7	18.7	18.7	27.4	17.5	18.1	18.1
Poland	30.5	19.5	22.2	20.8	24.0	15.1	17.9	17.7
Portugal	30.6	19.6	18.3	14.1	24.6	15.4	15.5	12.8
Romania	37.3	23.8	22.6	22.6	28.3	17.7	17.9	17.9
Slovakia	31.4	19.9	23.1	21.2	28.1	17.6	20.9	20.4
Slovenia	27.5	17.5	16.8	16.8	22.6	14.2	14.3	14.3
Spain	27.5	17.6	16.4	16.1	17.9	11.2	11.4	10.2
Sweden	15.0	9.5	10.4	11.0	9.9	6.2	7.7	8.2
United Kingdom	20.9	13.4	13.3	10.2	13.4	8.5	9.1	7.0
Albania	33.1	21.1	19.8	19.8	21.2	13.4	13.3	13.3
Andorra	16.9	10.9	10.1	10.1	9.3	5.9	5.7	5.7
Bosnia and Herzegovina	30.0	19.1	18.1	18.1	20.0	12.5	12.6	12.6
Croatia	30.7	19.5	18.7	18.7	24.4	15.3	15.5	15.5
Iceland	11.5	7.3	7.4	7.4	5.5	3.5	4.3	4.3
Lichtenstein	21.5	13.6	15.9	15.9	21.4	13.5	15.9	15.9
Norway	17.3	11.1	10.6	9.6	7.8	4.9	6.0	6.0
San Marino	27.2	17.3	16.5	16.5	27.2	17.3	16.5	16.5
Serbia and Montenegro	38.5	24.6	23.1	23.1	25.0	15.7	15.7	15.7
Switzerland	19.9	12.8	14.4	14.4	13.2	8.4	9.8	9.8
TFYR Macedonia	42.2	27.1	25.0	25.0	21.6	13.6	13.6	13.6
EU27	26.0	16.6	17.0	16.8	18.7	11.7	12.9	12.8
Total	26.2	16.8	17.1	16.9	17.9	11.2	12.3	12.3

(a) based on European specific ratios
(b) based on region specific ratios
(c) based on country specific ratios complemented with region specific ratios when country values are missing.

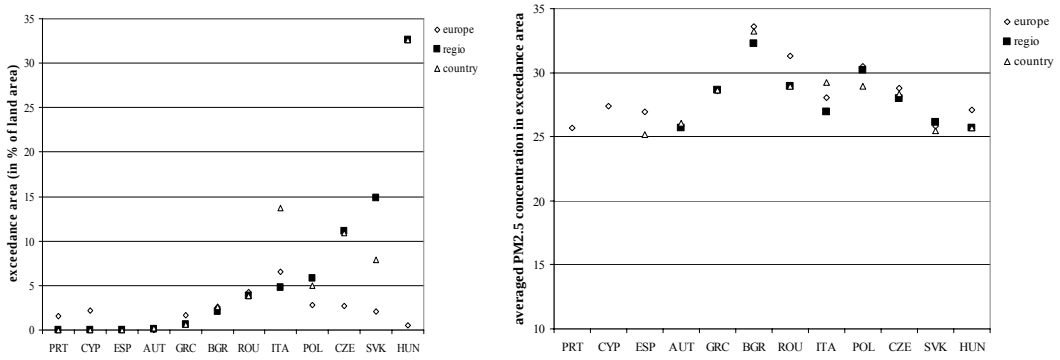


Figure 3.4 Area of exceedance (as fraction of land area) and PM2.5 concentration in the exceedance area estimated using differenmt PM2.5/PM10 concentration ratios. Only Member States where an exceedance is estimated by either one of the approaches are shown.

The AEI is calculated here as the concentration in the urban grid cells weighted according to the population in that cell. An urban cell is defined here as a grid cell with a population density of more than 500 inhabitants per km^2. The total population in urban cells amounts to be substantially lower than the urban population according to the UN World Urbanisation Prospects (UN, 2006): only 64% of the urban population (47% of the total population) in the EU27 is included in the AEI calculation. While covering such a small part of the urban population, it might be argued that the AEI does not reflect the population exposure as required by the directive. Therefore, a second calculation was made in which the urban population in mixed rural/urban cells was included in the AEI averaging procedure. In this way the covered urban fraction increased to 87% of the EU27 urban population (64% of the total population). However, it turned out that the differences in AEI caused by the two methods are much smaller than the differences caused by the choice of concentration ratios, see Figure 3.5. Irrespective of the calculation method, in 10 MS the AEI is in 2005 well above the obligation of 2015. In 5 MS the AEI is, depending on the calculation method, just below or above the level of 20 $\mu g/m^3$. In the other 12 MS the AEI is estimated to be well below the binding value of 20 $\mu g/m^3$.

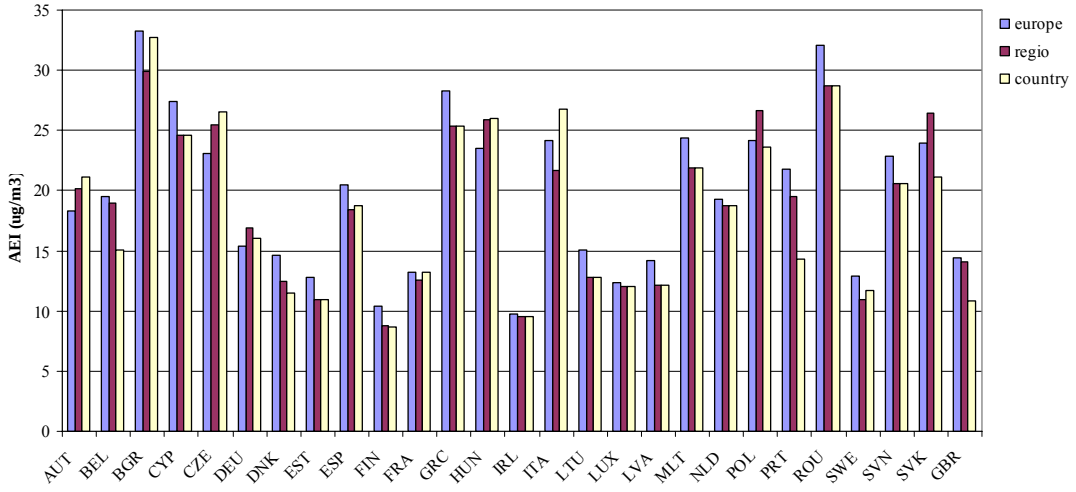


Figure 3.5 Average exposure indicator (AEI) calculated using European, region and country specific concentration ratios

3.4. Health Impact Assessment

The concentration map based on the region specific ratios (Figure 3.3) is used as input for a health impact assessment. The methodlogy is as described in section 1.2. with impact assessment at the 10x10 km resolution of the interpolated map. It is assumed that the population within a grid cell is exposed to the same grid cell averaged concentration. The estimated effect of PM on health is taken as an increase in incidence per unit increase in concentration according to Table 3.4. Results are given in Figure 3.6 and at the national level in Table 3.5.

Table 3.3 Estimates of relative risk of mortality, coefficients of concentration response function (B)

Health outcome	Exposure metric	Relative risk per 10 µg/m3 (95% CL)	Reference
Mortality from cardiopulmonary disease, adults > 30 year	$PM_{2.5}$	1.059 (1.015-1.105)	Pope *et al*, 2002
Mortality for lung cancer, adults > 30 year	$PM_{2.5}$	1.082 (1.011 – 1.158)	Pope *et al*, 2002
Total mortality, adults > 30 year; excluding violent death	$PM_{2.5}$	1.006 (1.002 – 1.010)	Pope *et al*, 2002
Total mortality, adults > 30 year; excluding violent death	PM_{10}	1.043 (1.026-1.061)	Kunzli *et al*, 2000

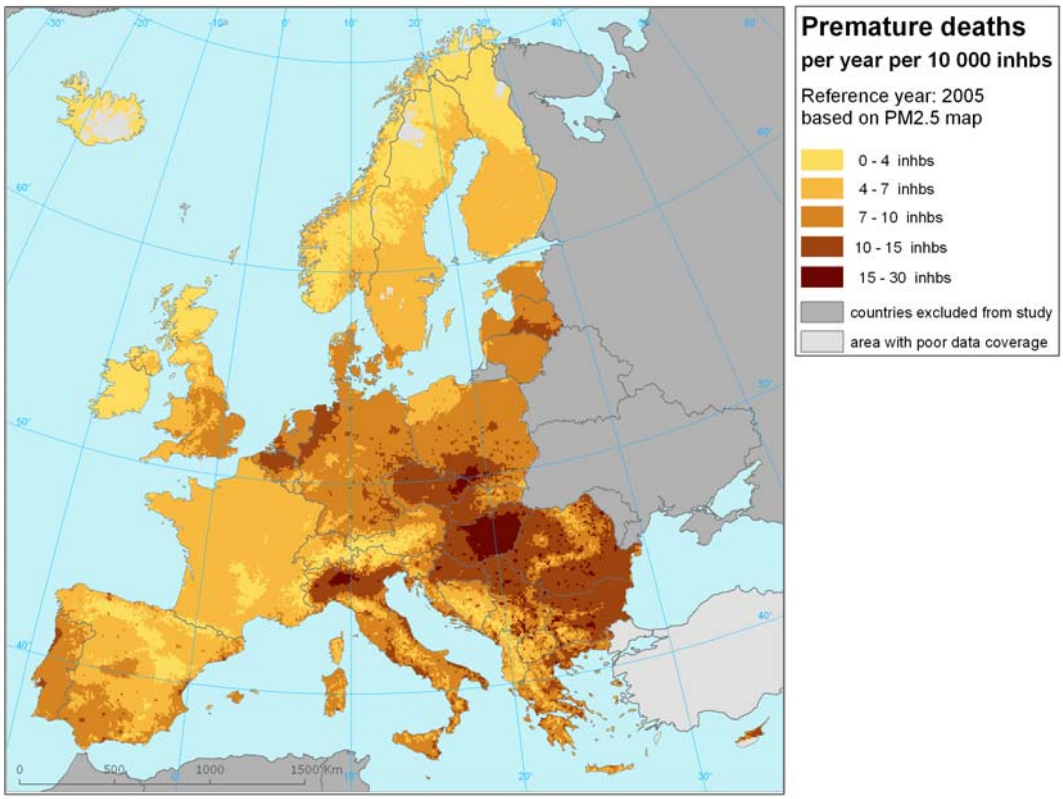


Figure 3.6 Premature deaths (all causes) attributable to PM2.5 (2005).

At one hand the map reflects the spatial differences in $PM_{2.5}$ concentrations, on the other hand, national boundaries are recognized caused by the demographic and health related input data at the national level.

The number of premature deaths attributable to exposure to PM_{10} and $PM_{2.5}$ are in good agreement; the estimated number attributable to PM_{10} is about 8% higher than the corresponding $PM_{2.5}$ number. The relative risk factors are of totally different origin. While the $PM_{2.5}$ risk factor (Pope et al, 2002) is based on an epidemiological study in six cities in the USA; the study by Kunzli is made in France, Switzerland and Austria and estimates the impact of PM_{10} exposure. There is considerable concern about the transferability of risk factors from one region to another where different conditions apply. These differences could be in population health, nutritional status, lifestyle, demographic variables, genetic disposition, and exposure to multiple stressors (psychosocial as well as environmental). Additionally the chemical composition and size distribution of the PM show strong spatial differences. The current results suggest that for one health outcome (total mortality) the sensitivity for these differences are small or they happen to cancel out.

For the other health outcomes the number of attributable cases to PM exposure is also given in Table 3.5.

Table 3.4 Premature deaths attributable to ambient particulate matter. Results are given for total mortality (all causes) based on PM10 exposure (second column) and PM2.5 exposure (third column), for cardiopulmonary diseases and lung cancer.

country	PM_{10}	$PM_{2.5}$	CPD	LC
Austria	6805	6939	3924	661
Belgium	11847	10727	5591	1590
Bulgaria	14429	12324	9335	747
Cyprus	1195	1008	598	51
Czech Republic	13385	13659	8955	1550
Germany	77061	78047	44139	8389
Denmark	4525	4247	2263	562
Estonia	1116	1032	913	89
Spain	42795	35948	18943	3837
Finland	2639	2521	1672	229
France	37965	34227	13943	3956
Greece	15829	13398	8269	1559
Hungary	15865	16234	10711	2147
Ireland	1524	1407	928	161
Italy	77667	65520	37067	7541
Lithuania	3043	2849	2372	241
Luxembourg	257	237	125	28
Latvia	2313	2143	1812	175
Malta	449	370	253	35
Netherlands	16673	15030	7398	2070
Poland	39049	39711	23802	5831
Portugal	12030	10164	6486	714
Romania	33550	28730	23560	2195
Sweden	5575	5376	3097	385
Slovenia	2064	1780	1047	205
Slovakia	5655	5795	4245	561
United Kingdom	51537	46249	31148	5511
Albania	2754	2333	1657	230
Andorra	39	32	16	4
Bosnia and Herzegovina	3755	3200	2267	334
Croatia	6700	5764	3787	635
Iceland	92	83	50	11
Lichtenstein	23	23	17	3
Norway	3096	2686	1600	250
San Marino	31	26	12	4
Serbia and Montenegro	17735	15063	10360	1303
Switzerland	4963	5007	2586	521
TFYR Macedonia	2813	2368	1990	190
EU27	496842	455673	272594	51021
Total	538843	492261	296936	54505

4. Revisiting health impact statistics: studies since 2002

4.1. Chronic $PM_{2.5}$ exposure; Coefficients for the relative risk of mortality

4.1.1. Introduction

A large number of epidemiological studies have shown an association between mass concentration of ambient particulate matter and an increased human mortality rate. Most epidemiological studies find a range of health outcomes. Data also indicate that air pollution is directly linked to the pulmonary toxicity, lung cancer, cardiovascular morbidity and mortality in the general population (Pope et al., 2006, Simkhowitch et al, 2008). The most vulnerable groups seem to be people with already developed diseases such as respiratory, cardiovascular disease or diabetes, or other susceptible groups such as with genetic predisposition, elderly etc.

General consensus is that air pollution can trigger an inflammatory response via reactive oxygen species (ROS) - dependent mechanism and stimulate local inflammatory reaction in the lungs. The ROS and pro-inflammatory cytokines released into the blood stream may cause adverse cardiovascular effect. Ultrafine particles may translocate into the circulation and induce oxidative stress and pro-inflammatory changes directly in heart and in endothelial cells. Several studies indicate that $PM_{2.5}$ particles are more hazardous than larger PM (Schwartz *et al*., 1997; Miller et al, 1979) as these particles more efficiently penetrate and deposit in the alveolar region of the respiratory system and can be translocated via blood stream (Cassee 2006). However, Brunekreef and Forsberg, (2005) examined the epidemiological evidence for effects of coarse particles on health and found that time series studies relating ambient PM to mortality in some places provided evidence of an independent effect of coarse PM on daily mortality, but in most urban areas, the evidence was stronger for fine particles. The few long-term studies of effects of coarse PM on survival did not provide any evidence of association.

Over the last decade numerous of epidemiological studies provided direct evidence of adverse health effect associated with both short term and long term exposures and addressed biological mechanisms and causality. General methodology to assess relative risk and quantification of health outcomes is constantly being reviewed by WHO, NAS, EPA and international committees and collaborative projects. Currently, many studies (both epidemiological and *in vivo)* which utilize methodologies that make it possible to quantify and monitor source of exposure (specific organic compounds and PM) and evaluate adverse health effect are in progress or planned, and results are not yet available.

Prospective cohort studies investigating long-term exposure are suggested as primary basis for estimating mortality effects related to air pollution. These studies evaluate the health effects in a specific population over a period of years. Compared with time series studies, which provide estimates of health effects due to recent exposure, the prospective cohort studies give a more complete assessment of the impact of air pollution since it includes long-term, cumulative effects (Krupnick et al., CAFE peer reviewed, 2005). The WHO reports from 2004 and 2006 recommend using the ACS study as reference, and most ongoing European studies and projects are following this recommendation (CAFE, Apheis, Boldo et al., 2006, and others). However, there is a need to investigate if the recommended coefficient based on estimate of a 6 % increase in mortality hazard rates per $10\mu g/m^3$ (Pope et al 2002), must be updated. To be able to do this, we have reviewed the most recent literature and projects and compared the results from the ACS study to the available European studies (NLCS-AIR Study).

The time-series-studies are used to find the daily / acute exposure and are especially useful for finding mortality within vulnerable groups. However, as we (so far) have concentrated on the relative risk for the general population, the relative risks for vulnerable groups have not yet been investigated in this review.

4.1.2. Method

A literature search was made in PubMed for articles published between 2000-2008. From the 297 articles found we chose to concentrate on European and North-American studies, and separated the remaining articles into long-term (18) and short-term studies.

In addition we investigated the findings of the Clean Air for Europe (CAFE) project, and the recommendations from WHO.

The ongoing EC project funded under framework programs (FP6 and FP7) such as Intarese, Heimtsa, Henvinet, Envirisk and recently finished DROPS were originally planned to be used as their aim is to evaluate effect of air pollution on human health. Unfortunately, data from these studies are not available yet.

4.1.3. Results and discussion

Results of the review of available studies are summarised in table 4.1. The two American studies (ACS and Six Cities) are the most comprehensive studies available. Both have been reanalysed, extended and followed up. These studies have used data from single monitoring stations to characterize exposure of subjects living in that city, or spatial interpolation from multiple monitoring stations.

AMERICAN STUDIES
Six Cities:
Measurements have shown that the annual mean $PM_{2.5}$ concentrations have decreased in the decade of the 1990s compared to the mid-1970-80s when the study originally took place. This can decrease the risk of mortality, especially for cardiovascular and respiratory disease (Laden et al, 2006). However, the follow-up also found statistically significant increased risk in overall mortality associated with each 10µg/m^3 increase compared to the original study, giving a rate ratio (RR) of 1,16 [1,07-1,26].

ACS:
This study is stated as very robust for statistical approaches. It has been extended and reanalyzed, with confirming results. A detailed investigation of covariate effects found a significant modifying effect of education with risk of mortality associated with fine particles declining with increasing educational attainment. This was included as covariates in the follow up (Pope et al 2002). It is believed that more covariates are necessary for estimating the risk. Current ongoing analysis using the extended follow-up information will explore the role of ecologic, economic, and, demographic covariates in the particulate air pollution and mortality association. This analysis will also provide insight into the role of spatial autocorrelation at multiple geographic scales, and whether critical instances in time of exposure to fine particles influence the risk of mortality from cardiopulmonary and lung cancer (Krewski et al 2005). In his study Pope presents three different sets, one based on averaged concentrations over 1979-1983, the second for averaged 1990-2000 and a third is the averaged for these two periods. When comparing the 79/83 & 99/00 numbers there is an increase in RR. This is discussed in WHO Comparative Quantification of Health Risks (WHO/Cohen et al, 2004). However, it is still recommended to use average coefficient as the European situation may represent a mixture of pollution situation in various periods of ACS study. The average coefficient was also used in CAFE (2005) throughout the follow-up period, as is recommended by WHO/THF. A more recent report from WHO (2006) explains this choice in more details.

ACS Inter-city study:
One study in a subpopulation of the ACS study in Los Angeles, estimated exposure to $PM_{2.5}$ at intra-urban scale, and found the RR higher than for the between-city studies for both all-cause, cardiopulmonary and lung cancer (1,17 [1,05-1,3], 1,12 [0,97-1,30], 1,44[0,98-2,11]), suggesting that health effects associated with within-city gradients in $PM_{2.5}$ concentrations may be larger than previously found across metropolitan areas (Jerret et al, 2005).

Woman health initiative study
This study examined the association of long-term exposure to $PM_{2,5}$ with cardiovascular events in women. The exposure was measured using the monitor located nearest to each woman's residence. Each increase of 10 $\mu g/m^3$ was associated with a 76 % increase in the risk of death from cardiovascular disease. They also compared the between-city and within-city effect, assessing hazard ratios for a 10 $\mu g/m^3$ increase of 1,63 (1,10-2,4) and 2,28 (1,10-4,75) respectively (Miller et al 2007).

EUROPEAN STUDIES
Cohorts from Europe have tended to confirm the US findings (Hoek et al, 2002; Nafstad et al, 2004), although the emphasis has been on different pollutants and on different exposure assessment methods. Most European studies have estimated exposure at the home address using dispersion or stochastic modelling and variables such as living close to busy roads (Beelen et al, 2008a). Long term exposure to traffic-related air pollution and lung cancer risk study investigates the lung cancer incidences following air pollution exposure but not mortality (Beelen et al 2008b). Gallus et al (2008) reviewed European studies on long-term exposure to ambient particulate matter and lung cancer. Long-term exposure to urban air pollution and myocardial infarction and cardiopulmonary mortality was also studied (Rosenlund et al 2006).

NLCS-AIR:
The NLCS-AIR Study have estimated the $PM_{2.5}$ concentration by converting PM_{10} concentrations, measured in the NAQMN from 1992 to 1996 into $PM_{2.5}$ using a single ratio, established from monitoring data in the Netherlands. The RR estimates for $PM_{2.5}$ (measuring inter-city gradients) for natural cause, cardiovascular and lung cancer is respectively (although not statistically significant): 1,06 [0,97-1,16], 1,04 [0,90-1,21] and 1,06 [0,82-1,38] (Beelen et al 2008a).

Norwegian cohort:
Recently the relation between concentration of air pollution and cause-specific mortality four-year exposures to nitrogen dioxide and particulate matter pollutants in 470 neighborhood was cstudied. In this study the hazard ratios were investigated in men and women in two age groups (51-70 and 71-90 years) for deaths from CVD and lung cancer according to exposure from PM2.5 (quartile increase with concentrations 6.56-11-45; 11.46-14.25; 14.26-18.43; 18.44-22.34 $\mu g/m^3$). Their measurement show increased effect for both men and women and both age groups, but with larger effect for the younger. The most vulnerable group appeared to be young women, where rather large effects were found for both cardiovascular disease and lung cancer (HR of 1.14 and 1.27, respectively). HR for all causes of deaths showed that it appears to be no effect of PM2.5 in the two lowest quartiles below 14 micrograms/m3, implicating that there could be a threshold effect (Næss et al 2007).

4.1.4. Summary and remarks

We believe it is reasonable to compare US results with European as the mix of PM will be approximately the same, as are housing stock, seasonality, lifestyle, range of weather conditions, background health status, etc. However, the emphasis in US studies has been on different pollutants, on different exposure assessment methods and on different endpoints.
Additionaly, WHO report 2006 discusses differences such as PM2.5 arithmetic average concentrations, between US and European studies. The two largest US studies are based on between-city studies, while the European takes inter-city gradients into consideration. Recent studies show that

the within-city effect might be larger than the between-city effect (Miller et al, 2007; Jerret et al, 2005). This indicates that assessing the exposure based on the average concentration in the whole city, might lead to an underestimation of the risk for people who live in neighbourhood with high levels of concentration. The results from the NLCS-AIR study (intra city) are in the same range as the coefficient used as a reference today (Pope et al, 2002), but because of the differences in measurements these results might not be comparable.

Most (European) studies are based on small numbers which may increase the uncertainty to assess the risk. Moreover, latency period for different adverse health effects varies; it might be large difference between development of cancer and cardiovascular or cardiopulmonar diseases. It is known that the latency for development of cancer is quite large; it may take several decades before the effect is visible. Some studies only counts mortality after less than 10 years which might be too early to detect the effect.

It is clear that there is an association between suspended PM concentrations and mortality and morbidity rates. Such effects depend on particle size and concentration and can fluctuate with daily fluctuations in PM10 or PM2.5 levels. The relation between PM10 or PM2.5 exposure and acute health effects is supposed to be linear at concentrations below 100 $\mu g/m^3$. Currently no threshold has been reported below which no effects occur. The influence of co-polluting gaseous pollutants could explain part of the observed variance in short-term health effects and reduce the contribution of suspended PM (Shwela, 2000).

Overview of the literature

American studies:

- The Harvard Six Cities Study (Six Cities)
  - Dockery et al 1993
  - Reanalyzed by Krewski et al 2000 (confirmed results)
  - Commented by Lipfert 2003
  - Overview of reanalysis by Krewski et al 2003
  - Retrospective quality data quality audits by Hoover et al 2003
  - Validation by Krewski et al 2004
  - Reanalyzed by Krewski et al 2005 part 1(validation and replication)
  - Reanalyzed by Krewski et al 2005 part 2 (sensitivity analysis)
  - Extended follow-up by Laden et al 2006
  - Comment to the updated Six Cities by Gamble and Nicolich 2006
  - Compared with a Medicare cohort by Eftim et al 2008 (somewhat higher risks)
- The American Cancer Society Study (ACS)
  - Pope et al 1995,
  - Reanalyzed by Krewski et al 2000 (confirmed)
  - Extended by Pope et al 2002
  - Commented by Lipfert 2003
  - Overview of reanalysis by Krewski et al 2003
  - Retrospective quality data quality audits by Hoover et al 2003
  - Ongoing analysis by Krewski et al 2005
  - Compared with a Medicare cohort by Eftim et al 2008 (somewhat higher risks)
- Woman health initiative study
  - Miller et al 2007

European study:

- The Netherlands Cohort Study on Diet and Cancer (age 55-69) (NLCS-AIR Study)
  - Hoek et al 2002
  - Beelen et al 2008
- Norwegian (Oslo) Cohort study
  - Næss et al 2007

WHO still recommends the use of only one risk factor and one coefficient when assessing the health impact of ambient PM exposure. Obviously more knowledge and mechanistic information is needed to be able to identify dose response effect and to assess the risk. Many critical issues such as background levels of exposure, threshold/non-threshold approach, additional endpoints (such as life years lost, and more specific morbidity and mortality subcategories), vulnerable groups, source

specific measurement of pollutant, quantification of uncertainities etc need to be discussed. Design of epidemiological studies, and selection criteria should be carefully considered and clear. The choice of endpoints including molecular markers and biomarkers of individual susceptibility and focus on vulnerable groups can considerably help to identified association between exposure and adverse health effect. A more sophisticated molecular/ toxicological-based source specific approach to air quality management is likely to contribute significantly to the hazard and risk identification and to the development of cost effective abatement strategies.

4.1.5. Conclusion

The present approach used for the evaluation of the health impact of air pollution obviously is not sufficient, since it does not take into consideration all aspects of dose response relationships. There may be differences in health outcomes depending on whether PM2.5 is present at low, medium or high concentration; whether there is a threshold; or whether it is present in combination with other pollutants. However, our review suggests that there is still not enough knowledge to propose a new method for the evaluation of the health impact of air pollution (PM2.5), and therefore the use of the estimated coefficient 6% per 10μg/m^3 of PM2.5 (based on recent prospective studies) as the primary basis for estimating all cause mortality seems still reasonable for European conditions. For lung cancer and cardiopulmonary mortality this gives an adjusted RR of 9 % and 14 %, respectively (Table 4.1)

4.2. Ozone exposure: Some considerations on the relative risk of mortality

4.2.1. Method

The literature search used to extract recent work on the effects of air pollution discussed in chapter 4.1 included health effects from ozone. The findings of the Clean Air for Europe (CAFE) project, and the recommendations from WHO were similarly considered. As before, unavailability of results from current EU financed work, such as Intarese, Heimtsa, Henvinet, Envirisk and the recently finished DROPS has meant the results of these projects are not included in this study.

4.2.2. Risk estimates for Ozone

Risk estimates for ozone (O^3) are mostly performed using time-series studies. Specifically, the WHO meta-estimates indicated a relative risk of 1.003 (95% CI = 1.001 – 1.004) for a 10 μg/m^3 change in 8-hour ozone. For standard atmospheric pressure and temperature, 1 ppb ozone equals 1.96 μg/m^3. In addition, the average ratio between 1-hour and 8-hour ozone is 1.33 (Schwartz 1997). WHO estimate implies a 0.44% change in daily mortality (95% CI = 0.15 – 0.59%) per 10 ppb change in 1-hour maximum ozone (Krupnick et al., 2005) .CAFÉ also propose estimate for ozone based on the WHO-sponsored meta-analysis of 15 European cities (Anderson et al., 2004). An estimate of 0.3% per 10 μg/m^3 ozone (8 hour) is suggested, with an implicit threshold of 35 ppb (8-hour average). The metaanalysis by Levy et al. (2001) includes 50 time-series analyses from 39 published articles. models. This analysis generated an estimate of 0.5% (95% CI = 0.3 –0.7%) per 10 μg/m3 change in 24-hour average ozone which represents 0.39% change in daily mortality per 10 ppb change in daily 1-hour maximum ozone (95% CI= 0.24-0.55%). Stieb et al. (2002) also reported a similar effect estimate (0.51% per 10 ppb change in daily 1-hour maximum ozone).

In contrast, a lower effect estimate is provided by the National Morbidity, Mortality, and Air Pollution Study (NMMAPS). The revised analysis of this large study, conducted in 90 US cities found an effect estimate of 0.17% per 10 ppb change in 1-hour maximum ozone after conversion from the 24-hour average reported in the published study (Dominici et al. 2003). This estimate is similar to the lower bound of the WHO estimate. Based on the currently published data it seems that the WHO analysis provides a reasonable estimate of the effect of ozone.

Table 4.1 Adjusted relative risks and 95% confidence interval in American and European long term studies

Study	Author	Duration of cohort	# subjects	Note	Age and sex	General Mortality		Cardiopulmonar Mortality		Cardiovascular Mortality		Lung Cancer Mortality	
American						RR	CI	RR	CI	RR	CI	RR	CI
ACS a	Pope et al, 2002	1999-2000		Extended follow-up	M F, 30-	5.9 %	2,0-9,9 %	7.9 %	2,3-14 %			12.7 %	4,1-22 %
ACS a	Pope et al, 2002	1979-1983, 1999-2000	295 223	Extended follow-up, mean	M F, 30-	1.06	1,02-1,11	1.09	1,03-1,16			1.14	1,04-1,23
Inter-city ACS	Jerret et al, 2005		22 905	Study of sub-population	M F, 30-	1.17	1,05-1,3	1.12	0,97-1,30			1.44	0,98-2,11
Six cities b, *	Dockery et al, 1993	1979-1988	8 096	Original study	M F	13.2 %	4,2-23 %			18%	5,8-32 %	18%	-11-57 %
Six cities b	Laden et al, 2006	1979-1988, 1990-1998		Entire extended follow-up		1.16	1,07-1,26			1.28	1,13-1,44	1.27	0,96-1,69
WHI c	Miller et al 2007	1994-1998	65 893	Overall effect	F, 50-79					1.76	1,25–2,47		
	Miller et al 2007	1994-1998		Between-city effect	F, 50-79					1.63	1,10-2,40		
	Miller et al 2007	1994-1998		Within-city effect	F, 50-79					2.28	1,10-4,75		
European													
NLCS-AIR d	Beelen et al, 2008	1987-1996	120 852	Within-city effect	M F, 55-69	1,06#	0,97-1,16#			1.04	0,9-1,21	1.06	0,82-1,38
Norwegian e, §	Næss et al, 2008	1992-1998	143 842	Within-city effect	M, 51-70					1.10	1,05-1,16	1.07	0,97-1,18
	Næss et al, 2008	1992-1998			F, 51-70					1.14	1,06-1,21	1.27	1,13-1,43
	Næss et al, 2008	1992-1998			M, 71-90					1.05	1,01-1,08	1.07	0,97-1,18
	Næss et al, 2008	1992-1998			F, 71-90					1.03	1,00-1,05	1.16	1,02-1,32

a Estimated and adjusted based on the baseline random-effects Cox proportional hazards model, controlling for age, sex, race, smoking, education, body mass, alcohol consumption, occupational exposure and diet.
b Rate ratios have been adjusted for age in 1-yr categories, sex, current smoker, current pack-years of smoking, former smoker, former pack-years of smoking, less than high schooleducation, and linear and quadratic terms for body mass index.
c Estimates are adjusted for age, race or ethnic group, educational level, household income, smoking status, systolic blood pressure, body-mass index, and presence or absence of a history of diabetes, hypertension or hypercholesterolemia
d Full cohort analyses adjusted for age, sex, smoking status, and area level indicators of socioeconomic status. Case–cohort analyses adjusted for age, sex, BMI, active smoking, passive smoking, education, occupational exposure, marital status, alcohol use, vegetable intake, fruit intake, energy intake, fatty acids intake, folate intake, fish consumption, and area-level indicators of socioeconomic status.
e Adjusted for occupational class and lenght of education
*Rate Ratio
Natural Cause
§ Hazard Ratio quartile increase

Though RR for Ozone may be more of importance when looking at short-term effects of pollutant several authors suggest that the use of studies of the long-term exposure to ozone should be consider. Though in the analysis of the ACS cohort, Pope et al. (2002) did not find any association between annual concentrations of ozone and life expectancy, a week evidence indicating a possible inflammatory response to ozone exposure and evidence of long term effects on lung function was found for long-term summertime exposure to ozon (Poppe et al., 2002, Krupnick et al., 2005).

Results of other study (Chen et al 2005) show that the relative risk increase when using a two-pollutant model combining PM and O^3. In this study, in females, the RR for fatal coronary heart disease with each10$\mu g/m^3$ increase in $PM_{2.5}$ was 1,42 [1,06-1,90] and 2,00 [1,51-2,64] in the single-pollutant model and in the two pollutant model with O^3 respectively.
Several studies suggest that long-term exposure to Ozone or in combination with PM exposure can be associated with adverse health outcomes and should be therefore taken into consideration when estimating RR.

5. Summary and conclusions

-) accounting for smaller scale exposure as well as at the residential address of an urban population will increase the estimated exposure of the total population above that estimated on the basis of a total urban population exposed to a single averaged air concentration.

-) in addition, the movement of a city population daily towards the centre, including commuting on traffic corridors, increases the general exposure level. For a limited percentage of the population this increase will mean exposure to higher concentration brackets.

-) Whilst the proportion of the population freshly exposed to higher concentration brackets may be small, this translates into very large absolute numbers for Europe as a whole.

-) these smaller proportions still represents an underestimates the proportion of the total European population subjected to higher exposure, as a significant fraction of the non-urban population commutes into urban areas daily. This group may be expected to be subject to substantial increases in exposure.

-) whilst daily movements may mean that the average exposure of European urban populations may be of the order of 20% greater than estimated by a simple urban average, such averaging may give misleading results. This increased exposure, and the potential health effect, will in reality be focused on an identifiable target group with a significantly increased exposure, rather than being averaged across a larger number.

-) regions of Europe experience different relationships between $PM_{2.5}$ and PM_{10} concentrations at rural, urban and trafficked locations, encouraging regionally specific assessments.

-) when the exposure of the urban population in Europe to $PM_{2.5}$ is estimated, it is found that for 10 Member States the Average Exposure Index lies above the binding value for 2015 of 20 $\mu g/m^3$, in 5 Member States it lies at or below this level, whilst for 12 Member States the average exposure index is cearly below.

-) the total estimated premature deaths from exposure to $PM_{2.5}$ approximates those from exposure to PM_{10}.

-) review of recent literature on the health impacts o exposure to $PM_{2.5}$ does not support updating the previous coefficient for estimating mortality of 6% per 10$\mu g/m^3$ of $PM_{2.5}$

-) there is evidence to suggest combined adverse effects of exposure to $PM_{2.5}$ along with exposure to ozone.

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